December 2013 Issue 10

The International Medical e-Network devoted to
Fetal Alcohol Spectrum Disorders

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INTRODUCTION

Though the prevalence of Fetal Alcohol Spectrum Disorder has yet to be officially calculated, in most countries, international studies estimate that FASD may affect between 1% and 5% of the population.

For example, in the UK (population 63.7 million) 1%-5% would translate into between 637,000 - 3,185,000 people with FASD. How many people do you suppose have FASD in your country?

We must also consider how FASD affects the wider community and those who support and manage the FASD population, including family members, teachers, doctors, police, etc. The emotional, as well as the financial cost of FASD to society is immeasurable.

To help prevent FASD, NOFAS-UK has begun training General Practitioners and is in its fifth year producing training for Midwives. To date the charity has trained and provided resources for over 10,000 midwives. To learn more, read Chapter 15, The Baby Bundle Project in the book FETAL ALCOHOL SPECTRUM DISORDERS – INTERDISCIPLINARY PERSPECTIVES, found on Amazon.co.uk. To watch a video of the midwives training and other films go to: www.nofas-uk.org

In this issue of the FETAL ALCOHOL FORUM the articles and abstracts not only provide research and evidence of the cost and challenges regarding FASD, but it also includes links to hopeful developments in the FASD world, such as the AUSTRALIAN FETAL ALCOHOL SPECTRUM DISORDER ACTION PLAN 2013-2016 by FARE (Foundation for Alcohol Research & Education).

Canada is producing some of the leading FASD experts and promising innovations. In November, the FASD Centre in Manitoba, Canada and the Scottish Government led a collaboration which saw almost 100 health and social care professionals and 50 caregivers in Scotland trained by the Canadians in assessing, recognizing and managing FASD.

On 9th September, International FASD Day, the first FASD Global Gathering was organized from Victoria, Canada. The LIVING WITH FASD SUMMIT connected over 2,300 people by organizing an international Skype Conference. Every day for two weeks, participants were able to hear two hour presentations and ask questions of 22 international FASD experts. The link to this exceptional conference can be found in this issue as well as an article written by its creators, David Gerry and Cheryl Sivertson.

You will hear some of the participants who have FASD speak in the Summit. These
impressive young people along with some new FASD research provide us with hope and optimism for better life outcomes for those born with FASD and all of us who support them.

The table below shows the FASD Studies done worldwide during the past 6 months.

NOTE: FASD studies worldwide during the past 6 months

<table>
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<tr>
<th>Country</th>
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<td>USA</td>
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We always appreciate your comments and valuable feedback at info@nofas-uk.org. You can download issues of the FETAL ALCOHOL FORUM from our website: www.nofas-uk.org, or if you would like to be added to the FETAL ALCOHOL FORUM mailing list, please click here.

Susan Fleisher
Publisher

Beata Ewertowska
Editor

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Associate Editor
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*In the interest of brevity, Fetal Alcohol Spectrum Disorder has been abbreviated to FASD

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SPECIAL FEATURE – LIVING WITH FASD 2013 TELESUMMIT

An Online Resource for Families Living With Fetal Alcohol Spectrum Disorder

Are you a parent (biological, foster, step, adoptive, grand, etc.) living with someone who has Fetal Alcohol Spectrum Disorder (FASD)? Are you hungry to connect with other parents and hear about strategies from experts, especially those who have raised children with FASD? Yet can't afford the time away from home or the cost of travel and hotel to attend conferences on FASD?

The Living With FASD 2013 Summit may be exactly what you’ve been looking for!

The purpose of this Summit is to provide families with information about practical strategies to implement at home, based on the newest FASD research. Speakers have been specially selected because they have experience on a professional level and also have first-hand experience raising children with FASD.

Discover the first-hand challenges our experts faced and the lessons they learned. Gain insight into the knowledge they gained, the resources they used, and techniques they developed. From this unique perspective, they can provide you insight on a range of topics.

- See more at: http://livingwithfasd.com/#sthash.0Miluo0T.9eRbzUYR.dpuf

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I. A POSITIVE PERSPECTIVE FROM NORTHERN IRELAND - HOPE IN THE FIGHT AGAINST FASD

Article on Foetal Alcohol Spectrum Disorder Based on Dr Mitchell’s Speech
Dr Elizabeth Mitchell
Deputy Chief Medical Officer
Department of Health Social Services & Public Safety Northern Ireland

I was delighted to be asked to speak at the “A Pregnant Pause” seminar in Lisburn on International Foetal Alcohol Spectrum Disorder Day, 09 September 2013 (http://www.science.ulster.ac.uk/inhr/news/Fetal-Alcohol-Spectrum-Disorder.php). The event, which was organised by the South Eastern Health and Social Care Trust and the University of Ulster, provided a great opportunity to raise awareness of this issue and reflect on the challenges ahead in terms of preventing FASD and supporting those affected.

Most importantly, I felt the event helped to give us all a real sense of hope. Hope that we can help and support everyone to reach their full potential in life. Hope from advances in neuroscience, which show that the brain continues to develop and is not “set in stone” – though this work is still in its infancy there is much optimism from the new science of “neuroplasticity”. And finally, hope that we can get better at preventing this condition and supporting those affected.

I recognise that I am not an expert in this area, and those who spoke at the event – with eloquence, passion and experience – were the real experts and were much better qualified to speak on this topic. I was also taken by the fact that those in the audience – from maternity services, social services, the community and voluntary sector, and those affected by FASD – deal with this issue, and its outcomes, on a more regular basis than I do and therefore know more about this topic. However, it was important that I had the opportunity to set out the Department’s strategic vision in this area.

Alcohol misuse is one of the major public health challenges we face. Drug misuse often gets the headlines, but in Northern Ireland alcohol remains our drug of choice. It is so prevalent that all Health and Social Care Professionals will have experience of dealing with the consequences of alcohol related harm. Indeed, many people will also have firsthand experience of dealing with the consequences of alcohol misuse through our families, friends, or work colleagues.

This is a societal issue – 74% of adults in Northern Ireland drink alcohol and almost four in five drinkers exceed the daily guidelines at least once a week. Proportionately, and paradoxically, fewer people here drink than in the rest of the UK and Ireland as we have more abstainers. However, the real issue is how we drink. We have an unhealthy “weekend” binge drinking culture, and statistics show that 30% of those who consume alcohol binge drink (more alcohol and drug statistics for Northern Ireland are available at the following link: http://www.dhsspsni.gov.uk/index/stats_research/stats-public-health/stats-drug-alcohol.htm).

Extrapolating from these survey figures we can estimate that about 170,000 adults in Northern Ireland drink at hazardous levels and a further 47,000 adults drink at harmful levels. This gets to the root of the issue, because it is the harm that alcohol misuse causes which we want to prevent. Alcohol misuse:

- increases risk of getting cancer, cardiovascular conditions, liver disease;
- increases risk of poor mental health and self-harm;
• increases the risk of being involved in accidents and of being involved in, or a victim of, violence or assaults; and
• it can lead to Foetal Alcohol Spectrum Disorder.

To prevent and address this issue the Department of Health, Social Services, and Public Safety has developed, and is overseeing the implementation of, a cross-sectoral strategy to prevent and address the harm related to all substance misuse – known as the New Strategic Direction for Alcohol and Drugs (NSD) Phase 2 (http://www.dhsspsni.gov.uk/new_strategic_direction_for_alcohol_and_drugs_phase_2__2011-2016).

Obviously we recognise that the Health Service, and Health Professionals, have a key role to play in helping to prevent and address this issue, but the Strategy also recognises that in order to be effective we also need buy-in and support from the education sector, the criminal justice sector, civil society, parents, individuals, the Department of Social Development in respect of licensing laws (http://www.dsdni.gov.uk/index/law_and_legislation/social_policy.htm), and many others.

The NSD Phase 2 therefore recognises the need to reduce overall alcohol consumption in the population, and it sets out outcomes across five main areas: prevention and early detection and intervention; harm reduction; treatment and support; legislation and, law and criminal justice; and monitoring, evaluation and research.

I firmly believe that we have to do more collectively to challenge drinking behaviours at both an individual and a population-wide level. The NSD Phase 2 recognises the wider influences that determine our drinking culture in Northern Ireland. Therefore, we are looking at universal approaches such as reducing availability and accessibility, and potentially introducing minimum unit pricing, which helps to send out the message that alcohol isn't an ordinary product.

Raising awareness, and informing and educating those who are pregnant or are trying to conceive, is very important. I believe we need to be clearer about the harm alcohol use in pregnancy can cause. Currently, the four UK Chief Medical Officers (CMOs) guidelines state: “Pregnant women or women trying to conceive should avoid drinking alcohol; if they do choose to drink, to minimise the risk to the baby, they should not drink more than one to two units of alcohol once or twice a week and should not get drunk” (for more information see: http://www.knowyourlimits.info/know%E2%80%A6-about-conception-and-pregnancy).

I know many people believe this is a mixed message, and one that makes it difficult to present clear recommendations to pregnant women. This is an issue that has been raised to us on a number of occasions, and I understand and acknowledge these concerns. Some people will also believe that the second part of the guidance gives people a excuse to continue to drink. However, there is a clear harm reduction message in the current guidelines that can be used to support those who won't, or can't, stop drinking. This is important because by reducing levels of consumption we can reduce risk and it also gives those who can't stop drinking a goal to work towards, rather than setting them a target they believe they can never achieve and thus stopping them even trying.

The current guidelines reflect the evidence available at the time they were developed in 2006 and since their publication the evidence base has been further developed. In addition, I believe we need to give further thought to not just what the evidence says, but how we communicate it. The four UK CMOs have agreed to look again at all the alcohol guidelines. Given the concerns that have been expressed to us, we have specifically asked that this review included looking at the alcohol in pregnancy guidelines. It is anticipated that a review of the evidence will be completed in early 2014, and subsequently there will be a need to look again at how we communicate these messages clearly, whether or not the supporting evidence base has changed.

Another area we have emphasised specifically in our alcohol and drug strategy is the need to promote and ensure the use of alcohol brief interventions, within the health sector and beyond. As professionals, we all need to take responsibility for identifying those with a problem. However, this cannot just be about identification, we need to provide further support, encouragement, and
motivation to individuals through a brief intervention. We have made some good progress in rolling out brief interventions in primary care, we now need to look at other relevant sectors and I believe maternity services should be a high priority.

FASD also links into the wider work we are doing on Hidden Harm, which refers to the children and young people born to, or living with, substance misusing parents or carers. Whether or not a child is affected by alcohol misuse in the womb they can still be affected by their parent’s substance misuse in later life and we continue to implement a regional action plan to address this issue (see: http://www.dhsspsni.gov.uk/regional_hidden_harm_action_plan.pdf)

The “Pregnant Pause” seminar, held on International Foetal Alcohol Spectrum Disorder Awareness Day, provided the perfect opportunity to raise awareness of this issue, to take stock of where we are to date, and most importantly to challenge ourselves to do more in the future. The speakers provided many ideas on how we can all work together collectively to prevent this issue and we now need to look at turning these ideas into firm action that informs our everyday work.

Most importantly, the event helped to give us all hope. Hope that we can make a difference, hope that we can prevent this condition, and hope for those affected.

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II. DIAGNOSING FASD IN THE ERA OF DSM-5: GOOD NEWS FOR THE FORENSIC CONTEXT

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With the publication of the new Diagnostic and Statistical Manual, Fifth Edition (DSM-5), in the United States, the neurodevelopmental effects of fetal alcohol spectrum disorders (FASD) are featured for the first time in the DSM's history as a mental health condition. Diagnosed as “Neurodevelopmental Disorder associated with Prenatal Alcohol Exposure (ND-PAE),” the condition is included under “Specified Other Neurodevelopmental Disorder” (Code 315.8) with six other disorders (i.e., Intellectual Disability, Communication Disorders, Autism Spectrum Disorders, Attention-Deficit/Hyperactivity Disorder, Specific Learning Disorder, Motor Disorders) in the Neurodevelopmental Disorder section of the DSM-5. This is very good news
because now, for the first time in its 40-year history as a known medical condition, the brain-based mental health sequellae of FASD can be diagnosed by mental health professionals. While multidisciplinary diagnosis involving an array of medical, allied health, and mental health specialists remains critical for diagnosing and treating children and adolescents in the clinical setting and the “gold standard” for diagnosing adults in high-stakes forensic settings, the DSM-5 now guides diagnosis of ND-PAE by individual psychiatrists or psychologists.

In the previous nosological system, FAS was a medical diagnosis of exclusion in dysmorphic patients. The number of patients who were identified with any FAS was quite limited due to costly diagnostic systems (CDC, HHS, NOFAS, 2005) and skewed toward the typical facial features. Developmental pediatricians, geneticists, and pediatric dysmorphologists ruled out all genetic causes of abnormal faces and neurodevelopmental issues before diagnosing a child with FAS or partial FAS (CDC, HHS, NOFAS, 2005). Unfortunately, few children actually made it into the offices of these specialist pediatricians, particularly those from rural, inner city, and small town communities. Even fewer non-dysmorphic individuals came to the attention of such subspecialists. The complex facial morphometric protocols led to family physicians’ and pediatricians’ reluctance to diagnose these conditions, even with a clear history of PAE, obvious developmental delays, and/or overt intellectual disability. Since “Alcohol-related Neurodevelopmental Disorder (ARND)” had no diagnostic code, most cases of FASD were not diagnosed at all. While pediatric specialists diagnose FAS (in the presence of dysmorphic features, it is psychiatrists who have typically treated the wide range of related neurodevelopmental and psychiatric sequellae, often without appreciating the correlation with prenatal alcohol exposure. Training about FASD is scarce in medical schools and residency programs, and typically non-existent in graduate psychology programs. Most psychiatrists are ill-equipped to construct the complex neurodevelopmental formulations needed to help these patients achieve their optimal level of functioning, and most psychologists have been uniformed about FASD altogether. In turn, treating clinicians in outpatient settings, hospitals, long-term treatment centers, and the justice systems are challenged by the underlying neurochemical, neurophysiologic, and neuroanatomic alterations associated with alcohol-induced prenatal brain injury.

Since the brain-based neurodevelopmental issues (and not the facial features) respond to treatment, we and others believe that a shift toward recognition of ND-PAE will shore up the proverbial cracks in the academic, mental health, and justice systems. Hopefully, the diagnosis will improve coordinated systems of care, comprehensive disability services, and caregiving environments to prevent children ending up in juvenile court (Streissguth, et al., 1991, Steinhausen, et al., 1993, Streissguth et al., 1996) and cycling through adult corrections. We in the field know all too well that the “FASD transition to adulthood” often leads to homelessness, prostitution, substance abuse, unemployment, and crime (Streissguth & O'Malley, 2000, Streissguth, et al., 2004). “Failure to thrive” in society is the typical outcome for individuals with FASD due to their adaptive functioning deficits, which are well beyond their predicted performance based on IQ.

**What is ND-PAE?**

The DSM-5 notes that ND-PAE involves a “range of developmental disabilities following exposure to alcohol in utero” and includes both individuals with ARND as well as those with FAS and partial FAS (pFAS). Neither ARND nor ND-PAE requires the presence of physical abnormalities (i.e., facial features, growth deficit). The following criteria are listed in DSM-5 (Section 3) as the neurodevelopmental manifestations of ND-PAE:

The DSM-5 notes the following with respect to physical (i.e., medical) symptoms: “The current diagnostic guidelines allow ND-PAE to be diagnosed both in the absence and in the presence of the physical effects of prenatal alcohol exposure (e.g., facial dysmorphology required for a diagnosis of fetal alcohol syndrome).” Eliminating strict criteria for facial abnormalities in ND-PAE diagnosis is consistent with strong evidence in multiple studies that FAS is not associated with worse outcomes compared to ARND (e.g., Streissguth et al., 1996). In fact, the reverse is true. Researchers tend to attribute worse life course outcomes in ARND to the absence of physical features, which reduces the odds of diagnosis and treatment.
ND-PAE: Implications for Forensic Practice

Since the mid-1990s, we have known that FASD (i.e., ND-PAE) is associated with a high risk of criminal behavior, with first offenses typically committed during the juvenile years before brain development is complete even under normal circumstances. Moreover, youths with ND-PAE have triple-jeopardy: they are born with permanent brain damage, their brains develop abnormally in childhood (Treit et al., 2013), and it is not until the mid-20s that brain development is complete. The prefrontal cortex is the last area of the brain to develop. This area of the brain handles decision-making, judgment, and impulse control - all of which are implicated in most criminal conduct. These areas are highly sensitive to prenatal alcohol exposure, with binge episodes in the first weeks of pregnancy as damaging as more moderate use throughout pregnancy (Meier & West, 2001).

Another important change in DSM-5 involves recognition of how FASD/ND-PAE and ID/MR are connected. Under DSM-IV TR, ID/MR (Intellectual Disability/Mental Retardation) required a full scale IQ of 70 or below. The U.S. Supreme Court in Atkins v. Virginia (536 U.S. 304 [2002]) adopted the DSM-IV TR diagnostic criteria for ID/MR but based its decision prohibiting execution of defendants convicted of capital crimes upon the presence of neurodevelopmental/behavioral, cognitive and executive functioning deficits. The Court’s theory was that people with such deficits are not as accountable for their criminal behavior as non-disabled individuals, and because of this, are not capable of learning from consequences for their behavior and thus are not able to be deterred from future criminal behavior by the death penalty. Unfortunately, the Court left establishing ID/MR definitions that conformed to its opinion to the states. To date, two states have eliminated the full scale IQ threshold. Consequently, due to lack of conforming definitions, the same defendant can be put to death in some states but not in others.

The DSM-5 diagnostic criteria for ID/MR eliminates the 70 or below IQ score threshold. The criteria now are:
A. Deficits in intellectual functions, such as reasoning, problem solving, planning, abstract thinking, judgment, academic learning and learning from experience, confirmed by both clinical assessment and individualized standardized intelligence testing.
B. Deficits in adaptive functioning that result in failure to meet developmental and socio-cultural standards for personal independence and social responsibility. Without ongoing support, the adaptive deficits limit functioning in one or more activities of daily life, such as communication, social participation, and independent living, across multiple environments, such as home, school work, and community.
C. Onset of intellectual and adaptive deficits during the developmental period.

The significance of these criteria is that the very evidence supporting a diagnosis of ND-PAE constitutes Criteria A and B of an ID/MR diagnosis.

Competency to stand trial, waive constitutional rights, and enter a plea have historically been problematic. Most persons with FASD have full scale IQ scores above 70, but prosecutors, defense counsel, and judges generally do not grasp that the deficits associated with FASD result in lack of understanding of cause and effect, actions and consequences, and appreciation that the behavior constitutes a crime. Due to DSM-5, there likely will be a significant increase in the prevalence of people with FASD also being found to have ID/MR. This will depend on states changing the law to conform to the new DSM-5 definition, state courts adopting the change in contravention of existing state law and, ultimately, whether the U.S. Supreme Court adopts the new definition.

Regarding competency, death penalty and mitigation, a fundamental condition upon which criminal responsibility reposes is that individuals have the capacity to reason right from wrong and to choose right from wrong. This perspective provides the moral justification for imposing criminal responsibility and punishment on offenders (R v. Ruzic, 153 C.C.C. 1, Supreme Court of Canada. The treatment of criminal offenders as rational, autonomous, choosing agents underlies this principle of criminal law (G Ferguson, “A Critique of Proposals to Reform Insanity Defense” [1989] 14 Queen’s L.J. at p. 140). Unfortunately, people afflicted with FASD are usually not rational,
autonomous, choosing agents, able to reason right from wrong, and able to choose right from wrong.

Some courts have concluded that since the Supreme Court ruled that FASD is a mitigating factor in capital cases, but has not addressed it in non-capital offenses, mitigation doesn’t apply to the latter. This response is illogical since it follows that if FASD constitutes mitigation in crimes of murder, it should apply equally to all criminal prosecutions. The most forceful argument for excluding defendants with FASD from the death penalty, for recognizing that special attention is needed when evaluating accountability for one’s behavior, and for treating FASD in all cases as a mitigating factor where its deficits are connected to the criminal behavior is set out in the case of Dillbeck v. State, 643 S.2d 1027 (Florida Supreme Court, 1994):

“Just as the harmful effects of alcohol on the mature brain of an adult imbiber is a matter within the common understanding, so to is the detrimental effect of this intoxicant on the delicate, evolving brain of a fetus held in utero . . . we can envision few things more certainly beyond one’s control than the drinking habits of a parent prior to one’s birth.”

With the DSM-5 now recognizing that FASD is both a medical condition and a complex mental disorder and that full scale IQ is diagnostically irrelevant in terms of degree of impairment, attention now turns to the legal community. Will state courts begin to appreciate the message in Atkins that a brain-based condition like FASD carries with it every bit as much disability as ID/MR and treat afflicted individuals differently in the justice system? The place to start is educating psychiatrists and psychologists who can now make the diagnosis, and educating the legal community so that they appreciate the implications of the diagnosis. It is our hope that these things will happen now that DSM-5 has opened the door to a modern era of FASD diagnosis.

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III. THE BIGGEST FASD GLOBAL GATEHRING - 2,350 AND STILL COUNTING
By David Gerry & Cheryl Sivertson

David Gerry
President, Insightful Marketing Inc.
BSc, Biology and Psychology, University of Victoria
After fostering children with FASD, created the FASD Community Circle in 2002, established pediatric/adult FASD assessment and diagnostic clinics, advocate for clients with FASD, supporting parenting programs and projects to provide collaborative housing.

Cheryl Sivertson
Vice President, Insightful Marketing Inc.
BSc, Computer Science, University of Victoria.
20 years programming, data and information design and analysis, teaching technology and instructional design skills. Cheryl's love of

www.nofas-uk.org
technology and passion for helping others lead to her collaboration with David Gerry

Until 2000, I had never heard of FASD. Two years prior, my wife was working as a social worker, facilitating supervised access for children in foster care with their biological parents. When another placement was breaking down for two siblings—who had seen far too many foster homes in their short lives—we were asked to step in. The intensity and frequency of these children’s emotional and physical meltdowns suggested more than just trauma related to their early life and subsequent multiple foster care placements. We went searching for answers.

REALIZING WE LIVED WITH FASD

My first epiphany arrived when I attended the annual FASD conference in Vancouver, Canada. Presentation after presentation described these two kids to the letter. When I came back home, I felt hopeful that we had an explanation for what was going on. We then went looking for appropriate support services and to obtain an assessment and diagnosis for both children. Our hopes were shattered when we learned that assessment and diagnostic services would be 33 months away and we’d have to travel outside our city. Having no choice, we pursued this path.

33 months felt like 33 years, as these kids needed immediate help (and we did, too!). Without a diagnosis, we had access to few (if any) appropriate support services (i.e., a teachers aid at school). The arduous journey to obtain a diagnosis motivated me to help other parents avoid the unnecessary trials and tribulations we faced during this time.

CREATING FASD SERVICES AND PROGRAMS

In 2001, with two partners, I started the FASD Community Circle – Victoria. Our first initiative was to create multidisciplinary assessment and diagnostic services on Vancouver Island, Canada. Along with this pediatric clinic, we created a family support position whose job was to help families directly translate our team’s clinical reports and recommendations into necessary and appropriate support services for each of the children the team diagnosed. Our family support position was later used as part of the prototype for the BC provincial government’s program called “FASD Keyworkers.” We ran regular parent support groups and, later, a specialized parent support group for mothers who self-identified as having FASD. In 2007, we also created the first multidisciplinary diagnostic and assessment clinic for women in British Columbia (BC) who suspected they might have FASD.

LEAVING AN FASD LEGACY

As the ED of a charity for 10 years that specialized in providing FASD services and resources, I had accumulated a wealth of knowledge, training, and contacts related to FASD and was determined to leave some sort of ongoing FASD legacy to continue to help parents, families, and professionals better understand and support those affected by FASD. I wasn’t quite sure how to go about sharing all that I had learned these many years. All I knew was that I needed to find some sort of vehicle to transmit this hard-earned knowledge.

The idea to create some sort of FASD gathering came in early 2012 from discussions I had with my wife, Grace, and my friend Cheryl Sivertson. If our plan was successful, and paved the way for an annual gathering, this would also create an opportunity for researchers, clinicians, professionals, service providers to connect with one another and parents. We were excited at the possibilities for:

1. **Researchers:**
   - Possibility to recruit research subjects;
   - Mechanism to make research findings relevant and accessible to families.

2. **Clinicians** (health care, psychology, doctors, occupational therapists, etc.):
   - Clinicians have a direct window into the most pressing issues families are facing;
   - Mechanism to make clinicians expertise accessible to families.

www.nofas-uk.org
3. **Professionals** (legal, social work, corrections, educators, etc.):
   - Broaden and deepen their training; awareness of the most current issues families are facing; and learning from clients and other professionals on recent effective strategies;
   - Families learn practical strategies for tackling behavioural issues (daily life and for the long run) using a brain-based approach: try differently, not harder.

4. **Parents, caregivers, family** (biological, foster, adoptive, spouse, children, siblings, etc.):
   - Inform researchers, clinicians, professionals and other service providers about most pressing challenges and gaps in service;
   - Access to: the most up-to-date research; strategies from some of the most skilled clinicians and leading professionals in the field; practical tips on how to be an effective advocate when engaging “other” service providers.

5. **Other Service Providers** (employers, community recreation centers, government, daycare, Summer camps, etc.):
   - Provides mechanism to become FASD aware;
   - Parents act as a lifelong external brain for those with FASD and, therefore, require support from a range of service providers (who are FASD-informed) across the lifespan of their child.

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**RECRUITING SPEAKERS**

One of my first tasks was to contact FASD experts I personally knew and ask them to speak to specific in-demand topics. We decided our ideal speaker would be a professional and a parent to a child with FASD. We knew these individuals would have the necessary hands-on experience, thus would understand the struggles parents face each day. In addition, their professional background would give them insider knowledge about how to be an effective advocate for children with FASD—skills they could share with our global audience. Of our 22 speakers, 17 fit this criteria. We also decided to invite three speakers who have FASD; we felt it was important for the audience to hear first person accounts of what it is like to grow up with FASD—the struggles, the triumphs, and the strategies that lead to more successful daily living. The remaining three speakers were selected because the nature of their work was practical and would be immediately useful for parents in daily life.
HOW THE SUMMIT WAS DELIVERED

Starting on September 9th, 2013, participants had the opportunity to listen live to Summit speakers via the Internet (using a web browser) or by calling into a conference line using SkypeTM or a local phone number. Some of our sessions were followed up by a live listener question and answer segment. This was an opportunity for participants to get their most pressing FASD questions answered by our experts. Questions could be asked by typing them into our web interface or by asking them in our special, closed, Facebook group called "Living With FASD". For registrants who were not able to listen live, we also offered the ability to download MP3 recordings and written transcripts of each interview and live Q&A segment.

OUTCOMES

So how did it go? 2,300+ people registered from more than 12 countries. Participants came from large, urban centres as well as remote communities in places like Alaska and South Africa. Internet access was easy for many, but in some of the rural areas the ability to phone in via a local phone number was essential (sporadic Internet capabilities).

The value of connecting participants with Summit speakers indirectly (via their interviews) and directly (live Q&A, phone, and email) cannot be overemphasized. As an example, one of our speakers is a research psychologist, Dr. Claire Coles, who presented results from an intervention study (MILES, Math Interactive Learning Experience). In her study, she focused on tailoring math teaching techniques for the unique needs of each student with FASD. To date Dr. Cole has received 15+ requests from Summit participants for follow up information so they can pursue having teachers in their local area trained in the MILE technique. Here is feedback from one Summit participant:

"Hello! I was absolutely thrilled to be able to be included in this summit!!! There was SO much very helpful information. One thing in particular that I am trying is the MILE program. I can't wait to start! I should also let you know that I wrote a letter for my 10 year old daughter from her perspective to her class. The teacher read it and immediately afterwards, my daughter had TWENTY children asking her to join them at recess! I cannot tell you what that meant, as my daughter is in grade 5 and has been a social outcast for the past 5 years. It was the best day of her life!"

As part of the Summit, we started the “Living With FASD” Facebook discussion group, the purpose of which was to create a virtual FASD support group, directly connecting people from around the world. In this way, participants could not only ask us questions (and the speaker), but could ask each other questions and share experiences, resources, and practical tips. To date this very active group has 570+ members, and continues to grow each day. People in this group are very eager to reach out, seek help, and offer tips and support to each other based on their own experience. The level of engagement in this group far exceeds any expectations we may have had in the beginning. The Internet being available to the masses has made it possible for people to connect with others, and create community, regardless of distance.

As moderators, we log into the Facebook group each day, to monitor posts and to contribute to the very active discussions. One of the ways I have been able to contribute is to help people in isolated areas connect with services. An example is a mother from a rural area of Mississippi, USA who talked about her struggles and her need for support. In preparing for this Summit, I had made contact with many national and regional groups in the USA, including the chair of an organization of state FASD coordinators. Through this connection, I was able to link this mother with a resource person in her area, who helped her obtain specialized education services for her children in a local school. It is exciting, and hopeful, to be able to make these kinds of links. Here I am living on Vancouver Island (in Canada) and I was able to connect someone from rural Mississippi with a resource in her own community!

www.nofas-uk.org
IN SUMMARY

Our intention for the Summit was to use technology to facilitate access to FASD experts and training for parents and families living with FASD. The Internet is a fantastic way to provide access to FASD services, training, and resources, and is especially helpful for those living in remote communities or communities that do not have FASD specific services. Repeatedly, we received grateful feedback:

“Thank you, thank you, thank you! I have always wanted to attend an FASD conference, however I never had the time nor the money. Right now, I am at home in my living room, looking after my child, all while being able access the valuable information each speaker is offering.”

The frequency with which we receive this kind of feedback suggests we accomplished this goal and have begun to meet parent’s unmet needs.

A second objective was to directly connect participants to researchers, professionals, and clinicians to: aid in directing new research opportunities; broaden and deepen professional understanding about; and by interacting with parents, clinicians gain insight into the most pressing issues families living with FASD are facing.

We also set the intention to provide new opportunities for people to share experiences and resources. During and since the Summit, we have been surprised and touched by the number of messages from families, who expressed huge appreciation for what a difference this Summit made to them. Their feedback was that it reduced their sense of isolation, created a vibrant community for them and gave them hope that they could get the advice, knowledge, resources and support they needed, as they parent their children with FASD.

Our hope is that the Living With FASD 2013 Summit is the first of many annual events that forms a permanent back bone of communication to grow and spread knowledge and informed action about FASD, across both continents and generations. If you would like to get your copy of the 22 Summit interviews from 2013, check out http://www.LivingWithFASD.com/nofasuk , or email David Gerry at info@LivingWithFASD.com

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IV. FETAL WELLBEING AND ITS IMPACT ON FUTURE HEALTH: SOME PERSONAL PERSPECTIVES ON A HIGHLY CONTROVERSIAL ISSUE

Based on a speech at the Royal College of Obstetrics and Gynaecology, October 17th 2013

Sir Al Aynsley-Green

Introduction
In September 2012 I was invited to Toronto, Canada to give a keynote speech at the annual meeting of the outstanding pan-Canadian NeuroDevNet organisation. Whilst there, a key afternoon session was focussed on the practical and theoretical challenges of Fetal Alcohol Spectrum Disorder. The programme covered new scientific advances including clinical and legal perspectives on biomarkers in FASD, screening through meconium samples at birth, and a public forum
attended by lawyers, lay people and families and clinical and research staff on FASD entitled ‘How much is too much?’

For me coming from England, this was a deeply thought provoking session that was introduced by a courageous adoptive family who spoke alongside their two inspirational young people to an audience of several hundred participants. The girls were 16 year-old twin sisters, adopted as infants from Russia; concerns were soon expressed over their slow developmental progress, and after referral, the Diagnosis of Fetal Alcohol Spectrum Disorder was established. The girls were able to speak movingly with confidence on what its like to have FASD and the parents powerfully told of their own searing experiences and needs. Their ‘lived experiences’ provided powerful testimony of the importance and impact of this devastating and preventable disorder on their lives.

It was clear to me from this event that the Canadians are leading the world in FASD awareness and actions to address it. As they say, it is the most important preventable cause of brain damage in childhood, with a high prevalence not only in first nation people, but also in affluent families. They claim it is the most important cause of bad behaviour in schools and communities, with high prevalence in prisons and with major implications for the family and criminal legal systems.

**Fetal Alcohol Spectrum Disorder.**
Fetal alcohol spectrum disorder (FASD) is a term that describes a wide range of effects that can occur in an individual who was exposed to alcohol during pregnancy. These effects include physical, cognitive, social and emotional and or behavioural disabilities that last a lifetime. FASD is not a diagnostic term but an umbrella under which three specific medical diagnoses can be made:

1. Fetal Alcohol Syndrome (FAS)
2. Partial Fetal Alcohol Syndrome (pFAS)
3. Alcohol Related Neurodevelopmental Disorder (ARND)

**Canadian actions to address the condition**
Theses include:

- Federal and provincial government support
- Teaching young children of the dangers of alcohol during pregnancy
- Public awareness campaigns
- Educating teachers and medical staff
- Referral routes for specialist advice
- Research programmes & publications
- International conferences & dissemination

In the Province of Alberta alone there is a 10-year Government-led FASD strategic plan, the 2013 budget for FASD-related services being $18.3m. There is a well-organised and accessible programme for service delivery through 25 assessment and diagnostic clinics and a ‘Wrap-around’ services for affected school children. This is predicated on the belief that preventing FASD in just 10 babies saves enough money to fund all these services.

I first saw the teaching of young children on the importance of not drinking during pregnancy when I made it my business to see for myself the ‘Roots of Empathy’ programme in action in Winnipeg, Manitoba. This initiative led by the inspirational Mary Gordon sets out to deliver a parenting programme for 3-14 year-old

The concept is simple: a new mother in a local community agrees to bring her newly born infant to a designated class in her local primary school once every month for the first year of the child’s life. The children witness at first hand early human development and learn about empathy by trying to understand how to communicate with a baby who cannot talk. A trained facilitator imprints key
lessons about parenting in the minds of these young children. On the day I visited, she asked them ‘What do you never drink when you have a baby in your tummy?’ ‘We never drink alcohol Miss!’ chorused the children! This illustrates the commitment to preventing FASD through early education.

The Manitoba FASD Network is supported by Manitoba Health and Healthy Living initiative, with 5 Regional Health Authorities and the Winnipeg Regional Health Authority Child Health Program.

The Manitoba FASD Centre provides a comprehensive assessment service together with research, education and training. It also provides a focus for effective advocacy not only for political traction, but also for the needs of mothers, families and affected children. Its resources include Pregnancy and FASD Prevention Services; Diagnostic Services. Outreach & Support Services for Children, Youth & Families and for Adults living with FASD alongside Mental Health Services and Information & Education. A 24-hour, 7 days-a-week helpline is available for expecting mothers. Children are referred to the Assessment Centre where they undergo a comprehensive evaluation by a team comprising a social worker, geneticist, developmental paediatrician, and a Psychiatrist.

Practically useful information is available for schools and for affected children, highlighting their isolation, fidgeting, impulsivity, distractability, loud noise intolerance and learning difficulties whilst concurrently identifying the child’s strengths and successes thereby to generate confidence and self-esteem.

The Journal of Obstetrics and Gynaecology Canada in its special issue entitled Alcohol Use and ‘pregnancy Clinical Guidelines (Vol 32: number 8, supplement 3, August 2010) offers a comprehensive ‘state of the art’ report which includes five practically-relevant clinical scenarios to assist clinicians in managing common problems they will encounter.

Canada also leads the world in providing international events to bring together from around the world experts to debate key developments and issues. The First International Conference on the Prevention of FASD was held in Edmonton, Alberta in September 2013, alongside a further event on Legal issues of FASD. The issues addressed include: Implications for the legal systems and response of the criminal justice process, the relevance to family courts, implications for guardianships and social support alongside the role of legal systems in preventing FASD and, of special significance, ethical and economic implications.

This brief analysis of the enormity of the investment and activity taking place in Canada through its political focus on the subject, the extent of the services provided for mothers, families and affected children, and the quality of its education and research programmes confirms that it does lead the world in thinking about the subject of FASD. What does this mean for England?

**FASD in England**

There are undeniable facts to argue that England has a major societal problem caused by alcohol misuse. Increased consumption, soaring cirrhosis of the liver in young adults, binge drinking, alcohol-related crime and economic consequences are the subject of endless political rhetoric and media comment.

Despite this, it is essential to understand that important trends are now being seen in young people with an overall fall in alcohol consumption. This welcome change has been accompanied, however, by a deleterious trend in increasing binge drinking. The impact of this can be seen at night on our city streets where volunteer groups including Street Pastors do their best to care for young people under the influence of alcohol, listen to them and try to help. A recent publication by the Children’s Commissioner for England has reported that 30% of children live with an adult binge drinker, and 2.5% with a harmful drinker. Moreover, there are estimated to be 175,000 young carers affected by parental alcohol and or substance misuse.

Against these worrying facts, is it not likely that FASD will impact on maternal and fetal outcomes here too? But in contrast to Canada, where is the debate, where is the activity and where is the political focus? Does it matter? Is it a problem and how big is it? How can it be identified? What should be done about it?
All these are highly relevant questions, but answering them is compounded by controversy over the prevalence and significance of FASD, and the limited research base that allow opinions to be formed and stated without hard evidence of harm. Of special public controversy is the question of whether low alcohol intake is harmful.

Dr. Neil Ayton and his team in one of the few centres in England focusing on FASD reminds us that 80-90 % of women of childbearing age drink regularly, with 25% age 18-25 and 21% 25-44 drinking more than 14 units of alcohol per week. 15-20% of women continue during pregnancy yet 23% midwives seem not to be aware of current guidelines with only 59% being comfortable asking women about alcohol.

In contrast to the high political focus on FASD, our government in its Alcohol Strategy makes only passing reference to it: ‘Fetal alcohol spectrum disorders (FASD) result from mothers drinking alcohol during pregnancy. They are lifelong conditions that can have a severe impact on individuals and their families…. They are caused entirely by drinking during pregnancy and so are completely preventable. We do not have good information about the incidence of FASD, so it is likely that significant numbers of children are not diagnosed’.

In the light of my visits to Canada, in 2012 I wrote to the Chief Medical Officer drawing her attention to what I had seen and heard. In her courteous reply, she confirmed that Government shared my concern that alcohol is a major public health issue, but comments that: ‘we believe it will be necessary to make progress in developing agreed diagnostic criteria for the various conditions causing FASD before it would be possible to make estimates of any prevalence with any confidence. She concludes by saying ‘I believe, however, that it would be sensible to await WHO’s findings and their discussion of the diagnostic criteria used before deciding how we take further work forward in this country’

On sharing these opinions with colleagues in Canada, I was asked ‘Why are you guys sleep walking to disaster?’

Despite these perhaps unhelpful comments from the country’s most senior physician, nonetheless there are some very good things that are being done at present by a small number of motivated colleagues. There are FASD voluntary support organisations that provide excellent literature resources; a Professional Consensus Statement has recently been published, there are some outstanding clinical services, for example in Brighton & Oxted, and new research is being published.

The consensus statement outlines the processes undertaken to produce the conclusions, examines the background and purpose, suggests proposed care pathways for antenatal care, perinatal care, a follow up pathway, an FASD diagnostic pathway along with facial, behavioural and developmental assessment. This statement deserves widespread dissemination.

One of the most controversial aspects of FASD is why there is so much variability in affected infants, and the safe threshold lower limit for alcohol consumption. A recent publication suggesting that ‘a couple of glasses of wine each week does no harm’ has triggered extensive and sometimes polarised opinions.

The paper by Lewis and colleagues has addressed the variability problem by analysing the outcomes of children aged 8 in terms of IQ against documented alcohol intake during pregnancy coupled with scientific measurement of the activity of four key genes that control the clearance of alcohol from the body. They showed that variability in the ability to metabolise alcohol was strongly related to lower IQ at age eight. But this effect was only seen among the children of women who were moderate drinkers. There was no effect among children whose mothers abstained during pregnancy, suggesting that it was the fetal exposure to alcohol that lead to the difference in child IQ. Heavy drinkers were not included in the study.
Conclusions
Work in Canada has exposed the enormity of the critical thinking, political focus and practical steps being taken to address the most important preventable cause of brain damage in children. In contrast to this, the condition has, in my opinion become ‘log-jammed’ in England through prejudice, opinion and scientific uncertainty coupled with a lack of political traction. Disentangling this is no mean task. Urgent research is needed to define the prevalence in UK. There is undoubted maternal anxiety that should be addressed by effective education, but Canadian experience shows how there are also very relevant human rights, ethics and legal aspects which do not seem to be considered. Early identification of risk and the potential for interventions before and during pregnancy, at birth, in childhood and for managing adults with the disorder should be priorities for clinical services.
So, what’s to be done? I conclude with some pertinent questions:
How do we get:
• professional engagement and alignment?
• Effective political advocacy?
• Dissemination of best practice?
• Regional focal points?
• Management protocols & referral pathways?
• Research?
• Public awareness?
PSHE in schools
Via local health services
This speech is given in the Royal College of Obstetrics and Gynaecology, so my ultimate challenge is what does this mean for the RCOG?

Key messages:
Raising awareness of the enormity of what is being done in Canada on FASD compared to England Need for prevalence data, research young people’s understanding, ensuring that FASD is included in PSHE curriculum with concerted advocacy & action from the Royal Colleges and professional bodies, including the RCOG

Overall Exam questions:
Why is there this mismatch between Canada and England in focus and delivery?
Is it timely to re-examine our positions?
2168 words

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V. EXCERPT FROM FASD RELATIONSHIPS: WHAT I HAVE LEARNED SO FAR...

Dr R.J. Densmore
Family doctor, adoptive father of a child with FAS, Member of the Canadian Provincial Government FASD Action Plan Committee, website: fasdrelationships.ca, drdensmore@shaw.ca

www.nofas-uk.org
I am Dr. Rod Densmore, a primary care physician. Welcome to my world! Thank you for your interest in FASD. Our family adopted a child who has Fetal Alcohol Syndrome seventeen years ago and we’ve been students of this condition ever since. I have a passion for understanding the underlying brain mechanisms of behaviour—but always with a view to how this knowledge can be applied in practical ways for my family members and patients. FASD Relationships: What I Have Learned About Fetal Alcohol Spectrum Disorders summarizes my experiences thus far.

Fetal Alcohol Spectrum Disorder (FASD) is a general term that includes the various patterns of how prenatal alcohol exposure can affect people. FASD is caused by consumption of alcohol during pregnancy. Physical, learning, social and behavioural effects of prenatal exposure to alcohol last a lifetime.

People affected by FASD can be helped. The brain, although permanently changed by prenatal exposure to alcohol and adverse early life events, can regenerate. I am more optimistic about my child’s future now than at any point in the last seventeen years. This book puts forward suggestions and strategies that have worked. There is hope.

FASD is common—although older estimates suggest a prevalence of about one per cent, recent population studies conducted in schools have found that three or four percent of the students whose parents consented to testing have FASD. Conjecture? —Yes—but isn’t it likely that the fifty percent of kids whose parents chose not to allow testing have an even higher likelihood of FASD? Furthermore, in specific populations—special education classes, homeless individuals, those in prison, children in foster care—the rate is even higher. To make progress tackling drug abuse, alcoholism, repeat criminal offences, homelessness, learning disorders, foster or adoptive parent burnout, child abuse and neglect—some of society’s most costly and heart wrenching issues—caregivers and policy makers must understand FASD. This book’s primary aim is to help inform and equip caregivers, policy makers and the professionals who work with our kids. Better prepared, with a few handy tools and strategies at our disposal, maybe we can help F A S D stand for Myles Himmelreich’s guiding values and aspirations: “Faith, Ability, Strength and Determination.” These values have helped Myles, a remarkable young man who has FASD, transcend some of FASD’s limitations—and these values are something we can all aspire to.

"Chemistry" of FASD Relationships

Dan Dubovsky is a social worker and FASD expert who works for the Substance Abuse and Mental Health Services Administration (SAMHSA) in Maryland, USA. Dan is also an adoptive parent of Bill, a young man with FASD who was tragically killed in a car accident several years ago. Dan has created the acronym NURMU (noncompliant, uncooperative, resistant, manipulative, unmotivated) to describe how our kids are often viewed by adults that work with them (including, I am ashamed to say, on occasion, parents and physicians like me!)

I will add a rather cheeky acronym, PTHD (PMS, tired, hungry, depleted), which describes the internal state of caregivers, again including me! Before scolding me for talking about Pre-menstrual Syndrome (PMS) in the first part of this book please be aware I have four daughters and a wife and have lived with them through a total of one hundred twenty “girl-years” so, in-so-far as a man can talk about PMS, I consider myself at least “experienced”. Besides, I think men can fulfill diagnostic criteria for PMS too; my symptoms usually appear after being “on call” for a weekend (Post too Much work Syndrome) and after meetings with particularly odious colleagues (Post Meeting Syndrome.)

If the “catalyst” of righteous indignation (e.g. “I’ve told him a hundred times not to steal money from my wallet! —and he’s not a little kid anymore, he is 15! I should not have to keep a lock on my bedroom door when my kid is 15!”) is added to the toxic duo (of seeing kids as NURMU and being
in a state of PTHD) then the inevitable result is what our kids will feel as an attack. This attack could be as minor as a sigh of frustration or a look of exasperation. Caregivers are often “certain” that they are not angry. (But subjective self-assessment of how angry you are is spectacularly inaccurate!) Kids who have FASD, despite being less responsive in many areas, often have hyper-responsive “criticism detectors” (not unlike their parents—again, I am guilty!), so productive social exchanges and civility can be quickly lost in an interpersonal cycle of perceived rejection, negativity and detachment following what caregivers view as legitimate correction, redirection or discipline. If parents’ or teachers’ corrective efforts are not received, then their goal (to help kids not repeat similar mistakes) cannot be accomplished. Getting angrier will certainly not help kids understand more clearly. Curiosity can re-direct this angry response—“why ever did that kid steal the i-Pod?”

I do not know of a more potent force than insatiable curiosity to maintain attachments and preserve relationships with those affected by FASD. Yet this curiosity, this life-blood of maintaining relationships, can seem a distant mirage to depleted mums and dads who are weary from lack of sleep, emotionally exhausted and feeling that they have absolutely nothing more to give. I have experienced this feeling. However, because of their disability, many people with FASD depend even more than the rest of us on supportive, secure relationships with caring adults to help their development and maintain optimal functioning in the world. Creating and maintaining these relationships must be a main priority of all those who work with people (of all ages) who have FASD. Only through the communication channel that comes from a vital, caring relationship can a caregiver have any hope of being able to influence and help a child with FASD.

Chemistry of FASD Relationships: Figure 1.

FINALLY...SOME USEFUL ACRONYMS

“NURMU”
Noncompliant
Uncooperative
Resistant
Manipulative
Unmotivated
(Dubovsky, Dan)

“PTHD”
PMS
Tired
Hungry
Depleted
(Rod)

![Diagram](http://example.com/diagram.png)

**NURMU** Never before Rule out and Treat:
*ADHD
*Sensory Processing
*Learning Disabilities
*Attachment

Figure 1. Notes: If we view kids as NURMU, especially if we are a bit PTHD ourselves, and work ourselves up with righteous indignation, then an all-too-common result is the child perceiving an attack and withdrawing from the relationship. Caregivers able to question, “where did that behaviour come from?” or take the position “I have no idea what to do but I’ll try to at least keep my lines of connection and communication with my kid open” can possibly avoid this destructive attack/withdrawal situation. Vibrant, effective relationships between caregivers and their children...
are the most important factor in optimal development for all children, according to Vancouver, B.C., Canada psychologist, Gordon Neufeld. Kids with undiagnosed and/or untreated ADHD, FASD, sensory processing difficulties, learning disorders, attachment issues, abuse, or post-traumatic stress disorder (PTSD) might look like they are NURMU, but NURMU will disappear with correct diagnosis and appropriate interventions.

A New Word for you: Neuroinflammation (brain inflammation): How people with FASD are at increased risk of health problems because of this issue, but how hope is possible also.

My friends Don and Val Massey, neuropsychologists who practice in Edmonton, Alberta, Canada observed that a subset of their patients who have FASD showed signs of progressive deterioration of their thinking processes or cognitive decline as young adults (almost like the early-onset dementia seen in patients who have Down Syndrome). (Massey D-V, 2009) Don and Val’s observation is both a scare and a valuable “heads-up” for caregivers and organizations that provide services for people that have FASD.

I wondered what the cause of the cognitive decline could be and if interventions were possible. In 2012 I presented a workshop called Neuroinflammation: Can this Potential Cause of Cognitive Decline be Reversed in Individuals with FASD? at the Biennial Conference on Adolescents and Adults with Fetal Alcohol Spectrum Disorder held in Vancouver. (Densmore RJ, 2012) I’ll describe some parts of that workshop along some more recent information that I believe will be of general interest for all concerned about FASD.

Neuroinflammation means excessive inflammation caused, in part, by an accumulation of proinflammatory chemicals in the brain. We may have heard about the benefits of anti-inflammatory activity from, for example, certain foods such as olive oil or fish. Proinflammatory chemicals do the opposite, increasing cell damage by a number of mechanisms. “It is now well documented that neuroinflammation is actively involved in neurological diseases and disorders, like Alzheimer’s Disease (AD), amyotrophic lateral sclerosis, depression, epilepsy, multiple sclerosis and Parkinson's disease,” says Dr. P. Eikelenboom. (Eikelenboom P, 2006.)

As a caregiver of a young adult who has FASD, my main question is if we can stop or reverse progressive neuroinflammation-related dementia. Of course research about brain inflammation is ongoing. We’ll know a lot more in five years, but we know enough now to take some initial action steps to help prevent progressive cognitive decline in those we love and care for.

Psychologist Kathleen Kendall-Tackett has edited The Psychoneuroimmunology of Chronic Disease: Exploring the Links Between Inflammation, Stress, and Illness. (Kendall-Tackett K, 2010) This book provides an excellent introduction to neuroinflammation for readers with training in health sciences. Yes, as the title suggests, there are big words in this book! And these words and concepts are challenging for me as a doctor to get my mind around…but...before you tune out because of big words please realize that in the next decade, because of research done on neuroinflammation, for some patients we’ll have new ways to intervene and slow progression of or even reverse dementia and a variety of other physical and mental health problems including bipolar disease and depression. I’m 57. Medical school was a long time ago. My instructors at medical school didn’t teach about neuroinflammation. The term was not invented yet. But if I can learn some good ideas that’ll help my kid avoid anticipatable health catastrophes then the pain of having to stretch my tired old brain around new words and concepts will be more than worth it. So, if you are game, dear reader, I’ll try my best to explain some concepts about neuroinflammation in plain language (except for one paragraph that I will clearly label as “doctor-speak” lingo so you can safely skip past it!) Betcha you just scanned down the page to look at it, didn’t you?

If you have a horrible virus, bacteria or even worse, a cancer cell that has invaded, say, your lung—you want it outta there right now! You want the strongest immune response possible. Luckily, we all have some really bad-ass, nasty “thugs” in our immune system that fight these
invaders off but don’t hurt us too much (most of the time). For example, a type of white blood cell called a T Killer Lymphocyte chemically grabs onto an invading cancer cell or virus and signals for reinforcements such as the formidable monocyte (another type of white blood cell) to join the fight. Monocytes take no prisoners! They engulf and eat invading viruses and cancer cells. Thank God they are usually “on our side!

I don’t want to use too much medical terminology, but I would like readers to understand that several inflammatory cytokines tend to be very active “players” in the development of Alzheimer Disease. Donna Wilcock, working in Lexington, Kentucky, USA, found that in both Alzheimer Disease and the early dementia of Down Syndrome many inflammatory cytokines were altered (but most commonly IL-1beta, IL-6, TNF-alpha and TGF-beta). (Wilcock, 2012) IL-1beta stands for interleukin-1beta, IL-6 is interleukin-6, TNF-alpha is tumor necrosis factor-alpha and TGF-beta is transforming growth factor-beta. Don’t worry about remembering the names of these inflammatory cytokines unless you really want to surprise your grandma but you’ll see why I mention them shortly. Dementia is not simply a result of excessively high levels of these inflammatory chemicals in the brain. Rather, damage relates to abnormally high or low activity of inflammatory chemicals in various parts of the brain.

Fetal brain immune cells called microglia increase their production of inflammatory cytokines when exposed to alcohol. (Yes! These are exactly the same inflammatory cytokines that cause some parts of Alzheimer Disease and early-onset dementia of Down Syndrome…those inflammation chemicals with the big long names—IL-1beta, IL-6 and TNF-alpha) (Boyadjieva NI, 2010) (Sarkar DK, 2006) In FASD, blood vessels that pass through brain tissue are “leakier” than they should be. In part, this leakiness is caused by abnormalities in a chemical called the Shh molecule that acts as a kind of “caulking” between the endothelial cells that line our blood vessels. One consequence of leaky brain blood vessel lining cells is that immune system inflammatory cells and chemicals that are too powerful to be “allowed” in the brain “sneak” into brain tissues through the leaks.

In “doctor-speak:” In FASD the blood-brain barrier, (BBB), that protects the brain from excessive inflammation by blocking passage of many types of inflammatory blood cells and molecules is compromised. The BBB is weakened because by reduced astrocyte-mediated secretion of Sonic Hedgehog glycoprotein (Shh molecule). The Shh molecule binds to and activates Hedgehog receptors. Hedgehog receptor activity reduces inflammation. Adequate hedgehog receptor activity decreases production of inflammatory chemicals (including cytokines) and increases adhesion between the endothelial endothelial cells that line brain capillaries (thus reducing migration of inflammatory leukocytes into the brain).

In “plain-speak:” In FASD bad stuff, including inflammation cells and chemicals with a whole bunch of weird, long names, leak into parts of the brain where they shouldn’t be and cause damage. Some of these inflammation chemicals are exactly the same as the ones that play a role in the development of Alzheimer Disease so it’d be hard to come up with any logical reason why these same chemicals would not at least be partly related to the heartbreaking situation Don and Val Massey discovered when they saw some of their patients with FASD develop dementia as young adults.

Interventions to reduce neuroinflammation

If we agree overexposure to inflammatory chemicals is a harmful threat to brain health, we can do two things to reduce harm. First, reduce production of excessive levels of inflammatory chemicals. Secondly, do what we can to “get rid” of inflammatory chemicals that are already produced.

So what causes high levels of inflammatory chemicals to be produced?

An incomplete list of causes of, or conditions associated with increased production of inflammatory cytokines includes:
1) exposure to high levels of and/or prolonged stress, especially shaming or shunning (Goleman D, 2006)

2) traumatic brain injury (concussions, etc.), (Phan N, 2011)

3) exposure to alcohol including prenatally, (Zhang X, 2011)

4) mental health issues including schizophrenia, bipolar disease, and many types of depression (Taylor, 2012)

5) Autism (Rossignol DA, 2011.)

6) obesity (Taylor, 2012)


8) genetics that might predispose to damage to the blood-brain barrier—please see paragraph directly below

Damage to the blood-brain barrier allows excessive amounts of potentially toxic inflammatory chemicals to more easily leak into the brain and directly harm nerve cells. Specific genes that control several basic biochemistry pathways seem to be different in FASD. (Chudley A, 2010) Some of these gene expression differences may prove to underlie the very developmental differences that cause FASD. One altered biochemical pathway in FASD, that we have already discussed above as a factor contributing to increased brain vulnerability to inflammatory chemicals, is reduced production of the Shh molecule. (Lombard Z, 2007) Prenatal alcohol exposure also seems to influence genes that control production of TGF-beta, a chemical that regulates nerve cell growth and migration. (Chudley A, 2010) TGF-beta is one of the inflammatory chemicals we previously discussed that, if dysregulated, plays a role in causation of Alzheimer disease. (Wilcock, 2012)

For this excerpt i will include comments on just one of these causes of excessive neuroinflammatory chemicals-mental health issues. I am not a mental health epidemiologist or geneticist so I cannot provide definitive advice on how to reduce the effects of all the risk situations associated with increased levels of inflammatory cytokines that we described above. However, I will offer some suggestions, to at least start off a discussion about what we can do to reduce the risk of producing excessive levels of inflammatory cytokines:

4) To reduce mental health issues including schizophrenia, bipolar disease, and depression, attention to correctible underlying issues such as unsafe housing, inadequate sleep and nutrition needs to be maximized. For instance, since bipolar disease is 70%+ heritable, we need to take the 30% that we can control environmentally and “exploit” that 30% for all we are worth! How some of the families I work with (who have numerous family members with bipolar disease) have changed their environment includes moving schools so their kid does not grow up in an area where drug use and gangs are more common, using high dose omega 3’s, setting a high priority on regular bedtimes and waking times, setting a priority on getting a balanced diet and lots of exercise, planning summer activities so there is not a lot of unplanned screen time, but there are lots of physical activities and chances to explore creative activities like music, art, and dance and creation of opportunities to link up with extended family such as their uncle that likes to take the kids fishing.

How can we reduce the activity of inflammation chemicals once they have been produced? One priority is not to harm the effective processes we all have in our bodies that break down and eliminate these stress chemicals.

1) Good restorative sleep can reduce inflammatory cytokines by 70% according to Kathleen Kendall-Tackett. (2010) What she means by restorative sleep is enough slow wave and rapid eye
movement (REM) sleep. These are the “deepest” parts of sleep. If dangerous housing forces you to “sleep with one eye open” deep sleep is impossible. New mums always sleep with one eye open! Dads and other helpers need to give them the afternoon off so they can nap…yes—and new mums have to let dads and other caregivers go for walks with their little ones so they can catch up on sleep. (I Hope I don’t get into trouble for that one!) Even “skinny” folks with FASD are at increased risk of having obstructive sleep apnea—this condition leads to obstructed breathing especially at deep stages of sleep. Thus, the percentage of the night in deep sleep is reduced and breakdown and elimination of inflammation chemicals is impaired. Doctors can easily diagnose sleep apnea. After treatment, many patients report weight loss without trying to eat less, better energy, better attention and learning, and less depression. Doctors get all excited about diagnosing and treating sleep apnea because we know that treatment will reduce heart rhythm problems including sudden death. Further, enough slow wave sleep has been shown to reduce depression symptoms. In FASD, restless legs are also a common cause of reduced deep sleep. If this condition is suspected (if your kid kicks off all the blankets and if you need to wear hockey goalie equipment in order to safely share a tent with her on camping trips) be sure to ask your doctor to rule out restless legs syndrome and make sure blood levels including iron (ferritin) are checked.

2) Exercise breaks down and eliminates stress chemicals…yes, your Uncle Joe who told you to “work out that stress” had a good idea.

3) Omega 3’s I first became interested in omega 3’s when my favorite Canadian psychiatrist (favorite because he is so inquisitive and “thinks outside the box”), Trevor Young, discussed his research. Trevor developed a rat model for bipolar disease. He found out that as the membrane around nerve cell mitochondria (the little energy factories inside most cells) progressively deteriorated, bipolar symptoms got worse. Omega 3’s reduced the progressive deterioration of mitochondrial membranes…and improved bipolar symptoms. (Yong LT, 2008) Cool, eh? Kathleen Kendall-Tackett discusses omega 3’s throughout her book. (Kendall-Tackett, 2010) Nothing is a panacea. Recently, I think on the radio, I heard that high intake of omega 3’s might be linked to prostate cancer, but I tried to verify that concern by talking with a variety of my colleagues and pharmacists at our hospitals and, after researching the question, no one could back up the concern. It would be prudent to keep aware of updates on safety of omega 3’s but for now I will share what I know. Most of these recommendations are based on Kendall-Tackett’s book.

Omega 3’s are potent anti-inflammatory agents. They reduce inflammatory chemicals such as cytokines. Omega 3’s consist of EPA, DHA and ALA. I won’t bore you with the big long chemical names. ALA comes from flax seeds which is a nice source but ALA is only a ninth as active in the body as DHA or EPA so to accomplish what you want—which is an anti-inflammatory effect—you need nine times the dose if you are using ALA. EPA and DHA usually come from fish. Some people complain they smell like a fish when they take these…if that is you then sometimes krill-sourced omega 3’s can help.

What is the difference between DHA and EPA? Think D for “Dopey” (DHA is a bit sedating) and E for “Excited” (EPA is a bit alerting). One resourceful mum, armed with this information, found a high percent EPA preparation to give her kid in the morning when he was dopey and a high percent DHA preparation to give him at night when he was “bouncing off the walls.” She still finds this useful five years later. An anti-inflammatory effect from EPA and DHA can be found with doses in the 3000-4000mg range per day. (Kendall-Tackett K, 2010. p 92) EPA helps mood. To help mood, the optimal dose of EPA is 2000mg per day for most adults. I have a small number of patients who felt a bit worse at 2000mg of EPA per day but when they tapered to 1500mg of EPA per day they felt better than when they were not using any EPA. We used to think DHA did not have a mood effect but recently—I think at the June 2013 Canadian Psychiatric Association Vancouver meeting—I’d learned that DHA seems to reduce suicidal thoughts.

Be aware that no one would advocate omega 3’s as the sole treatment for severe depression especially if associated with suicidal thoughts…but omega 3’s are useful adjunctive measures. By the way, after a review of the evidence, the 2012 Canadian Psychiatric Association Clinical Practice Guidelines for Treatment of bipolar disease now list omega 3’s in anti-inflammatory doses (3000 to 4000 mg per day of the combination of EPA plus DHA) as one of a number of recommended treatment options for treatment of stabilized bipolar disease (but not for an acute
flare up of mania). So that’s interesting, eh? Omega 3’s are “evidence based medicine” these days. I wonder what possible rationale is followed when government drug plan coverages still exclude them? Sigh.

If you decide to go shopping for omega 3’s, beware! If you believe Dr Andy Weil, an integrative medicine specialist from U of Arizona, you’ll walk past the omega 3-6-9 pills and mutter comments that you’d rather not repeat in church beneath your breath because Dr Weil explains that omega 6 and 9 are already excessive in most western diets and are PRO not ANT inflammatory. Next, you want to see how much EPA and DHA is in each pill. Take out your magnifying glass! I believe that miscreants design labels for omega 3’s. Just look at the bottles. “1000 mg” will be featured prominently…remember, all you care about is the number of milligrams (mg) of EPA (you want it around 2000 mg per day) and the number of milligrams of DHA (you want it around 1500 mg per day). I bet you’ll find “1000 mg” does not pertain to EPA or DHA or anything the pill manufacturer cares to describe in the fine print.

Get out your magnifying glass now and read the side of the label…your single concern is how many pills it’ll take to get about 2000mg of EPA plus about 1500mg of DHA which will total about 3000-4000mg per day. That’s all you care about. Ignore the rest. For teens about 3000 mg per day and for 30 kilogram 7 yr.-olds about 800-1000mg of the combination of EPA plus DHA are reasonable doses.

Now an interesting patient’s story for you: a man I see who has a very severe intellectual disability was constantly picking at his colostomy, causing infections of his skin and driving his poor nurses nuts with concern. No one knows why he has an intellectual disability—if he were a little boy today the pediatrician would organize a comprehensive genetic work-up and search for the cause of his profound difficulties. (His IQ is 55, and his age is 58) Maybe he’d be found to have Fragile X syndrome…that is the most common genetic issue that leads to profound intellectual disabilities in my area. However, he has a flat philtrum (the groove between his lips and nose is quite flat), a thin upper lip, small eyes, and he’s very short, so I always wondered if he could, perhaps in addition to Fragile X, have FASD. Because the potent brain anti-inflammatory effect of minocycline (a common antibiotic) is an investigative therapy for helping to improve the developmental trajectory for kids with Fragile X (Vanesh H, 2013) I suggested his nurses try omega3’s as a low-risk anti-inflammatory strategy. They started about 2500mg of the combination of EPA and DHA per day. A month later, they said, “for the first time, he is appropriately responding to us, we can actually talk to him now.” Since we seemed to be on the right track, we decided to “max out” anti-inflammation strategies by increasing the omega 3’s to 4000 mg per day and added atorvastatin, minocycline, alendronate, and low dose Aspirin. Next month, we’ll see if these measures have helped further.

Did anti-inflammatory strategies help him quit picking at his colostomy and driving his poor caregivers nuts? Not much, unfortunately, but we were able to decrease picking by adding a medication often used for obsessive-compulsive problems, topiramate. Why am I describing this patient’s situation? Because even this patient with profound brain damage could improve if treated with anti-inflammatory strategies.

I’ve also seen omega 3’s improve depression in a young lady (who had not achieved remission of her depression symptoms despite a walking programme, several types of antidepressants, and numerous visits over several years to supportive psychiatrists that she likes.) This young lady has FASD. She is “clean” now for three years but was addicted to crack cocaine and methamphetamine (crystal meth) for almost a decade. She also sustained several severe concussions from assaults. She thought her mood was improving, she was getting out more frequently, and her score on the PHQ-9 (Depression symptom inventory) improved from the "severe" depression to the "mild" depression range after six weeks of 3500mg of the combination of EPA plus DHA per day. She’s pleased. We did not discontinue her other anti-depression medications.

Skeptics will say these are anecdotal case reports and I agree, but they are pretty cool, eh? To be fair, though, skeptics should read Kendall-Tackett’s book, Trevor Young’s research and the

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Canadian Psychiatric Association’s Guidelines on treatment of bipolar disease before they erroneously pronounce that omega 3’s not being evidence-based medicine. Omega 3’s are evidence-based medicine. You skeptics are like soo “yesterday!” Touché!

4) Medications  My advice, if you are interested in any medications that I will mention, is to discuss them with your health care provider. No medication should ever be taken without a weighing of potential benefits of the treatment versus possible side effects. Remember, though, that there is a high, and, at times, an unacceptably high cost to doing nothing to treat many conditions. Take depression, for example. We know that untreated depression progressively harms the very parts of our brain that allow us to learn new things, soothe ourselves (hippocampus) plan and weigh consequences (frontal cortex). Not treating depression is a dumb choice. Untreated depression will cause progressive brain damage. MRI’s show progressive decreases in the size of the hippocampus and prefrontal cortex in undertreated depression. Go ahead and have a good rant about evils of pills and side effects if you need to but for heaven’s sake even if you want to avoid pharmaceuticals, if you have depression, participate in evidence-based treatments such as adhering to an exercise programme, going to good counseling, making sure you don’t have sleep apnea, using a C-PAP mask if you do have sleep apnea, making sure you don’t have restless legs syndrome and correcting your low iron if that is contributing to restless legs, getting a blood test to make sure you are not hypothyroid or low in vitamin D or vitamin B12 or magnesium, and consider reasonable alternative strategies such as St John’s Wort (a good choice for treatment of mild or moderate depression but not adequate for severe depression, as I understand.) I’d add omega 3’s to this list also. And if after a month or two you were not getting better—change something, get more suggestions…don’t leave severe symptoms unresolved because of the ongoing brain damage that is caused by undertreated mental health issues.

If you are interested in pharmaceutical medications, here are a few ideas that help decrease inflammation. Two antidepressants, mirtazepine and trazodone increase slow wave sleep. Slow wave sleep is what we all really care about...because that's the part of sleep that—if you get enough of it—helps you decrease depression symptoms in part by breaking down and eliminating inflammatory chemicals from your system. Mirtazepine can be associated with weight gain, I think, in about 15% of people using the medication, so weight needs to be carefully monitored.

Minocycline is an antibiotic that turns out to have brain anti-inflammatory effects. If you happen to be young person with miserable acne, minocycline is a common recommendation for the skin problem. It is also used for rosacea in older folks. I prescribe minocycline for about a dozen patients in my practice who are younger, with bad acne or older, with rosacea and who also have significant mental health and cognitive issues such as bipolar disease and/or FASD. The man who used to pick at his colostomy that I described above is the first person I’ve suggested minocycline for that does not also have a skin infection problem. Ophthalmologists use minocycline as a potent antiinflammatory medication for patients with retinal problems. Dr. Kendall-Tackett’s book (Kendall-Tackett K, 2010 p 172) described investigative use of minocycline in multiple sclerosis (MS). Minocycline can penetrate into the brain and reduce production of inflammatory cytokines by microglial cells. (Microglial cells are part of the brain’s immune system.)

5) Diet  For years now, Dr. Andy Weil has been advocating an anti-inflammatory diet. (Weil A, 2006) Based on lots of fruits and vegetables, small portions of (complex) carbohydrates and modest sized servings of fish and meat along with liquid oils such as olive oil, Dr. Weil’s suggestions are similar to “The Mediterranean Diet.” Andy believes eating this way reduces pro-inflammatory chemicals and increases anti-inflammatory chemicals in the body. Interested readers could learn more by perusing his website: http://www.drweil.com/

Punch Lines regarding neuroinflammation

The brain should be an oasis—it should be protected from some of the nastiest, harshest parts of the immune system. In FASD, the brain is less protected. Harsh inflammatory chemicals have more access to the brain in FASD and these chemicals are the same as those that, to some extent, cause Alzheimer’s.
Take home points: because the brain in FASD is at higher than average risk of being harmed by inflammation chemicals, society should help people with FASD take steps to reduce excessive brain inflammation. Many of my patients do not have enough money to eat adequately. They cannot afford to follow the guidelines offered in The Canada Food Guide, let alone eat the foods and supplements included in Dr. Weil's anti-inflammatory diet. They eat a lot of carbohydrates because that’s what the food bank gives them. Such a diet is pro-inflammatory. I can prescribe many different types of meds for my patients because these are covered by drug plans but currently omega 3’s are not covered.

So, what’s my kid who has FASD doing about anti-inflammatory strategies? My “kid” is now a 25 yr.-old adult. My child has used 2000-3300mg of omega 3’s daily for several years, walks everywhere (sometimes a couple of hours a day), and, thank goodness, seems to sleep very soundly. Last week my kid was delighted to have passed a detailed exam called "Food safe." For several years regular employment as a custodian at a cleaning company worked well, but recently my child left that job because the boss said things about FASD that were put-downs. I’m glad that job is finished. If I’d known about the boss’s stigmatizing remarks I would have intervened earlier. I hope a similar job, i.e. one that includes predictable routines of physical activities can be found, but with a more appropriate boss. There is never a dull day and seldom is there a predictable day, but, knock on wood, at present, my kid’s thriving.

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1. VISUAL OUTCOME IN INFANTS BORN TO DRUG-MISUSING MOTHERS PRESCRIBED METHADONE IN PREGNANCY
Neonatal Unit, Royal Hospital for Sick Children, Glasgow, UK.

ABSTRACT
Background: Flash visual evoked potentials (VEPs) were abnormal in a cohort of 100 neonates exposed to maintenance methadone in utero. This prospective cohort study now describes clinical visual and electrophysiological outcomes at 6 months.

Methods: Visual assessment included modified Atkinson test battery; strabismus, nystagmus, reduced visual acuity, delayed visual maturation or refractive error (>3 dioptres) defined a fail. Pattern-onset VEPs were recorded to 120', 60' and 15' checks.

Results: 81 drug-exposed and 26 comparison infants (79% and 52% of the original cohorts) were assessed at a median age of 27 weeks (range 26-30). 90% of drug-exposed infants had been additionally exposed to illicit drugs and 41% to excess alcohol in utero. 40% of the drug-exposed cohort failed clinical visual assessment: the relative risk of abnormal assessment was 5.1 (95% CI 1.3 to 20; p=0.02). Nystagmus was particularly common. VEP peak times were slower and amplitudes smaller in drug-exposed infants, of whom 70% had one or more abnormal VEP parameter. Abnormal visual outcome at 6 months was not associated with the pattern of additional drug exposure or a history of neonatal abstinence.

Conclusions: Abnormal visual electrophysiology in infants born to drug-misusing mothers prescribed maintenance methadone persists to 6 months of age, and is associated with abnormal clinical visual assessment.


2. VALIDATION OF THE FETAL ALCOHOL SPECTRUM DISORDER (FASD) 4-DIGIT DIAGNOSTIC CODE
Susan J Astley

ABSTRACT
Background: The fetal alcohol spectrum disorder (FASD) 4-Digit Diagnostic Code has been used by interdisciplinary diagnostic teams worldwide for 17 years. It was created to improve the ease, accuracy, and reproducibility of diagnoses across the full spectrum of FASD. Over the years, a number of FAS/D diagnostic guidelines have been proposed. As the field of FASD moves forward, it will be important to adopt a single set of diagnostic guidelines worldwide. To achieve this, the performance (validity) of current diagnostic guidelines must be rigorously assessed and reported.

Objective: To summarize the body of evidence that has amassed over 20 years that validates the performance of the FASD 4-Digit Diagnostic Code.

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**Methods:** The evidence validating the 4-Digit Code is documented across 35 studies published between 1992 and 2012, including new information presented in this report. These studies and data sources include the delineation of the FAS facial phenotype; creation of the 4-Digit Code (1997-2004); our 10-year, foster-care FAS screening program; our MRI/fMRI/MRS studies; analysis of 2,550 individuals evaluated for FASD over 20 years in the WA State FASDPN clinics, and analysis of 622 patient satisfaction/follow-up surveys; surveys of 10,000 professionals attending the University of Washington FASD diagnostic clinic trainings; and surveys of over 700 professionals worldwide who completed the 4-Digit Code Online Course.

**Conclusion:** The 4-Digit Code is a simple, comprehensive, evidence-based, validated diagnostic system. It has served as the cornerstone of a fully integrated FASD screening, diagnostic, intervention, prevention, and surveillance program in Washington State for the past 20 years.

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3. **PREGNANCY PLANNING AND LIFESTYLE PRIOR TO CONCEPTION AND DURING EARLY PREGNANCY AMONG DANISH WOMEN**

Backhausen MG, Ekstrand M, Tydén T, Magnussen BK, Shawe J, Stern J, Hegaard HK.
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**ABSTRACT**

**Objective:** To investigate the extent to which Danish women attending antenatal care plan their pregnancies and to determine the association between pregnancy planning and the intake of folic acid, alcohol consumption and smoking habits prior to conception and before the 16th week of gestation.

**Methods:** A cross-sectional survey of 258 women. Main outcome measures: intake of folic acid, alcohol consumption and smoking. Pregnancy planning was assessed by the London Measure of Unplanned Pregnancy (LMUP) and the five graded Swedish Pregnancy Planning Scale.

**Results:** Most (77%) of the participants reported that their pregnancies were very or fairly well planned. Higher median LMUP scores were observed in women taking folic acid (p < 0.001), in those consuming less alcohol, and in women who stopped smoking prior to pregnancy (p = 0.043). However, 43% of the respondents with a high degree of pregnancy planning and 98% of those with a low degree of planning had not taken folic acid prior to pregnancy. Binge drinking during early pregnancy was reported by 20% of women with a high degree of planned pregnancy and 31% of those with a low degree (p = 0.1).

**Conclusion:** Pregnancy planning was associated with a healthier lifestyle but still many women could improve their lifestyle in connection to pregnancy. Their level of alcohol consumption is higher than that recommended for best pregnancy outcome.


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4. PRENATAL EXPOSURE TO ALCOHOL, AND GENDER DIFFERENCES ON CHILD MENTAL HEALTH AT AGE SEVEN YEARS
Niclasen J, Nybo Andersen AM, Teasdale TW, Strandberg-Larsen K.
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ABSTRACT
Background: It remains uncertain whether exposure to lower doses of alcohol is damaging to the developing fetus. The present study aimed to investigate associations for boys and girls between prenatal exposure to binge drinking and lower doses of alcohol in pregnancy, and parent-reported behavioural and emotional development at age seven.

Methods: This study used data from the Danish National Birth Cohort. Associations between cumulated alcohol exposure and binge drinking from full pregnancy and parent scores on the Strengths and Difficulties Questionnaire (SDQ) measured at age seven were investigated. The SDQ was used as continuous externalising/internalising scores, and as above/below cut-off for the specific scales of hyperactivity/inattention, conduct, emotional and peer problems. Inclusion criteria were information on alcohol exposure from three interviews, SDQ scores at age seven and being born full term (n=37 152).

Results: Controlling for relevant confounders, small positive associations were observed between binge drinking and internalising (relative change in mean: 1.04-1.06), externalising scores (relative change in mean: 1.01-1.07), and conduct scores (OR 1.12 to 1.23) for boys. No associations were observed with lower doses of alcohol.

Conclusions: Exposure to binge drinking is weakly associated with impaired behavioural and emotional development measured at age seven. Large differences in background characteristics were observed between the groups defined by cumulated alcohol exposure, leaving the interpretations of findings uncertain.


5. RESCUE OF HOLOPROSENCEPHALY IN FETAL ALCOHOL-EXPOSED CDON MUTANT MICE BY REDUCED GENE DOSAGE OF PTCH1
Hong M, Krauss RS.
Department of Developmental and Regenerative Biology, Icahn School of Medicine at Mount Sinai, New York, New York, United States of America.

ABSTRACT
Holoprosencephaly (HPE) is a commonly occurring developmental defect in which midline patterning of the forebrain and midface is disrupted. Sonic hedgehog (SHH) signaling is required during multiple stages of rostroventral midline development, and heterozygous mutations in SHH pathway components are associated with HPE. However, clinical presentation of HPE is highly variable, and carriers of heterozygous mutations often lack apparent defects. It is therefore thought that such mutations must interact with more common modifiers, genetic and/or environmental. We have modeled this scenario in mice. Cdon mutant mice have a largely subthreshold defect in SHH signaling, rendering them sensitive to
a wide spectrum of HPE phenotypes by additional hits that are themselves insufficient to produce HPE, including transient in utero exposure to ethanol. These variable HPE phenotypes may arise in embryos that fail to reach a threshold level of SHH signaling at a specific developmental stage. To provide evidence for this possibility, here we tested the effect of removing one copy of the negative regulator Ptch1 from Cdon(-/-) embryos and compared their response to ethanol with that of Cdon(-/-);Ptch1(+/+) embryos. Ptch1 heterozygosity decreased the penetrance of HPE in this system by >75%. The major effect of reduced Ptch1 gene dosage was on penetrance, as those Cdon(-/-);Ptch1(+-) embryos that displayed HPE did not show major differences in phenotype from Cdon(-/-);Ptch1(+/+) embryos with ethanol-induced HPE. Our findings are consistent with the notion that even in an etiologically complex model of HPE, the level of SHH pathway activity is rate-limiting. Furthermore, the clinical outcome of an individual carrying a SHH pathway mutation will likely reflect the sum effect of both deleterious and protective modifier alleles and their interaction with non-genetic risk factors like fetal alcohol exposure.

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STRAIN DEPENDENT NEUROCHEMICAL CHANGES INDUCED BY EMBRYONIC ALCOHOL EXPOSURE IN ZEBRAFISH
Mahabir S, Chatterjee D, Gerlai R.
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ABSTRACT
Fetal Alcohol Spectrum Disorder (FASD) is a preventable disease of the child resulting from alcohol (ethanol) consumption by pregnant women. Despite being preventable, FASD represents a prevalent problem throughout the world. Embryonic alcohol induced abnormalities in behavioral responses to social stimuli have been shown in humans and zebrafish. The neurobiological mechanisms underlying the abnormalities remain obscured. Here we start a mechanistic analysis by investigating the effect of embryonic alcohol exposure on the neurochemistry of zebrafish. The differing severity of symptoms seen in FASD may be partially due to genetic factors. To explore such genetic effects, here we analyzed two distinct zebrafish strains: AB and TU. Zebrafish were exposed to one of the following concentrations of alcohol, 0.00%, 0.25%, 0.50%, 0.75%, or 1.00% (vol/vol %) at 24 hours post-fertilization (hpf) for 2h. From whole brain extracts we analyzed the amount of neurotransmitters dopamine and serotonin and their metabolites across 4 different developmental time points: 15, 40, 70 and 102 days post-fertilization (dpf) using high performance liquid chromatography (HPLC). AB zebrafish exhibited a significant dose dependent embryonic alcohol exposure effect which increased in robustness with age. However, TU showed no such concentration effect: the levels of neurochemicals remained mainly unaltered by embryonic alcohol exposure in all age groups. We also analyzed the amount of alcohol reaching the embryo in the two strains and ruled out the possibility that TU has a more protective chorion. We conclude that the uncovered strain differences are due to genetic differences that protect TU from the deleterious effects of embryonic alcohol exposure.

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ABSTRACT

Aims: The study aimed to investigate (a) the association between low to moderate prenatal alcohol exposure (PAE) and the use of alcohol, tobacco and illicit drugs in adolescence and (b) whether the associations are modified by gender and ethnicity.

Methods: The subjects of the study were 5922 children and adolescents, aged from 11 to 17 years, enrolled in the cross-sectional German Health Interview and Examination Survey for Children and Adolescents (the KiGGS study). Information on PAE is based on parental self-report questionnaires. Use of alcohol, tobacco and illicit drugs was assessed through self-report questionnaires for adolescents.

Results: Low to moderate PAE was associated with an increased risk of drinking alcohol (adjusted odds ratio (OR) 1.73, 95% confidence interval (CI) 1.34, 2.18) and also of illicit drug use (adjusted OR 1.62, 95% CI 1.23, 2.14). The associations between PAE and the use of alcohol, tobacco and illicit drugs differed according to gender and ethnicity. Gender-stratified analyses resulted in adverse effects of PAE on drinking alcohol, smoking and illicit drug use in females; however, in German males, the associations disappeared. Stronger associations between PAE and the outcome measures were found in non-Germans.

Conclusions: Our findings indicate that low to moderate levels of maternal alcohol intake during pregnancy are a risk factor for use of alcohol, tobacco and illicit drugs by the offspring, with stronger associations in females and non-Germans.
"maternal morbidity", "perinatal death".

**Results:** Studies of FGR use multiple definitions, both with respect to cutoffs for defining restricted growth as well as growth norms; however the most common definition for epidemiological research was SGA using a birthweight less than the 10th percentile. Following this definition, SGA births accounted for 8.9% of all live births in 2010 in France. Major risk factors identified in the literature were previous SGA birth (4 fold increase in risk) (LE2), diabetes and vascular diseases (5 fold) (LE3), chronic hypertension (2 fold) (LE2), preeclampsia (5 to 12 fold according to severity) (LE2), pregnancy induced hypertension (2 fold) (LE2), smoking (2-3 fold) (LE2), drug and alcohol use (2-4 fold) (LE2), maternal age over 35 (3 fold) (LE2) and ethnic origin (2-3 fold for African-American or Asian origins) (LE2). Other risk factors with adjusted odds ratios around 1.5 were primiparity (LE2), multiple pregnancy (but only starting at 30 weeks of gestation) (LE2), socioeconomic disadvantage (LE2) and body mass index (BMI<18.5kg/m2) (LE2) SGA is associated with a four-fold increased risk of stillbirth (LE2) as well as higher rates of cesarean and induced labor before 37 weeks.

**Conclusions:** FGR is a complication of pregnancy with adverse consequences for fetal wellbeing. Sociodemographic and clinical risk factors can help to identify pregnant women at risk for this complication.


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**PubMed**


9. PERICONCEPTIONAL ALCOHOL CONSUMPTION CAUSES FETAL GROWTH RESTRICTION AND INCREASES GLYCOGEN ACCUMULATION IN THE LATE GESTATION RAT PLACENTA

Gårdebjer EM, Cuffe JS, Pantaleon M, Włodek ME, Moritz KM.
School of Biomedical Sciences, The University of Queensland, St Lucia, Queensland 4072, Australia.

**ABSTRACT**

**Introduction:** Alcohol consumption is a common social practice among women of childbearing age. With 50% of pregnancies being unplanned, many embryos are exposed to alcohol prior to pregnancy recognition and formation of the placenta. The effects of periconceptional (PC) alcohol exposure on the placenta are unknown.

**Methods:** Sprague-Dawley rats were exposed to alcohol (12.5% v/v ad libitum) from 4 days prior to 4 days after conception and effects on placental growth, morphology and gene/protein expression examined at embryonic day (E) 20.

**Results:** PC ethanol (EtOH)-exposed fetuses were growth restricted and their placental/body weight ratio and placental cross-sectional area were increased. This was associated with an increase in cross-sectional area of the junctional zone and glycogen cells, especially in PC EtOH-exposed placentas from female fetuses. Junctional Glut1 and Igf2 mRNA levels were increased. Labyrinth Igf1 mRNA levels were decreased in placentas from both sexes, but protein IGF1R levels were decreased in placentas from male fetuses only. Labyrinth mRNA levels of Slc38a2 were decreased and Vegfa were increased in placentas following PC EtOH-exposure but only placentas from female fetuses exhibited increased Kdr expression. Augmented expression of the protective enzyme 11βHsd2 was found in PC EtOH-exposed labyrinth.

**Discussion:** These observations are consistent with a stress response, apparent well beyond
the period of EtOH-exposure and demonstrate that PC EtOH alters placental development in a sex specific manner.

**Conclusion:** Public awareness should be increased to educate women about how excessive drinking even before falling pregnant may impact on placental development and fetal health.


10. **DEFICITS IN RESPONSE INHIBITION CORRELATE WITH OCULOMOTOR CONTROL IN CHILDREN WITH FETAL ALCOHOL SPECTRUM DISORDER AND PRENATAL ALCOHOL EXPOSURE**

Centre for Neuroscience Studies, Queens University, Kingston, ON, Canada.

**ABSTRACT**

Children with fetal alcohol spectrum disorder (FASD) or prenatal alcohol exposure (PAE) frequently exhibit impairment on tasks measuring inhibition. The objective of this study was to determine if a performance-based relationship exists between psychometric tests and eye movement tasks in children with FASD. Participants for this dataset were aged 5-17 years and included those diagnosed with an FASD (n=72), those with PAE but no clinical FASD diagnosis (n=21), and typically developing controls (n=139). Participants completed a neurobehavioral test battery, which included the NEPSY-II subtests of auditory attention, response set, and inhibition. Each participant completed a series of saccadic eye movement tasks, which included the antisaccade and memory-guided tasks. Both the FASD and the PAE groups performed worse than controls on the subtest measures of attention and inhibition. Compared with controls, the FASD group made more errors on the antisaccade and memory-guided tasks. Among the combined FASD/PAE group, inhibition and switching errors were negatively correlated with direction errors on the antisaccade task but not on the memory-guided task. There were no significant correlations in the control group. These data suggest that response inhibition deficits in children with FASD/PAE are associated with difficulty controlling saccadic eye movements which may point to overlapping brain regions damaged by prenatal alcohol exposure. The results of this study demonstrate that eye movement control tasks directly relate to outcome measures obtained with psychometric tests that are used during FASD diagnosis, and may therefore help with early identification of children who would benefit from a multidisciplinary diagnostic assessment.


PubMed, Matern Child Health J. 2013 Nov 1. [Epub ahead of print]

11. **PREVENTION OF SECONDARY CONDITIONS IN FETAL ALCOHOL SPECTRUM DISORDERS: IDENTIFICATION OF SYSTEMS-LEVEL BARRIERS**

Petrenko CL, Tahir N, Mahoney EC, Chin NP.
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**ABSTRACT**

www.nofas-uk.org
Fetal alcohol spectrum disorders (FASD) impact 2-5 % of the US population and are associated with life-long cognitive and behavioral impairments. Individuals with FASD have high rates of secondary conditions, including mental health problems, school disruptions, and trouble with the law. This study focuses on systems-level barriers that contribute to secondary conditions and interfere with prevention and treatment. Using a phenomenological methodology, semi-structured interviews and focus groups were conducted with parents of children with FASD and service providers. Data were analyzed using a framework approach. Participants emphasized the pervasive lack of knowledge of FASD throughout multiple systems. This lack of knowledge contributes to multi-system barriers including delayed diagnosis, unavailability of services, and difficulty qualifying for, implementing, and maintaining services. FASD is a major public health problem. Broad system changes using a public health approach are needed to increase awareness and understanding of FASD, improve access to diagnostic and therapeutic services, and create responsive institutional policies to prevent secondary conditions. These changes are essential to improve outcomes for individuals with FASD and their families and facilitate dissemination of empirically supported interventions.


12. VESTED INTERESTS IN ADDICTION RESEARCH AND POLICY. IS THE ALCOHOL INDUSTRY DELAYING GOVERNMENT ACTION ON ALCOHOL HEALTH WARNING LABELS IN AUSTRALIA?
Mathews R, Thorn M, Giorgi C.
Foundation for Alcohol Research and Education, Canberra, ACT, Australia.

ABSTRACT
Aims: This paper examines the strategies and arguments used by segments of the alcohol industry to delay the introduction of mandatory health warning labels on alcohol containers in Australia. These strategies are compared with those used by the tobacco industry to delay the introduction of warning labels for cigarettes.

Methods: Submissions made by members of the alcohol industry to the Australian Government's review of labelling and Parliamentary Inquiry into Fetal Alcohol Spectrum Disorders were analysed.

Results: Segments of the alcohol industry have delayed the introduction of mandatory alcohol health warning labels in Australia by questioning the rationale and evidence base for labels; arguing that they will cause damage to public health and the economy; lobbying and seeking to influence government and political representatives including through monetary donations; and introducing its own voluntary labelling scheme. The arguments made by these organizations against the introduction of mandatory health warning labels for alcohol are flawed and their empirical basis is limited.

Conclusion: The Australian Government has delayed the introduction of mandatory alcohol health warning labels in Australia by 2 years, until at least December 2013. The campaigning of some parts of the alcohol industry appears to have been instrumental in this decision.


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13. IN WESTERN AUSTRALIA, 70% OF MOTHERS OF BABIES WITH FETAL ALCOHOL SYNDROME DID NOT HAVE AN ALCOHOL-RELATED DIAGNOSIS RECORDED DURING PREGNANCY
Alati R.
School of Population Health, Centre for Youth Substance Abuse Research, University of Queensland, Brisbane, Queensland, Australia.

ABSTRACT

No Abstract Avaiable

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14. A REVIEW OF ENVIRONMENTAL CONTRIBUTIONS TO CHILDHOOD MOTOR SKILLS
1Centre for Child and Adolescent Health, School of Social and Community Medicine, University of Bristol, Bristol, United Kingdom.

ABSTRACT

Although much of children's motor skills have a heredity component, at least half of the variance is likely to be influenced by the environment. It is important to ascertain features of the environment that are responsible so that toxins can be avoided, children at risk can be identified, and beneficial interventions initiated. This review outlines the results of published studies and recommends the areas where further research is required. We found much confusion with little comparability concerning the ages or measures used. Few studies had sufficient power and few allowed for confounders. We found that research to date implicates associations with prenatal drinking ≥4 drinks of alcohol per day; diabetes; taking antidepressant drugs; being deficient in iodine or iron; dietary fish; and postnatal depression. The child appearing to be most at risk was born of low birth weight (but not due to preterm delivery) or with neonatal problems.

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15. INFLUENCE OF PRENATAL ALCOHOL EXPOSURE ON RETINAL DEVELOPMENT AND CELL DIFFERENTIATION
Institute of Neurobiology, Henan University, Kaifeng 475004, China. jinbo_deng@henu.edu.cn.

ABSTRACT

The aim of the present study was to investigate the effects of prenatal alcohol exposure
(PAE) on the development and cell differentiation of retina in offspring. The mouse model of PAE was made. HE staining and immunofluorescent labeling were carried out to visualize the structure, development and cell differentiation of the retina from postnatal day 0 (P0)-P30 offspring. The results showed that PAE can lead to the retardation of retinal development, the reduction of number of bipolar cells and horizontal cells, the disorder of horizontal cells' polarity, as well as the retinal thickening in a dose-dependent manner. The data suggest that alcohol exposure during pregnancy can lead to the developmental retardation of retina and decreased number of bipolar cells and horizontal cells in the retina of offspring.


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16. A SCREEN OF ZEBRAFISH MUTANTS IDENTIFIES ETHANOL-SENSITIVE GENETIC LOCI
Swartz ME, Wells MB, Griffin M, McCarthy N, Lovely CB, McGurk P, Rozacky J, Eberhart JK. Waggoner Center for Alcohol & Addiction Research, Institute for Cell and Molecular Biology, University of Texas at Austin, Austin, Texas.

ABSTRACT
Background: Fetal alcohol spectrum disorders (FASD) are a highly variable set of phenotypes caused by fetal alcohol exposure. Numerous factors influence FASD phenotypes, including genetics. The zebrafish is a powerful vertebrate model system with which to identify these genetic factors. Many zebrafish mutants are housed at the Zebrafish International Resource Center (ZIRC). These mutants are readily accessible and an excellent source to screen for ethanol (EtOH)-sensitive developmental structural mutants.

Methods: We screened mutants obtained from ZIRC for sensitivity to EtOH teratogenesis. Embryos were treated with 1% EtOH (41 mM tissue levels) from 6 hours postfertilization onward. Levels of apoptosis were evaluated at 24 hours postfertilization. At 4 days postfertilization, the craniofacial skeleton, peripheral axon projections, and sensory neurons of neuromasts were examined. Fish were genotyped to determine whether there were phenotype/genotype correlations.

Results: Five of 20 loci interacted with EtOH. Notable among these was that vangl2, involved in convergent extension movements of the embryonic axis, interacted strongly with EtOH. Untreated vangl2 mutants had normal craniofacial morphology, while severe midfacial defects including synophthalmia and narrowing of the palatal skeleton were found in all EtOH-treated mutants and a low percentage of heterozygotes. The cell cycle gene, plk1, also interacted strongly with EtOH. Untreated mutants have slightly elevated levels of apoptosis and loss of ventral craniofacial elements. Exposure to EtOH results in extensive apoptosis along with loss of neural tissue and the entire craniofacial skeleton. Phenotypes of hinfp, mars, and foxi1 mutants were also exacerbated by EtOH.

Conclusions: Our results provide insight into the gene-EtOH interactions that may underlie EtOH teratogenesis. They support previous findings that EtOH disrupts elongation of the embryonic axis. Importantly, these results show that the zebrafish is an efficient model with which to test for gene-EtOH interactions. Understanding these interactions will be crucial to understanding of the FASD variation.


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17. INAPPROPRIATE FEEDING BEHAVIORS AND DIETARY INTAKES IN CHILDREN WITH FETAL ALCOHOL SPECTRUM DISORDER OR PROBABLE PRENATAL ALCOHOL EXPOSURE

Werts RL, Van Calcar SC, Wargowski DS, Smith SM.
Waisman Center, University of Wisconsin School of Medicine and Public Health, Madison, Wisconsin; Department of Nutritional Sciences, University of Wisconsin-Madison, Madison, Wisconsin.

ABSTRACT

Background: Prenatal alcohol exposure (PAE) is a leading cause of significant neurobehavioral and neurocognitive deficits. Its potential consequences for eating behaviors, nutritional status, and other nutritional issues in childhood have received little attention.

Methods: Nineteen children (11 boys, 8 girls) of mean age 9.6 years, referred for fetal alcohol spectrum disorder (FASD) screening and assessment, were analyzed with physical exams and caregiver questionnaires to identify possible abnormalities in food and eating behaviors. Fourteen children contributed 24-hour diet recalls and were assessed for nutritional status.

Results: Seventy-nine percent of participants were diagnosed with FASD and 63.2% had confirmed PAE. Fifty percent of girls were overweight or obese, whereas 37% of boys had reduced stature, weight, or body mass index for their age. Recurring feeding problems included constant snacking (36.8%), lack of satiety (26.3%), and picky eating/poor appetite (31.6%). None had oral feeding problems. Constipation was common (26.3%). Macronutrient intakes were largely normal, but sugar consumption was excessive (140 to 190% of recommendations) in 57% of subjects. Vitamin A intake exceeded the upper limit for 64% of participants, whereas ≥50% had intakes <80% of recommended daily allowances (RDAs) for choline, vitamin E, potassium, β-carotene, and essential fatty acids; 100% had vitamin D intakes <80% of the RDA.

Conclusions: PAE may be associated with altered acquisition and distribution of body mass with increasing age. Disordered eating was common. The increased feeding behaviors surrounding lack of satiety suggest that self-regulation may be altered. Constipation could reflect low dietary fiber or altered gastrointestinal function. These exploratory data suggest that children with PAE may be at risk for nutritional deficiencies, which are influenced by inappropriate food preferences, disordered eating patterns, medication use, and the stressful dynamics surrounding food preparation and mealtime.


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18. BINGE DRINKING PRIOR TO PREGNANCY DETECTION IN A NONHUMAN PRIMATE: BEHAVIORAL EVALUATION OF OFFSPRING

Golub MS, Hogrefe CE, Vandevoort CA.
Department of Environmental Toxicology, University of California, Davis, California.

ABSTRACT

Background: Minimal scientific information is available to inform public health policy on binge
drinking prior to pregnancy detection. The nonhuman primate provides a valuable animal model for examining consequences to reproduction and offspring function that may result from this common pattern of alcohol abuse.

**Methods:** Adult female rhesus monkeys were dosed with 1.5 g/kg per day ethanol (EtOH) by gavage 2 d/wk beginning 7 months prior to mating and continuing to pregnancy detection at 19 to 20 days gestation. Postnatal evaluation of control (n = 6) and EtOH-treated (n = 4) infants included a neonatal neurobehavioral assessment, a visual paired comparison (cognitive) test at 35 days of age, and mother-infant interaction at 100 to 112 days of age.

**Results:** Alcohol-exposed neonates did not differ from controls in posture and reflex measures. Longer durations of visual fixation, suggesting slower visual processing, and greater novelty preference were seen in the alcohol group. At early weaning age, as infants spent more time away from their dams, more of the reunions between mother and infant were initiated by the mothers in the alcohol-exposed group, suggesting a more immature mother-infant interaction.

**Conclusions:** Intermittent high-dose alcohol exposure (binge drinking) discontinued at early pregnancy detection in rhesus monkey can result in altered behavioral function in the infant. Mediating effects on ovum, reproductive tract, and early embryo can be explored in this model. Studies of longer-term consequences in human populations and animal models are needed.


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19. **WHAT RESEARCH IS BEING DONE ON PRENATAL ALCOHOL EXPOSURE AND FETAL ALCOHOL SPECTRUM DISORDERS IN THE RUSSIAN RESEARCH COMMUNITY?**

Svetlana Popova1 2 3 4, Aleksandra Yaltonskaya5 6, Vladimir Yaltonsky5 7, Yaroslav Kolpakov5 7, Ilya Abrosimov5 7, Kristina Pervakov1, Valeria Tanner1 and Jürgen Rehm1 2 4 8

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8 Epidemiological Research Unit, Klinische Psychologie and Psychotherapie, Technische Universität Dresden, Chemnitzer St. 46, D-01187 Dresden, Germany

**ABSTRACT**

**Aims:** Although Russia has one of the highest rates of alcohol consumption and alcohol-attributable burden of disease, little is known about the existing research on prenatal alcohol exposure (PAE) and Fetal Alcohol Spectrum Disorders (FASDs) in this country. The objective of this study was to locate and review published and unpublished studies related to any alcohol-related problems.
aspect of PAE and FASD conducted in or using study populations from Russia.

**Methods:** A systematic literature search was conducted in multiple English and Russian electronic bibliographic databases. In addition, a manual search was conducted in several major libraries in Moscow.

**Results:** The search revealed a small pool of existing research studies related to PAE and/or FASD in Russia (126: 22 in English and 104 in Russian). Existing epidemiological data indicate a high prevalence of PAE and FASD, which underlines the strong negative impact that alcohol has on mortality, morbidity and disability in Russia. High levels of alcohol consumption by women of childbearing age, low levels of contraception use, and low levels of knowledge by health and other professionals regarding the harmful effects of PAE put this country at great risk of further alcohol-affected pregnancies.

**Conclusions:** Alcohol preventive measures in Russia warrant immediate attention. More research focused on alcohol prevention and policy is needed in order to reduce alcohol-related harm, especially in the field of FASD.

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20. **DETERMINANTS OF ALCOHOL CESSATION, REDUCTION AND NO REDUCTION DURING PREGNANCY**
Kitsantas P, Gaffney KF, Wu H, Kastello JC.
Department of Health Administration and Policy, MS 1J3, College of Health and Human Services, George Mason University, 4400 University Drive, Fairfax, VA, 22030, USA, pkitsant@gmu.edu.

**ABSTRACT**

**Purpose:** Despite public health initiatives targeting the harmful effects of alcohol exposure on fetal growth, 12 % of pregnant women report current alcohol use. For women who reported drinking alcohol prior to pregnancy, we examined several factors as predictors of three alcohol use patterns during the third trimester of pregnancy: cessation, reduction and no reduction.

**Methods:** Using the 2002-2009 Pregnancy Risk Assessment Monitoring System (PRAMS) dataset (311,428 records), a multinomial logistic regression model was constructed to compare alcohol risk by category: (1) cessation vs. reduction (2) no reduction vs. reduction.

**Results:** In this sample, 49.4 % drank alcohol before pregnancy. Among those who drank before pregnancy, ~87 % quit drinking during pregnancy, 6.6 % reduced, and about 6.4 % reported no reduction. Older women and those with higher education were more likely to reduce than quit their alcohol use. Conversely, women who were black or Hispanic, overweight, obese, or multiparas were more likely to quit than to reduce their prenatal alcohol consumption. Several stressors such as abuse during pregnancy increased their risk of not quitting or not reducing alcohol during the last trimester of pregnancy.

**Conclusions:** Differentiating prenatal alcohol use patterns can inform the design of targeted interventions and public health policies to meet the Healthy People 2020 objective for achieving a national rate of 98.3 % alcohol abstinence during pregnancy.

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21. MATERNAL SUPPLEMENTATION OF NUCLEOTIDES IMPROVES THE BEHAVIORAL DEVELOPMENT OF PRENATAL ETHANOL-EXPOSED MICE
Dong W, Wu Z, Xu L, Fang Y, Xu Y.
Department of Nutrition and Food Hygiene, School of Public Health, Peking University, Beijing, China.

ABSTRACT
Maternal ethanol consumption during pregnancy can induce learning deficits in the offspring. The objective of this study was to assess whether supplementation of exogenous nucleotides during pregnancy and lactation would ameliorate prenatal ethanol-induced learning and memory deficits in the offspring of mice, and to explore the possible mechanisms. In the present study, pregnant C57BL/6J mice were exposed to ethanol (5 g/kg body weight) intragastrically from gestational day (GD) 6 to GD15. The dams in exogenous nucleotide intervention groups were fed with feed containing 0.01 %, 0.04 %, or 0.16 % nucleotide powder, with control and ethanol groups receiving normal feed. The dams were allowed to deliver naturally and to breast feed their offspring. After weaning, behavioral tests were carried out in the offspring of each group. Serum oxidation indexes were analyzed, and the hippocampus of each offspring was collected and detected for acetyl cholinesterase (AChE) activity and the expression of p-CREB, CREB, and BDNF. The results showed that maternal supplementation with exogenous nucleotides during pregnancy could ameliorate prenatal ethanol-induced learning and memory deficits in the offspring of mice, through improving their antioxidant capacity, reversing hippocampus AChE levels, and allowing the expression of some proteins related to learning and memory. However, different sensitivities were found between the two sexes.

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22. IDENTIFYING THE NEUROBEHAVIORAL PHENOTYPE OF FETAL ALCOHOL SPECTRUM DISORDER IN YOUNG CHILDREN
Petra Breiner, Irena Nulman, Gideon Koren

ABSTRACT
Background: Most children with Fetal Alcohol Spectrum Disorder (FASD) do not display the typical facial changes, making the diagnosis much more challenging due to poor specificity of the brain dysfunction exhibited by these children. We have recently described and validated a behavioral phenotype of FASD using items from the Child Behavior Checklist (The Neurobehavioral Screening Test, NST). This tool has high sensitivity and specificity in separating children aged 6-13 yrs with FASD from those with ADHD and from healthy controls.

Objectives: To test the validity of the NST for children aged 4-6 years in order to help facilitate diagnosis of FASD in young children.

Methods: Children referred to Motherisk for FASD diagnosis are all tested using the Child Behavior Checklist. We compared the scores of children 4-6 yrs diagnosed with FASD to those referred but not receiving a diagnosis, as well as to normal healthy control children of
the same age range.

**Results:** Out of the 10 items of NST used at age 6-13 years, 3 are not scored in children 4-6 years of age. Using the 7 remaining items, children with FASD endorsed significantly more items (6.7+/-1.3) than healthy controls (2.3+/-1.2), or alcohol-exposed children who were not given an FASD diagnosis (4.7+/-1.9). Using a cut-off of 5 out of 7 items, the NST had a 94% sensitivity and 96% specificity in identifying children with FASD. Nine of 19 children exposed to alcohol with whom an FASD diagnosis could not be confirmed, scored 5 or more on the NST.

**Conclusions:** In this pilot study, the NST has shown very high sensitivity and specificity and can be used to identify children who are very likely to be diagnosed with FASD.

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23. **ALCOHOL EXPOSURE AMONG PREGNANT WOMEN IN SUB-SAHARAN AFRICA: A SYSTEMATIC REVIEW**  
Celia L Culley, Tasha D Ramsey, Godfrey Mugyenyi, Gertrude N Kiwanuka, Joseph Ngonzi, Stuart MacLeod, Gideon Koren, Brian E Grunau, Matthew O Wiens

**ABSTRACT**

**Background:** The prevalence of general alcohol use in many countries of sub-Saharan Africa (SSA) is high. However, research examining alcohol use in among pregnant women within this population is limited. A review of the current status of research examining the prevalence of alcohol exposed pregnancies (AEP) is required to inform future research aiming to decrease this occurrence and its subsequent socio-economic complications.

**Objective:** The primary objective was to identify all published papers estimating prevalence and risk-factors of alcohol use among pregnant women in SSA. A secondary objective was to determine changes in alcohol use following pregnancy recognition.

**Methods:** PubMed/Medline, Embase, IPA, CINAHL were systematically searched using MeSH terms and keywords from inception date to March 2013. Studies from SSA reporting prevalence of alcohol use among pregnant women were included.

**Results:** Twelve studies were identified. Studies varied significantly according to design and study population. Prevalence of alcohol use during pregnancy ranged from 2.2%-87%. The most important risk-factors for alcohol use included tobacco use, partner violence, urban living, and having a male partner who drank alcohol. Only three studies examined changes in alcohol use prior to and following pregnancy recognition with absolute reductions of between 9% and 15%.

**Conclusions:** Although the burden of alcohol use during pregnancy is likely a significant problem, limited data currently exist for the majority of SSA countries. Furthermore, significant variation likely exists within various populations. Further research is required to explore alcohol use in pregnancy. Strategies to decrease AEP must be developed and implemented in standard pre-natal care.

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http://www.jptcp.com/pubmed.php?articleId=437

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ABSTRACT

Background: The estimated prevalence of fetal alcohol syndrome (FAS) is 8 for every 1000 live births. FAS has serious, lifelong consequences for the affected children and their families. A variety of professionals deal with persons who have FAS, including pediatricians, general practitioners, neurologists, gynecologists, psychiatrists, and psychotherapists. Early diagnosis is important so that the affected children can receive the support they need in a protective environment.

Methods: A multidisciplinary guideline group has issued recommendations for the diagnosis of FAS after assessment of the available scientific evidence. This information was derived from pertinent literature (2001-2011) retrieved by a systematic search in PubMed and the Cochrane Library, along with the US-American and Canadian guidelines and additional literature retrieved by a manual search.

Results: Of the 1383 publications retrieved by the searches, 178 were analyzed for the evidence they contained. It was concluded that the fully-developed clinical syndrome of FAS should be diagnosed on the basis of the following criteria: Patients must have at least one growth abnormality, e.g., short stature, as well as all three characteristic facial abnormalities—short palpebral fissure length, a thin upper lip, and a smooth philtrum. They must also have at least one diagnosed structural or functional abnormality of the central nervous system, e.g., microcephaly or impaired executive function. Confirmation of intrauterine exposure to alcohol is not obligatory for the diagnosis.

Conclusion: Practical, evidence-based criteria have now been established for the diagnosis of the fully-developed FAS syndrome. More research is needed in order to enable uniform, evidence-based diagnostic assessment of all fetal alcohol spectrum disorders and optimize supportive measures for the children affected by them.


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activities impacted on Ms A's mood and identity. Ms A experienced improved recall for events recorded using SenseCam, and showed improvement on subjective ratings of identity. However, a corresponding improvement in mood was not seen, and the study was ended early at Ms A's request. Qualitative information was gathered to explore Ms A's experience of the study, and investigate psychosocial factors that may have impacted on the use of SenseCam. SenseCam may be of significant use as a compensatory memory aid for people with Korsakoff's syndrome and other types of alcohol-related brain damage (ARBD), but acceptance of memory impairment and consistent support may be among the factors required to support the use of such assistive technologies in a community setting.

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26. IMPACT OF MATERNAL NEGATIVE AFFECTIVITY ON LIGHT ALCOHOL USE AND BINGE DRINKING DURING PREGNANCY
Stene-Larsen K, Torgersen L, Strandberg-Larsen K, Normann PT, Vollrath ME.
Department of Psychosomatics and Health Behaviors, Norwegian Institute of Public Health, Oslo, Norway.

ABSTRACT
Objectives: To investigate whether maternal negative affectivity, a tendency to frequent negative emotions and views, is associated with light alcohol use and binge drinking during pregnancy.

Design: Cohort.


Population: The study includes complete information on 66 111 pregnant women and their partners.

Methods: We used data from the Norwegian Mother and Child Cohort study (MoBa) representing 39% of the pregnant population.

Main outcome measures: Light alcohol use (0.5-2 units one to four times per month) and binge drinking (an intake of 5 alcohol units or more) measured with the Alcohol Use Disorder Identification Test-Consumption (AUDIT-C).

Results: For each unit increase in maternal negative affectivity the odds for light alcohol use increased with 27% in the first trimester [95% confidence interval (CI) 1.19-1.36], and 28% in the second trimester (95% CI 1.18-1.39). With respect to binge drinking, each unit increase in maternal negative affectivity was associated with 55% higher odds in the first trimester (95% CI 1.44-1.67), and 114% higher odds in the second trimester (95% CI 1.70-2.69).

Conclusions: Negative affectivity is associated with both light alcohol use and binge drinking during pregnancy. The mechanisms mediating the relation between negative affectivity and alcohol use in pregnancy should be investigated further.

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27. **PERI-IMPLANTATIONAL IN VIVO AND IN VITRO EMBRYO-TROPHOBLAST DEVELOPMENT AFTER PERIGESTATIONAL ALCOHOL EXPOSURE IN THE CD-1 MOUSE**

Pérez-Tito L, Bevilacqua E, Cebral E.
Laboratorio de Reproducción y Fisiopatología Materno-Embrionaria, Instituto de Fisiología, Biología Molecular y Neurociencias (IFIBYNE-CONICET), Departamento de Biodiversidad y Biología Experimental (DBBE), Facultad de Ciencias Exactas y Naturales (FCEyN), Universidad de Buenos Aires (UBA), Buenos Aires, Argentina and.

**ABSTRACT**

Long-term pregestational ethanol exposure induced altered fertilization and preimplantation embryogenesis. We evaluated preimplantational embryo-trophoblast differentiation, growth and invasiveness after perigestational ethanol 10% ingestion for 15 days preceding and up to day 4 (treated females [TF]: TF-D4 group) or 5 (TF-D5) of CD-1 gestation (control females [CF] with water). In TF-D4, expanded and hatched blastocyst numbers were significantly reduced (p < 0.05) versus CF-D4. Abnormal embryos and percentage of pyknotic nuclei were increased, and early blastocyst growth (nuclear number/embryo) and mitotic index was reduced (p < 0.05) versus CF-D4. On day 5 of gestation, TF-D5 presented significantly reduced total embryos and advanced embryo type 3 number versus CF-D5 (p < 0.05). During in vitro development, up to 72-hour culture, TF-D5 had reduced embryo type 1 (the least developed) and 3 percentages (p < 0.05) versus controls, whereas embryo type 2 percentage increased (p < 0.05) versus CF-D5. Embryo-trophoblast growth was studied during culture by morphometry. Embryo size ranges were classified as small, medium and large embryos. At 48-hour culture, small and medium embryos of TF had significantly increased mean area versus CF (p < 0.05), whereas large embryos had reduced mean area at 24-hour culture. Perigestational alcohol exposure up to days 4-5 induced embryo differentiation retardation, abnormal blastocyst growth and alterations of embryo-trophoblast growth and expansion during implantation, suggesting impaired regulation of trophoblast invasion and a relation with early pregnancy loss after mouse perigestational alcohol consumption.


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28. **DEVELOPMENTAL REGULATION OF NEUROLIGIN GENES IN JAPANESE RICEFISH (ORYZIAS LATIPES) EMBRYOGENESIS MAINTAINS THE RHYTHM DURING ETHANOL-INDUCED FETAL ALCOHOL SPECTRUM DISORDER**

Haron MH, Khan IA, Dasmahapatra AK.
Department of Pharmacology, University of Mississippi, University, MS 38677, USA.

**ABSTRACT**

Although prenatal alcohol exposure is the potential cause of fetal alcohol spectrum disorder (FASD) in humans, the molecular mechanism(s) of FASD is yet unknown. We have used Japanese ricefish (Oryzias latipes) embryogenesis as an animal model of FASD and reported that this model has effectively generated several phenotypic features in the cardiovasculature and neurocranial cartilages by developmental ethanol exposure which is analogous to human FASD phenotypes. As FASD is a neurobehavioral disorder, we are searching for a molecular target of ethanol that alters neurological functions. In this communication, we have focused on...
neuroligin genes (nlgn) which are known to be active at the postsynaptic side of both excitatory and inhibitory synapses of the central nervous system. There are six human NLGN homologs of Japanese ricefish reported in public data bases. We have partially cloned these genes and analyzed their expression pattern during normal development and also after exposing the embryos to ethanol. Our data indicate that the expression of all six nlgn genes in Japanese ricefish embryos is developmentally regulated. Although ethanol is able to induce developmental abnormalities in Japanese ricefish embryogenesis comparable to the FASD phenotypes, quantitative real-time PCR (qPCR) analysis of nlgn mRNAs indicate unresponsiveness of these genes to ethanol. We conclude that the disruption of the developmental rhythm of Japanese ricefish embryogenesis by ethanol that leads to FASD may not affect the nlgn gene expression at the message level.

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29. EFFECTS OF PRENATAL COCAINE/POLYDRUG EXPOSURE ON SUBSTANCE USE BY AGE 15
Minnes S, Singer L, Min MO, Wu M, Lang A, Yoon S.
Case Western Reserve University Jack, Joseph and Morton Mandel School of Applied Social Sciences, United States. Electronic address: sonia.minnes@case.edu.

ABSTRACT

Objective: Examined effects of prenatal cocaine exposure (PCE) on tobacco, alcohol, marijuana and cocaine use by age 15.

Methods: Adolescent (n=358; 183 PCE, 175 non-prenatally cocaine exposed; NCE) drug use was assessed using urine, hair, and/or blood spot samples and self-report (Youth Risk Behavior Surveillance System; YRBSS) at ages 12 and 15. Logistic regression assessed effects of PCE on drug use controlling for other drug exposures, environment and blood lead levels (BLL).

Results: Adjusted percentages of drug use (PCE vs. NCE) were: tobacco 35% vs. 26% (p<.04), marijuana 33% vs. 23% (p<.04), alcohol 40% vs. 35% (p<.01), and any drugs 59% vs. 50% (p<.005). PCE adolescents were twice as likely to use tobacco (OR=2.02, 95% CI=1.05-3.90, p<.04), 2.2 times more likely to use alcohol (OR=2.16, 95% CI=1.21-3.87, p<.01) and 1.8 times more likely to use marijuana (OR=1.81, 95% CI=1.02-3.22, p<.04) than NCE adolescents. A race-by-cocaine-exposure interaction (p<.01) indicated PCE non-African American adolescents had greater probability of tobacco use (65%) than NCE non-African American youth (21%). PCE was associated with any drug use (OR=2.16, CI=1.26-3.69, p<.005), while higher BLL predicted alcohol use (p<.001). Violence exposure was a predictor of tobacco (p<.002), marijuana (p<.0007) and any drug (p<.04).

Conclusions: PCE and exposure to violence increased the likelihood of tobacco, marijuana or any drug use by age 15, while PCE and higher early BLL predicted alcohol use. Prevention efforts should target high risk groups prior to substance use initiation.

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30. **EFFECT OF PRENATAL ALCOHOL EXPOSURE ON CHILDHOOD ACADEMIC OUTCOMES: CONTRASTING MATERNAL AND PATERNAL ASSOCIATIONS IN THE ALSPAC STUDY**

School of Population Health, University of Queensland, Brisbane, Queensland, Australia; Centre for Youth Substance Abuse Research, University of Queensland, Brisbane, Queensland, Australia.

**ABSTRACT**

**Background:** The impact of low-to-moderate levels of alcohol consumption during pregnancy on child cognitive outcomes has been of recent concern. This study has tested the hypothesis that low-to-moderate maternal alcohol use in pregnancy is associated with lower school test scores at age 11 in the offspring via intrauterine mechanisms.

**Methods:** We used data from the Avon Longitudinal Study of Parents and Children (ALSPAC), a birth cohort study based in the South West of England. Analyses were conducted on 7062 participants who had complete data on: maternal and paternal patterns of alcohol use in the first trimester and at 18 weeks' gestation, child's academic outcomes measured at age 11, gender, maternal age, parity, marital status, ethnicity, household crowding, home ownership status and parental education. We contrasted the association of mother's alcohol consumption during pregnancy with child's National Curriculum Key Stage 2 (KS2) test scores with the association for father's alcohol consumption (during the time the mother was pregnant) with child's National Curriculum Key Stage 2 (KS2) test scores. We used multivariate linear regression to estimate mean differences and 95% confidence intervals [CI] in KS2 scores across the exposure categories and computed f statistics to compare maternal and paternal associations.

**Findings and conclusions:** Drinking up to 1 unit of alcohol a day during pregnancy was not associated with lower test scores. However, frequent prenatal consumption of 4 units (equivalent to 32 grams of alcohol) on each single drinking occasion was associated with reduced educational attainment [Mean change in offspring KS2 score was -0.68 (-1.03, -0.33) for maternal alcohol categories compared to 0.27 (0.07, 0.46) for paternal alcohol categories]. Frequent consumption of 4 units of alcohol during pregnancy may adversely affect childhood academic outcomes via intrauterine mechanisms.

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31. **PREVALENCE AND PREDICTORS OF EXCLUSIVE BREASTFEEDING AMONG WOMEN IN KILIMANJARO REGION, NORTHERN TANZANIA: A POPULATION BASED CROSS-SECTIONAL STUDY**

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**ABSTRACT**

**Background:** Exclusive breastfeeding (EBF) is a simple and cost-effective intervention to
improve child health and survival. Effective EBF has been estimated to avert 13% - 15% of under-five mortality and contribute to reduce mother to child transmission of HIV. The prevalence of EBF for infant less than six months is low in most developing countries, including Tanzania (50%). While the Tanzania Demographic Health Survey collects information on overall EBF prevalence, it does not evaluate factors influencing EBF. The aim of this paper was to determine the prevalence and predictors of exclusive breastfeeding in urban and rural areas in Kilimanjaro region.

**Methods:** A population-based cross-sectional study was conducted between June 2010 to March 2011 among women with infants aged 6-12 months in Kilimanjaro. Multi-stage proportionate to size sampling was used to select participants from all the seven districts of the region. A standardized questionnaire was used to collect socio-demographic, reproductive, alcohol intake, breastfeeding patterns and nutritional data during the interviews. Estimation on EBF was based on recall since birth. Multivariable logistic regression was used to obtain independent predictors of EBF.

**Results:** A total of 624 women participated, 77% (483) from rural areas. The prevalence of EBF up to six months in Kilimanjaro region was 20.7%, without significant differences in the prevalence of EBF up to six months between urban (22.7%) and rural areas (20.1%); (OR = 0.7, 95% CI 0.5,1.4). In multivariable analysis, advice on breastfeeding after delivery (Adjusted odds ratio, AOR = 2.6, 95% CI 1.5, 4.6) was positively associated with EBF up to six months. Compared to married/cohabiting and those who do not take alcohol, single mothers (AOR = 0.4, 95% CI 0.2, 0.9) and mothers who drank alcohol (AOR = 0.4, 95% CI 0.3, 0.7) had less odds to practice EBF up to six months.

**Conclusion:** Prevalence of EBF up to six months is still low in Kilimanjaro, lower than the national coverage of 50%. Strengthening of EBF counseling in all reproductive and child health clinics especially during antenatal and postnatal periods may help to improve EBF rates.

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**32. A REPORT ON THE FETAL ALCOHOL SPECTRUM DISORDERS STUDY GROUP MEETING OF 2012, THEME TITLE, "BIOMARKERS FOR FASD"
**
Miranda RC, Kable J, Reynolds JN, Valenzuela CF.
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**ABSTRACT**
The 2012 meeting of the Fetal Alcohol Spectrum Disorders Study Group (FASDSG) focused on the development and ethics of biomarkers for fetal alcohol exposure. This one-day international conference brought students and trainees together with clinicians and researchers to discuss the latest research on FASD. One keynote speaker discussed the value of profiling epigenetic modifications in readily available fetal tissues to diagnose fetal exposure to environmental agents, while the second speaker discussed the ethics of biomarker development within the context of core principles of justice, autonomy, beneficence and non-maleficence. Three sessions of short data talks informed the audience of research advances with particular emphasis on the diagnosis of FASD. Other activities included updates on FASD-related activities by representatives of government agencies, a report on the implementation FASD-related diagnostic criteria in the fifth edition of the Diagnostic and Statistical Manual (DSM-5) of the American Psychiatric Association and a networking lunch.
and the presentation of the "Merit Award" to Dr. Nathan Muraski for his work on behavioral outcomes of fetal alcohol exposure. The capstone of the meeting was the presentation of the "Henri Rosett" award to Dr. Denis Viljoen, in recognition of his role in raising awareness about the incidence of FASD in South Africa and in promoting FASD prevention and treatment programs as chairperson and chief executive officer of the Foundation for Alcohol Related Research (FARR).


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33. PERICONCEPTION MATERNAL CHARACTERISTICS AND EMBRYONIC GROWTH TRAJECTORIES: THE ROTTERDAM PREDICT STUDY
van Uitert EM, van der Elst-Otte N, Wilbers JJ, Exalto N, Willemsen SP, Eilers PH, Koning AH, Steegers EA, Steegers-Theunissen RP.
Department of Obstetrics and Gynaecology, Erasmus MC, University Medical Centre, 3015 GD Rotterdam, The Netherlands.

ABSTRACT
Study question: Are maternal characteristics and lifestyle factors associated with human embryonic growth trajectories?

Summary answer: Periconception maternal age is associated with increased, and smoking and alcohol use with decreased embryonic growth trajectories, estimated with crown-rump length (CRL) measurements.

What is known already: Fetal weight is associated with health and disease in later life. Maternal characteristics and lifestyle factors affect fetal growth in the second and third trimesters of pregnancy and at birth; however, little is known about the association of these characteristics with first trimester embryonic growth.

Study design, size, duration: In a tertiary centre, pregnant women were recruited and enrolled in a prospective periconception cohort study before 8 weeks of gestation. We selected 87 spontaneously conceived singleton pregnancies of women recruited in 2009 and 2010 that ended in non-malformed live births.

Participants/materials, setting, methods: We performed weekly three-dimensional ultrasound scans from enrolment up to 13 weeks of gestation. At enrolment, a questionnaire was completed. Embryonic CRL measurements were performed using the V-Scope software in the BARCO I-Space. Associations between maternal characteristics and embryonic growth were assessed using square root transformed CRL as response in linear mixed model analyses, adjusted for potential confounders.

Main results and the role of chance: Four hundred and ninety-six scans from 87 pregnancies were included. In the multivariable analysis, maternal age was positively associated with first trimester CRL (difference per maternal year of age 0.024√mm (95% confidence interval (CI) 0.009, 0.040), P = 0.001). At 6 and 12 weeks of gestation, the CRL of an embryo from a 40-year-old mother was estimated 2.0 mm (61%) and 7.2 mm (14%) larger, respectively, compared with an embryo from a 20-year-old mother. Smoking of 10 or more cigarettes per day was negatively associated with CRL (difference -0.211√mm (95% CI -0.416, -0.006), P = 0.04), with embryos that were 0.9 mm (18.7%) and 3.1 mm (5.5%) smaller at 6 and 12 weeks, respectively, compared with non-smokers. Periconception alcohol use was negatively associated with CRL growth rate (difference -0.0025√mm (95% CI -0.0047, -0.0005), P = 0.014)
0.0003)/day gestational age, P = 0.022), with embryos that were 0.2 mm (3%) and 1.1 mm (2%) smaller at 6 and 12 weeks, respectively, compared with non-alcohol users. Parity, BMI and moment of initiation of folic acid use were not significantly associated with embryonic CRL.

**Limitations, reasons for caution:** Due to the selection of pregnancies in a tertiary centre and the small number of pregnancies, the external validity of the results has to be confirmed using larger sample sizes and other population-based periconception cohort studies.

**Wider implications of the findings:** The association of maternal age and smoking with embryonic growth is in line with previous literature, whereas the association between embryonic growth and alcohol use is a new finding. However, concerning exposure to alcohol, the effect estimate was small and it is questionable whether this is of clinical value. More research is warranted to unravel underlying mechanisms and to assess the implications for preconception and early pregnancy care, such as the development and implementation of effective lifestyle interventions.

**Study funding/competing interest(s):** The work was funded by the Department of Obstetrics and Gynaecology, Erasmus MC, University Medical Centre, Rotterdam, The Netherlands. The authors declare no conflicts of interest.

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health development. It is cautiously concluded that the failure to control for these factors introduces residual and/or unmeasured confounding into the analyses, and thus masks the potential (small) effect of being exposed to low doses of alcohol in pregnancy. It is recommended that future studies control for factor scores rather than for the observed variables as is practice today.


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35. ALCOHOL AND BREASTFEEDING
Haastrup MB, Pottegård A, Damkier P.
Department of Clinical Chemistry & Pharmacology, Odense University Hospital, Odense, Denmark.

ABSTRACT
While the harmful effects of alcohol during pregnancy are well established, the consequences of alcohol intake during lactation have been far less examined. We reviewed available data on the prevalence of alcohol intake during lactation, the influence of alcohol on breastfeeding, the pharmacokinetics of alcohol in lactating women and nursing infants, and the effects of alcohol intake on nursing infants. A systematic search was performed in PubMed from origin to May 2013, and 41 publications were included in the review. Approximately half of all lactating women in Western countries consume alcohol while breastfeeding. Alcohol intake inhibits the milk ejection reflex, causing a temporary decrease in milk yield. The alcohol concentrations in breastmilk closely resemble those in maternal blood. The amount of alcohol presented to nursing infants through breastmilk is approximately 5-6% of the weight-adjusted maternal dose, and even in a theoretical case of binge drinking, the children would not be subjected to clinically relevant amounts of alcohol. Newborns metabolise alcohol at approximately half the rate of adults. Minute behavioural changes in infants exposed to alcohol-containing milk have been reported, but the literature is contradictory. Any long-term consequences for the children of alcohol-abusing mothers are yet unknown, but occasional drinking while breastfeeding has not been convincingly shown to adversely affect nursing infants. In conclusion, special recommendations aimed at lactating women are not warranted. Instead, lactating women should simply follow standard recommendations on alcohol consumption.


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36. A ROLE FOR GLUTATHIONE, INDEPENDENT OF OXIDATIVE STRESS, IN THE DEVELOPMENTAL TOXICITY OF METHANOL
Siu MT, Shapiro AM, Wiley MJ, Wells PG.
Division of Biomolecular Sciences, Faculty of Pharmacy, University of Toronto, Toronto, Ontario, Canada.
ABSTRACT
Oxidative stress and reactive oxygen species (ROS) have been implicated in the teratogenicity of methanol (MeOH) in rodents, both in vivo and in embryo culture. We explored the ROS hypothesis further in vivo in pregnant C57BL/6J mice. Following maternal treatment with a teratogenic dose of MeOH, 4g/kg via intraperitoneal (ip) injection on gestational day (GD) 12, there was no increase 6h later in embryonic ROS formation, measured by 2',7'-dichlorodihydrofluorescin diacetate (DCFH-DA) fluorescence, despite an increase observed with the positive control ethanol (EtOH), nor was there an increase in embryonic oxidatively damaged DNA, quantified as 8-oxo-2'-deoxyguanosine (8-oxodG) formation. MeOH teratogenicity (primarily ophthalmic anomalies, cleft palate) also was not altered by pre- and post-treatment with varying doses of the free radical spin trapping agent alpha-phenyl-N-tert-butylnitrone (PBN). In contrast, pretreatment with l-buthionine-(S,R)-sulfoximine (BSO), an inhibitor of glutathione (GSH) synthesis, depleted maternal hepatic and embryonic GSH, and enhanced some new anomalies (micrognathia, agnathia, short snout, fused digits, cleft lip, low set ears), but not the most common teratogenic effects of MeOH (ophthalmic anomalies, cleft palate) in this strain. These results suggest that ROS did not contribute to the teratogenic effects of MeOH in this in vivo mouse model, in contrast to results in embryo culture from our laboratory, and that the protective effect of GSH in this model may arise from its role as a cofactor for formaldehyde dehydrogenase in the detoxification of formaldehyde.


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37. RECOMMENDATIONS FROM A CONSENSUS DEVELOPMENT WORKSHOP ON THE DIAGNOSIS OF FETAL ALCOHOL SPECTRUM DISORDERS IN AUSTRALIA
Rochelle E Watkins1*, Elizabeth J Elliott234, Amanda Wilkins15, Raewyn C Mutch15, James P Fitzpatrick24, Janet M Payne1, Colleen M O’Leary16, Heather M Jones1, Jane Latimer4, Lorian Hayes5, Jane Halliday8, Heather D’Antoine9, Sue Miers10, Elizabeth Russell11, Lucinda Burns12, Anne McKenzie1, Elizabeth Peadon23, Maureen Carter13 and Carol Bower1

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8 Public Health Genetics, Genetic Disorders, Murdoch Childrens Research Institute, Melbourne, Australia
9 Menzies School of Health Research, Charles Darwin University, Darwin, Australia
10 National Organisation for Fetal Alcohol Spectrum Disorders, Adelaide, Australia
11 Russell Family Fetal Alcohol Disorders Association, Cairns, Australia
12 National Drug and Alcohol Research Centre, University of New South Wales, Sydney, Australia
13 Nindilingarri Cultural Health Services, Fitzroy Crossing, Australia

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ABSTRACT

Background: Fetal alcohol spectrum disorders (FASD) are underdiagnosed in Australia, and health professionals have endorsed the need for national guidelines for diagnosis. The aim of this study was to develop consensus recommendations for the diagnosis of FASD in Australia.

Methods: A panel of 13 health professionals, researchers, and consumer and community representatives with relevant expertise attended a 2-day consensus development workshop to review evidence on the screening and diagnosis of FASD obtained from a systematic literature review, a national survey of health professionals and community group discussions. The nominal group technique and facilitated discussion were used to review the evidence on screening and diagnosis, and to develop consensus recommendations for the diagnosis of FASD in Australia.

Results: The use of population-based screening for FASD was not recommended. However, there was consensus support for the development of standard criteria for referral for specialist diagnostic assessment. Participants developed consensus recommendations for diagnostic categories, criteria and assessment methods, based on the adaption of elements from both the University of Washington 4-Digit Diagnostic Code and the Canadian guidelines for FASD diagnosis. Panel members also recommended the development of resources to: facilitate consistency in referral and diagnostic practices, including comprehensive clinical guidelines and assessment instruments; and to support individuals undergoing assessment and their parents or carers.

Conclusions: These consensus recommendations provide a foundation for the development of guidelines and other resources to promote consistency in the diagnosis of FASD in Australia. Guidelines for diagnosis will require review and evaluation in the Australian context prior to national implementation as well as periodic review to incorporate new knowledge.

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causes a significant deficit in synaptic plasticity, namely long-term potentiation (LTP), in the dentate gyrus (DG) region of the hippocampus of male rats. PNEE has also been shown to induce an increase in oxidative stress and a reduction in antioxidant capacity in the brains of both male and female animals. In this study the interaction between LTP and the major antioxidant in the brain, glutathione (GSH), is examined. We show that depletion of the intracellular reserves of GSH with diethyl maleate (DEM) reduces LTP in control male, but not female animals, mirroring the effects of PNEE. Furthermore, treatment of PNEE animals with N-acetyl cysteine (NAC), a cysteine donor for the synthesis of GSH, increases GSH levels in the hippocampus and completely restores the deficits in LTP in PNEE males. These results indicate that in males GSH plays a major role in regulating LTP, and that PNEE may cause reductions in LTP by reducing the intracellular pool of this endogenous antioxidant.

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39. MATERNAL ALCOHOL USE AND SUDDEN INFANT DEATH SYNDROME
No Authors listed

ABSTRACT
No Abstract available

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40. INTEGRATED CARE FOR PREGNANT WOMEN ON METHADONE MAINTENANCE TREATMENT: CANADIAN PRIMARY CARE COHORT STUDY
Department of Family Medicine, St Joseph's Health Centre, 30 The Queensway, Toronto, ON M6R 1B5. ordeaa@stjoe.on.ca.

ABSTRACT
Objective: To describe the characteristics of a national cohort of pregnant women on methadone maintenance treatment (MMT) and to provide treatment outcome data for integrated care programs.

Design: Retrospective chart review.

Setting: Three different integrated care programs in geographically distinct cities: the Toronto Centre for Substance Use in Pregnancy in Toronto, Ont; the Herzl Family Practice Centre in Montreal, Que; and the Sheway clinic in Vancouver, BC.

Participants: Pregnant women meeting criteria for opioid dependence and attending an integrated care program between 1997 and 2009. Women were excluded if they were on MMT only for chronic pain.

Main outcome measures: Patient demographic characteristics, concurrent medical and psychiatric disorders, and substance use outcome data.
RESULTS: A total of 102 opioid-dependent pregnancies were included. The mean age was 29.7 years and 64% of women were white. Women in Montreal were more likely to have partners and had fewer children. Differences in living and housing situations among the sites tended to resolve by the time of delivery. Almost half of this cohort tested positive for hepatitis C. Women had a high prevalence of depression and anxiety across all sites. Half of this cohort was on MMT before conception and for the other half, MMT was initiated at a mean gestational age of 20.7 weeks, resulting in a mean dose of 82.4 mg at delivery. At the first visit, polysubstance use was common. Prescription opioid use was more frequent in Toronto and heroin use was more prevalent in Vancouver and Montreal. For the entire population, significant reductions were found by the time of delivery for illicit (P < .001) and prescription opioids (P = .001), cocaine (P < .001), marijuana (P = .009), and alcohol use (P < .001).

Conclusion: Despite geographic differences, all 3 integrated care programs have been associated with significant decreases in substance use in pregnant opioid-dependent women.


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41. ADOPTED CHILDREN FROM THE FORMER SOVIET UNION: ARE THEY AT RISK OF FETAL ALCOHOL SPECTRUM DISORDER?
Koren G.

ABSTRACT
Question: One of the families in my practice is considering adoption of a 2-year-old child from the former Soviet Union. The family has been reassured by the agency that a doctor will examine the child to rule out developmental delays. However, my understanding from your previous articles is that one cannot rule out fetal alcohol spectrum disorder (FASD) at that age. Are these children at increased risk of developing FASD?

Answer: You are correct: FASD cannot be ruled out at 2 years of age. The risk of FASD, neglect, and abuse among children in orphanages in the former Soviet Union is high. While adoption of children with known developmental delays should be encouraged and supported, most families seek to adopt with the assumption that these children will be healthy.


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42. ASSOCIATION BETWEEN MATERNAL ALCOHOL CONSUMPTION IN EARLY PREGNANCY AND PREGNANCY OUTCOMES
McCarthy FP, O’Keeffe LM, Khashan AS, North RA, Poston L, McCowan LM, Baker PN, Dekker GA, Roberts CT, Walker JJ, Kenny LC. Irish Centre for Fetal and Neonatal Translational Research (INFANT), University College Cork, and the National Perinatal Epidemiology Centre, Cork University Maternity Hospital, Wilton, Cork, Ireland; the Department of Maternal and Fetal Medicine and the Division of Women's Health, Women's Health Academic Centre, King's College London and King's Health Partners, London, United Kingdom; the Department of Obstetrics and Gynaecology,
Faculty of Medical and Health Sciences, and the National Centre for Growth & Development and Maternal and Fetal Health, Liggins Institute, University of Auckland, Auckland, New Zealand; the Women's and Children's Division, Lyell McEwin Hospital, University of Adelaide, and the School of Paediatrics and Reproductive Health, Robinson Institute, University of Adelaide, Adelaide, South Australia; and the Department of Obstetrics and Gynaecology, St James University Hospital, Leeds, United Kingdom.

ABSTRACT

Objective: To investigate the association between alcohol consumption and binge drinking before and during early pregnancy and adverse pregnancy outcomes.

Methods: We used data from 5,628 nulliparous pregnant participants recruited to the Screening for Pregnancy Endpoints (SCOPE) study, a prospective cohort study. Participants were interviewed at 15 weeks of gestation and information on alcohol intake before pregnancy and until the time of interview was obtained using a standardized questionnaire. Alcohol intake was classified as occasional (1-2 units per week), low (3-7 units per week), moderate (8-14 units per week), and heavy (greater than 14 units per week). Binge alcohol consumption was defined as consumption of 6 or more alcohol units in one session.

Results: Of the 5,628 participants, 1,090 (19%) reported occasional alcohol consumption, 1,383 (25%) low alcohol consumption, 625 (11%) moderate alcohol consumption, and 300 (5%) heavy alcohol consumption. Overall, 1,905 (34%) participants reported binge alcohol consumption in the 3 months before pregnancy, and 1,288 (23%) of these participants reported binge alcohol consumption during the first 15 weeks of pregnancy. Participants who consumed occasional to heavy amounts of alcohol in early pregnancy did not have altered odds of a small-for-gestational-age neonate, reduced birth weight, preeclampsia, or spontaneous preterm birth. Similarly, those who binge drank in early pregnancy did not have altered odds of these adverse pregnancy outcomes.

Conclusion: Alcohol consumption in early pregnancy was prevalent in this nulliparous cohort. There was no association between alcohol consumption before 15 weeks of gestation and small for gestational age, reduced birth weight, preeclampsia, or spontaneous preterm birth.

Level of Evidence: II.


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43. NEURODEVELOPMENTAL EPIGENETIC ETIOLOGIES: INSIGHTS FROM STUDIES ON MOUSE MODELS OF FETAL ALCOHOL SPECTRUM DISORDERS
Laufer BI, Diehl EJ, Singh SM.
Molecular Genetics Unit, Department of Biology, Western University, London, ON, N6A 5B7, Canada.

ABSTRACT

No Abstract Available.


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44. CAMKII REPRESSIONS TRANSCRIPTIONALLY-ACTIVE B-CATENIN TO MEDIATE ACUTE ETHANOL NEURODEGENERATION AND CAN PHOSPHORYLATE B-CATENIN
Flentke GR, Garic A, Hernandez M, Smith SM.
Department of Nutritional Sciences, University of Wisconsin-Madison, Madison, WI, 53706.

ABSTRACT
Prenatal ethanol exposure causes persistent neurodevelopmental deficits by inducing apoptosis within neuronal progenitors including the neural crest. The cellular signaling events underlying this apoptosis are unclear. Using an established chick embryo model, we previously identified ethanol's activation of CaMKII as a crucial early step in this pathway. Here we report that CaMKII is pro-apoptotic because it mediates the loss of transcriptionally active β-catenin, which normally provides trophic support to these cells. β-catenin overexpression normalized cell survival in ethanol's presence. CaMKII inhibition similarly restored β-catenin content and transcriptional activity within ethanol-treated cells and prevented their cell death. In contrast, inhibition of alternative effectors known to destabilize β-catenin, including GSK3β, Protein Kinase C, JNK, and calpain, failed to normalize cell survival and β-catenin activity in ethanol's presence. Importantly, we found that purified CaMKII can directly phosphorylate β-catenin. Using targeted mutagenesis we identified CaMKII phosphorylation sites within human β-catenin at T332, T472, and S552. This is the first demonstration that β-catenin is a phosphorylation target of CaMKII and represents a novel mechanism by which calcium signals could regulate β-catenin-dependent transcription. These results inform ethanol's neurotoxicity and offer unexpected insights into other neurodevelopmental and neurodegenerative disorders having dysregulated calcium or β-catenin signaling.


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45. SURVEILLANCE DURING PREGNANCY: METHODS AND RESPONSE RATES FROM A HOSPITAL BASED PILOT STUDY OF THE PREGNANCY RISK ASSESSMENT MONITORING SYSTEM IN IRELAND
O'Keeffe LM, Kearney PM, Greene RA.
National Perinatal Epidemiology Centre, Department of Obstetrics and Gynecology, 5th Floor, Cork University Maternity Hospital, Wilton, Cork, Ireland. l.okeeffe@ucc.ie.

ABSTRACT
Background: Many European countries including Ireland lack high quality, on-going, population based estimates of maternal behaviours and experiences during pregnancy. PRAMS is a CDC surveillance program which was established in the United States in 1987 to generate high quality, population based data to reduce infant mortality rates and improve maternal and infant health. PRAMS is the only on-going population based surveillance system of maternal behaviours and experiences that occur before, during and after pregnancy worldwide.

Methods: The objective of this study was to adapt, test and evaluate a modified CDC PRAMS methodology in Ireland. The birth certificate file which is the standard approach to sampling for PRAMS in the United States was not available for the PRAMS Ireland study. Consequently, delivery record books for the period between 3 and 5 months before the study
start date at a large urban obstetric hospital [8,900 births per year] were used to randomly sample 124 women. Name, address, maternal age, infant sex, gestational age at delivery, delivery method, APGAR score and birth weight were manually extracted from records. Stillbirths and early neonatal deaths were excluded using APGAR scores and hospital records. Women were sent a letter of invitation to participate including option to opt out, followed by a modified PRAMS survey, a reminder letter and a final survey.

**Results:** The response rate for the pilot was 67%. Two per cent of women refused the survey, 7% opted out of the study and 24% did not respond. Survey items were at least 88% complete for all 82 respondents. Prevalence estimates of socially undesirable behaviours such as alcohol consumption during pregnancy were high [>50%] and comparable with international estimates.

**Conclusion:** PRAMS is a feasible and valid method of collecting information on maternal experiences and behaviours during pregnancy in Ireland. PRAMS may offer a potential solution to data deficits in maternal health behaviour indicators in Ireland with further work. This study is important to researchers in Europe and elsewhere who may be interested in new ways of tailoring an established CDC methodology to their unique settings to resolve data deficits in maternal health.

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46. **A WAVELET RELATIONAL FUZZY C-MEANS ALGORITHM FOR 2D GEL IMAGE SEGMENTATION**
Rashwan S, Faheem MT, Sarhan A, Youssef BA.
Informatics Research Institute, City for Scientific Research and Technological Applications, Borg El Arab, Alexandria, Egypt.

**ABSTRACT**

One of the most famous algorithms that appeared in the area of image segmentation is the Fuzzy C-Means (FCM) algorithm. This algorithm has been used in many applications such as data analysis, pattern recognition, and image segmentation. It has the advantages of producing high quality segmentation compared to the other available algorithms. Many modifications have been made to the algorithm to improve its segmentation quality. The proposed segmentation algorithm in this paper is based on the Fuzzy C-Means algorithm adding the relational fuzzy notion and the wavelet transform to it so as to enhance its performance especially in the area of 2D gel images. Both proposed modifications aim to minimize the oversegmentation error incurred by previous algorithms. The experimental results of comparing both the Fuzzy C-Means (FCM) and the Wavelet Fuzzy C-Means (WFCM) to the proposed algorithm on real 2D gel images acquired from human leukemias, HL-60 cell lines, and fetal alcohol syndrome (FAS) demonstrate the improvement achieved by the proposed algorithm in overcoming the segmentation error. In addition, we investigate the effect of denoising on the three algorithms. This investigation proves that denoising the 2D gel image before segmentation can improve (in most of the cases) the quality of the segmentation.

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47. PRENATAL ALCOHOL EXPOSURE AND OFFSPRING COGNITION AND SCHOOL PERFORMANCE. A 'MENDELIAN RANDOMIZATION' NATURAL EXPERIMENT

MRC Integrative Epidemiology Unit, University of Bristol, Bristol, UK, School of Social and Community Medicine, University of Bristol, Bristol, UK, Section of Developmental Psychiatry, University of Nottingham, Nottingham, UK, Department of Health Sciences, University of Leicester, Leicester, UK, School of Medicine, University of Sheffield, Sheffield, UK, Clinical Genetics, University Hospitals of Leicester, Leicester, UK, School of Population Health, University of Queensland, Brisbane, Queensland, Australia and National Perinatal Epidemiology Unit, University of Oxford, Oxford, UK.

ABSTRACT

Background: There is substantial debate as to whether moderate alcohol use during pregnancy could have subtle but important effects on offspring, by impairing later cognitive function and thus school performance. The authors aimed to investigate the unconfounded effect of moderately increased prenatal alcohol exposure on cognitive/educational performance.

Methods: We used mother-offspring pairs participating in the Avon Longitudinal Study of Parents and Children (ALSPAC) and performed both conventional observational analyses and Mendelian randomization using an ADH1B variant (rs1229984) associated with reduced alcohol consumption. Women of White European origin with genotype and self-reported prenatal alcohol consumption, whose offspring's IQ score had been assessed in clinic (N = 4061 pairs) or Key Stage 2 (KS2) academic achievement score was available through linkage to the National Pupil Database (N = 6268), contributed to the analyses.

Results: Women reporting moderate drinking before and during early pregnancy were relatively affluent compared with women reporting lighter drinking, and their children had higher KS2 and IQ scores. In contrast, children whose mothers’ genotype predisposes to lower consumption or abstinence during early pregnancy had higher KS2 scores (mean difference +1.7, 95% confidence interval +0.4, +3.0) than children of mothers whose genotype predisposed to heavier drinking, after adjustment for population stratification.

Conclusions: Better offspring cognitive/educational outcomes observed in association with prenatal alcohol exposure presumably reflected residual confounding by factors associated with social position and maternal education. The unconfounded Mendelian randomization estimates suggest a small but potentially important detrimental effect of small increases in prenatal alcohol exposure, at least on educational outcomes.


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ABSTRACT
There are no biological treatments for fetal alcohol spectrum disorders (FASDs), lifelong conditions associated with physical anomalies, brain damage, and neurocognitive abnormalities. In preclinical studies, choline partially ameliorates memory and learning deficits from prenatal alcohol exposure. This phase I pilot study evaluated the feasibility, tolerability, and potential adverse effects of choline supplementation in children with FASD. We hypothesized that choline would be well tolerated with minimal adverse events. The study design was a double-blind, randomized, placebo-controlled trial. Participants included 20 children aged 2.5 to 4.9 years with prenatal alcohol exposure and FASD diagnoses. Participants were randomly assigned to 500 mg choline or placebo daily for 9 months (10 active, 10 placebo). Primary outcome measures included feasibility, tolerability, adverse effects, and serum choline levels. Seventeen participants completed the study. Compliance was 82% to 87%, as evidenced by parent-completed log sheets and dose counts. Periodic 24-hour dietary recalls showed no evidence of dietary confounding. Adverse events were minimal and were equivalent in the active and placebo arms with the exception of fishy body odor, which occurred only in the active group. There were no serious adverse events to research participants. This phase I pilot study demonstrates that choline supplementation at 500 mg/d for 9 months in children aged 2 to 5 years is feasible and has high tolerability. Further examination of the efficacy of choline supplementation in FASD is currently underway.


49. SPATIAL COGNITION AND SEXUALLY DIMORPHIC SYNAPTIC PLASTICITY BALANCE IMPAIRMENT IN RATS WITH CHRONIC PRENATAL ETHANOL EXPOSURE
An L, Zhang T.
College of Life Sciences, Nankai University, 300071 Tianjin, PR China.

ABSTRACT
Prenatal ethanol exposure can lead to long-lasting impairments in the ability of rats to process spatial information, as well as produce long-lasting deficits in long-term potentiation (LTP), a biological model of learning and memory processing. The present study aimed to examine the sexually dimorphic effects of chronic prenatal ethanol exposure (CPEE) on behavior cognition and synaptic plasticity balance (SPB), and tried to understand a possible mechanism by evaluating the alternation of SPB. The animal model was produced by ethanol exposure throughout gestational period with 4g/kg bodyweight. Offspring of both male and female were selected and studied on postnatal days 36. Subsequently, the data showed that chronic ethanol exposure resulted in birth weight reduction, losing bodyweight gain, microcephaly and hippocampus weight retardation. In Morris water maze (MWM) test, escape latencies were significantly higher in CPEE-treated rats than that in control ones. They also spent much less time in the target quadrant compared to that of control animals in the probe phase. In addition, it was found that there was a more severe impairment in females than that in males after CPEE treatment. Electrophysiological studies showed that CPEE considerably inhibited hippocampal LTP and facilitated depotentiation in males, while significantly enhanced LTP and suppressed depotentiation in females. A novel index, developed by us, showed that the action of CPEE on SPB was more sensitive in females than that in males, suggesting that it might be an effective index to distinguish the difference of SPB impairment between males and females.

50. NEUROPSYCHOLOGICAL DEFICITS ASSOCIATED WITH HEAVY PRENATAL ALCOHOL EXPOSURE ARE NOT EXACERBATED BY ADHD

Glass L, Ware AL, Crocker N, Deweese BN, Coles CD, Kable JA, May PA, Kalberg WO, Sowell ER, Jones KL, Riley EP, Mattson SN.

Center for Behavioral Teratology, Department of Psychology, San Diego State University.

ABSTRACT

Objective: Neuropsychological functioning of individuals with attention-deficit/hyperactivity disorder (ADHD) or heavy prenatal alcohol exposure has been well documented independently. This study examined the interaction between both factors on cognitive performance in children.

Method: As part of a multisite study, 344 children (8-16 y, M = 12.28, SD = 2.52) completed a comprehensive neuropsychological battery. Four subject groups were tested: children with histories of heavy prenatal alcohol exposure (AE) and ADHD (AE+, n = 90), alcohol-exposed without ADHD, (AE-, n = 38), nonexposed with ADHD (ADHD, n = 80), and nonexposed without ADHD (CON, n = 136).

Results: Separate 2(AE) × 2(ADHD) MANCOVAs revealed significant main and interactive effects of ADHD and AE on overall WISC-IV, D-KEFS, and CANTAB performance. Individual ANOVAs revealed significant interactions on 2 WISC-IV indices [Verbal Comprehension (VCI), Perceptual Reasoning (PRI)], and four D-KEFS and CANTAB subtests [Design Fluency, Verbal Fluency, Trail Making, Spatial Working Memory]. Follow-up analyses demonstrated no difference between AE+ and AE- groups on these measures. The combined AE+/ group demonstrated more severe impairment than the ADHD group on VCI and PRI, but there were no other differences between clinical groups.

Conclusions: These results support a combined AE+/ group for neuropsychological research and indicate that, in some cases, the neuropsychological effects seen in ADHD are altered by prenatal alcohol exposure. The effects of alcohol exposure on verbal comprehension and perceptual reasoning were greater than those related to having ADHD without alcohol exposure, although both conditions independently resulted in cognitive impairment compared to controls. Clinically, these findings demonstrate task-dependent patterns of impairment across clinical disorders.


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51. TOBACCO, ALCOHOL AND CANNABIS USE DURING PREGNANCY: CLUSTERING OF RISKS

Passey ME, Sanson-Fisher RW, D’Este CA, Stirling JM.

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ABSTRACT

Background: Antenatal substance use poses significant risks to the unborn child. We examined use of tobacco, alcohol and cannabis among pregnant Aboriginal and Torres Strait
Islander women; and compared characteristics of women by the number of substances reported.

**Methods:** A cross-sectional survey with 257 pregnant Indigenous women attending antenatal services in two states of Australia. Women self-reported tobacco, alcohol and cannabis use (current use, ever use, changes during pregnancy); age of initiation of each substance; demographic and obstetric characteristics.

**Results:** Nearly half the women (120; 47% (95%CI:40%, 53%) reported no current substance use; 119 reported current tobacco (46%; 95%CI:40%, 53%), 53 (21%; 95%CI:16%, 26%) current alcohol and 38 (15%; 95%CI:11%, 20%) current cannabis use. Among 148 women smoking tobacco at the beginning of pregnancy, 29 (20%; 95%CI:14%, 27%) reported quitting; with 80 of 133 (60%; 95%CI:51%, 69%) women quitting alcohol and 25 of 63 (40%; 95%CI:28%, 53%) women quitting cannabis. Among 137 women reporting current substance use, 77 (56%; 95%CI:47%, 65%) reported one and 60 (44%; 95%CI:35%, 53%) reported two or three. Women using any one substance were significantly more likely to also use others. Factors independently associated with current use of multiple substances were years of schooling and age of initiating tobacco.

**Conclusions:** While many women discontinue substance use when becoming pregnant, there is clustering of risk among a small group of disadvantaged women. Programmes should address risks holistically within the social realities of women's lives rather than focusing on individual tobacco smoking. Preventing uptake of substance use is critical.


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**52. FATE ANALYSIS OF ADULT HIPPOCAMPAL PROGENITORS IN A MURINE MODEL OF FETAL ALCOHOL SPECTRUM DISORDER (FASD)**

Kenta Kajimoto, Andrea Allan, Lee Anna Cunningham

**ABSTRACT**

Prenatal alcohol exposure can lead to fetal alcohol spectrum disorder (FASD) and associated behavioral impairments that may be linked to disruptions in adult hippocampal neurogenesis. Social and physical enrichment has been proposed as a potential therapeutic approach toward reversing behavioral deficits associated with FASD and is also a potent stimulator of adult hippocampal neurogenesis. In the present study, we utilized a genetic fate mapping approach in nestin-CreER<sup>12</sup>/YFP bitransgenic mice to identify the stage-specific impact of prenatal alcohol exposure on the stepwise maturation of adult hippocampal progenitors. Using a limited alcohol access “drinking-in-the-dark” model of FASD, we confirm previous findings that moderate prenatal alcohol exposure has no effect on adult neurogenesis under standard housing conditions, but abolishes the neurogenic response to enriched environment (EE). Furthermore, we demonstrate that this effect is primarily due to failed EE-mediated survival of postmitotic neurons. Finally, we demonstrate that the neurogenic deficit is associated with impaired spatial pattern recognition, as demonstrated by delayed learning of FASD-EE mice in an A–B contextual discrimination task. These results identify a potential maturational stage-specific mechanism(s) underlying impaired neurogenic function in a preclinical model of FASD, and provide a basis for testing regulatory pathways in this model through conditional and inducible manipulation of gene expression in the adult hippocampal progenitor population.


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53. CLINICAL CORRELATES OF FETAL ALCOHOL SPECTRUM DISORDER AMONG DIAGNOSED INDIVIDUALS IN A RURAL DIAGNOSTIC CLINIC
Mansfield Mela, Audrey McFarlane, Tolulope T Sajobi, Hasu Rajani

ABSTRACT
Background: Diagnosis of fetal alcohol spectrum disorder (FASD) is relevant for the reduction of long term adverse sequelae. However, the diagnostic guidelines require a multidisciplinary approach which may hinder access to diagnostic and management services. Most diagnostic clinics are located in urban areas. There is less emphasis on the operations, capacities, and outcomes from rural diagnostic clinics.

Methods: Over a ten and half years of clinic operations to diagnose children and subsequently adults, all consenting adults provided answers to interviews, participated in measurements and other diagnostic procedures. Information was collected on their contact with mental health services. Comparison of the findings with those from other established clinics included variables relevant to outcome measures.

Results: 375 individuals were referred, assessed and diagnosed according to the existing guidelines for FASD diagnosis. Alcohol-related neurodevelopmental disorder (ARND), which was closely associated with age, was the most prevalent FASD diagnosis. One third of those diagnosed had IQ above the average range and ADHD was the most relevant clinical correlate. The diagnostic clinic was able to complete diagnosis on potentially 37.5% of likely affected individuals.

Conclusion: FASD can be diagnosed in children and adults in a rural setting. ADHD and other mental disorders should be a focus for treatment in affected individuals especially adults. It is important to consider the impact of age on the outcome of FASD. To increase diagnostic capacity, clinic operations could be modelled similarly.

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54. PHARMACOKINETICS OF ETHANOL IN THE MATERNAL-FETAL UNIT
Irene Zelner, Gideon Koren

ABSTRACT
Due to its wide range of deleterious effects on the unborn baby, knowledge on the disposition of ethanol in the maternal-fetal unit is critical. This review summarizes and updates the existing evidence on ethanol disposition in the mother, the placenta and the fetus, and relates them to their potential fetal effects.

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55. BRIEF INTERVENTION AND DECREASE OF ALCOHOL CONSUMPTION AMONG WOMEN: A SYSTEMATIC REVIEW
Gebara CF, Bhona FM, Ronzani TM, Lourenço LM, Noto AR.
Department of Psychobiology, Research Center on Health and Substance Use (NEPSIS), Universidade Federal de São Paulo (UNIFESP), Rua Botucatu, 862 - 1° andar, 04023-062 São Paulo - SP, Brazil. carla_gebara@yahoo.com.br.

ABSTRACT
Problems related to alcohol consumption are priority public health issues worldwide and may compromise women's health. The early detection of risky alcohol consumption combined with a brief intervention (BI) has shown promising results in prevention for different populations. The aim of this study was to examine data from recent scientific publications on the use of BI toward reducing alcohol consumption among women through a systematic review. Electronic searches were conducted using Web of Science, PubMed(Medline) and PsycInfo databases. In all databases, the term "brief intervention" was associated with the words "alcohol" and "women", and studies published between the years 2006 and 2011 were selected. Out of the 133 publications found, the 36 scientific articles whose central theme was performing and/or evaluating the effectiveness of BI were included. The full texts were reviewed by content analysis technique. This review identified promising results of BI for women, especially pregnant women and female college students, in different forms of application (face-to-face, by computer or telephone) despite a substantial heterogeneity in the clinical trials analyzed. In primary care, which is a setting involving quite different characteristics, the results among women were rather unclear. In general, the results indicated a decrease in alcohol consumption among women following BI, both in the number of days of consumption and the number of doses, suggesting that the impact on the woman's reproductive health and the lower social acceptance of female consumption can be aspects favorable for the effectiveness of BI in this population.

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56. PREVALENCE OF FETAL ALCOHOL SPECTRUM DISORDERS IN CHILD CARE SETTINGS: A META-ANALYSIS
Lange S, Shield K, Rehm J, Popova S.
Social and Epidemiological Research Department, Centre for Addiction and Mental Health, 33 Russell St, Toronto ON Canada M5S 2S1. lana.popova@camh.ca.

ABSTRACT
Background: Children often enter a child-care system (eg, orphanage, foster care, child welfare system) because of unfavorable circumstances (eg, maternal alcohol and/or drug problems, child abuse/neglect). Such circumstances increase the odds of prenatal alcohol exposure, and thus this population can be regarded as high risk for fetal alcohol spectrum disorders (FASD). The primary objective was to estimate a pooled prevalence for fetal alcohol syndrome (FAS) and FASD in various child-care systems based on data from existing studies that used an active case ascertainment method.

Methods: A systematic literature review, using multiple electronic bibliographic databases, and meta-analysis of internationally published and unpublished studies that reported the prevalence of FAS and/or FASD in all types of child-care systems were conducted. The
pooled prevalence estimates and 95% confidence intervals (CIs) were calculated by using the Mantel-Haenszel method, assuming a random effects model. Sensitivity analyses were performed for studies that used either passive surveillance or mixed methods.

**Results:** On the basis of studies that used active case ascertainment, the overall pooled prevalence of FAS and FASD among children and youth in the care of a child-care system was calculated to be 6.0% (60 per 1000; 95% CI: 38 to 85 per 1000) and 16.9% (169 per 1000; 95% CI: 109 to 238 per 1000), respectively.

**Conclusions:** The results confirm that children and youth housed in or under the guardianship of the wide range of child-care systems constitute a population that is high-risk for FASD. It is imperative that screening be implemented in these at-risk populations.

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57. **IDENTIFICATION OF THE PREGNANT WOMAN WHO IS USING DRUGS: IMPLICATIONS FOR PERINATAL AND NEONATAL CARE**
Casper T, Arbour MW.

**ABSTRACT**
Neonatal abstinence syndrome (NAS) is a set of drug withdrawal symptoms that affect the central nervous, gastrointestinal, and respiratory systems in the newborn when separated from the placenta at birth. Maternal substance use of opioids, benzodiazepines, barbiturates, and alcohol can cause NAS. Universal drug screening via questioning pregnant women is recommended, but identification of drug use is incomplete with this method. This article provides resources for the identification and management of drug use during pregnancy for midwives who provide care not only during the prenatal period but also during the intrapartum and postpartum periods. The impact of drug use on newborns can be significant and may require pharmacologic assistance with the transition to extrauterine life. Challenges involved in caring for the woman who is using drugs during pregnancy include ordering toxicology screens on the newborn, alerting social services, and educating the woman about her newborn's progress. Several measures to comfort a newborn with NAS may help to enable a mother to provide the best care for her newborn.

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J Popul Ther Clin Pharmacol Vol 20(3):e212-e228; September 6, 2013

58. **SENSORY CONTROL OF BALANCE: A COMPARISON OF CHILDREN WITH FETAL ALCOHOL SPECTRUM DISORDERS TO CHILDREN WITH TYPICAL DEVELOPMENT**
Tracy L Jirikowic, Sarah W McCoy, Anat Lubetzky-Vilnai, Robert Price, Marcia A Ciol, Deborah Kartin, Lin-Ya Hsu, Beth Gendler, Susan J Astley

**ABSTRACT**
Background: Inefficient central processing and integration of visual, vestibular, and somatosensory information may contribute to poor balance and diminished postural control in
children with fetal alcohol spectrum disorders (FASD).

**Objectives:** This pilot study examined sensorimotor performance and the sensory control of balance using a battery of clinical tests in combination with an experimental laboratory assessment that quantifies sensory subsystem use (i.e., sensory weighting) among a systematically diagnosed sample of children with FASD and children with typical development.

**Methods:** Using a case-control design, 10 children with FASD (8.0-15.9 years; 20% female) were compared to 10 age- and sex-matched controls on standardized clinical measures and on kinematic outcomes from the Multimodal Balance Entrainment Response system (MuMBER), a computerized laboratory assessment whereby visual, vestibular, and somatosensory input is manipulated at different frequencies during standing balance.

**Results:** Children with FASD showed poorer sensorimotor performance across clinical outcomes with significant group differences (p < .05) on parent-reported movement behaviors (Sensory Processing Measure and Movement Assessment Battery for Children-2 Checklist) and performance on the Dynamic Gait Index. Experimental kinematic outcomes yielded statistically significant group differences (p < .10) on a small proportion of somatosensory and vestibular sensory weighting fractions and postural sway velocity in response to the manipulation of sensory input.

**Conclusions:** Preliminary findings showed small group differences in sensorimotor and sensory weighting behaviors, specifically those that rely on the integration of vestibular sensation. Differences must be examined and replicated with a larger sample of children with FASD to understand the impact on balance control and functional sensorimotor behaviors.

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59. **DEVELOPMENT OF AUDITORY EVENT-RELATED POTENTIALS IN INFANTS PRENATALLY EXPOSED TO METHADONE**

Paul JA, Logan BA, Krishnan R, Heller NA, Morrison DG, Pritham UA, Tisher PW, Troese M, Brown MS, Hayes MJ.

Department of Obstetrics/Gynecology, University of Texas Medical Branch, Galveston, TX, 77555.

**ABSTRACT**

Developmental features of the P2 auditory ERP in a change detection paradigm were examined in infants prenatally exposed to methadone. Opiate dependent pregnant women maintained on methadone replacement therapy were recruited during pregnancy (N = 60). Current and historical alcohol and substance use, SES, and psychiatric status were assessed with a maternal interview during the third trimester. Medical records were used to collect information regarding maternal medications, monthly urinalysis, and breathalyzer to confirm comorbid drug and alcohol exposures. Between birth and 4 months infant ERP change detection performance was evaluated on one occasion with the oddball paradigm (.2 probability oddball) using pure-tone stimuli (standard = 1 kHz and oddball = 2 kHz frequency) at midline electrode sites, Fz, Cz, Pz. Infant groups were examined in the following developmental windows: 4-15, 16-32, or 33-120 days PNA. Older groups showed increased P2 amplitude at Fz and effective change detection performance at P2 not seen in the newborn group. Developmental maturation of amplitude and stimulus discrimination for P2 has been reported in developing infants at all of the ages tested and data reported here in the
older infants are consistent with typical development. However, it has been previously reported that the P2 amplitude difference is detectable in neonates; therefore, absence of a difference in P2 amplitude between stimuli in the 4-15 days group may represent impaired ERP performance by neonatal abstinence syndrome or prenatal methadone exposure.


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60. THE BRUININKS-OSERETSKY TEST OF MOTOR PROFICIENCY-SHORT FORM IS RELIABLE IN CHILDREN LIVING IN REMOTE AUSTRALIAN ABORIGINAL COMMUNITIES

Barbara R Lucas1234*, Jane Latimer26, Robyn Doney5, Manuela L Ferreira26, Roger Adams7, Genevieve Hawkes8, James P Fitzpatrick26, Marminge Hand9, June Oscar109, Maureen Carter11 and Elizabeth J Elliott11226

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7 School of Physiotherapy, University of Sydney, Sydney, Australia
8 Western Australia Country Health Services, Derby, Australia
9 University of Notre Dame, Broome, Australia
10 Marninwarntikura Women’s Resource Centre, Fitzroy Crossing, Australia
11 Nindilingarri Cultural Health Services, Fitzroy Crossing, Australia
12 The Sydney Children’s Hospital Networks (Westmead), Westmead, Australia

ABSTRACT

Background: The Lililwan Project is the first population-based study to determine Fetal Alcohol Spectrum Disorders (FASD) prevalence in Australia and was conducted in the remote Fitzroy Valley in North Western Australia. The diagnostic process for FASD requires accurate assessment of gross and fine motor functioning using standardised cut-offs for impairment. The Bruininks-Oseretsky Test of Motor Proficiency, Second Edition (BOT-2) is a norm-referenced assessment of motor function used worldwide and in FASD clinics in North America. It is available in a Complete Form with 53 items or a Short Form with 14 items. Its reliability in measuring motor performance in children exposed to alcohol in utero or living in remote Australian Aboriginal communities is unknown.

Methods: A prospective inter-rater and test-retest reliability study was conducted using the BOT-2 Short Form. A convenience sample of children (n = 30) aged 7 to 9 years participating in the Lililwan Project cohort (n = 108) study, completed the reliability study. Over 50% of mothers of Lililwan Project children drank alcohol during pregnancy. Two raters simultaneously scoring each child determined inter-rater reliability. Test-retest reliability was determined by assessing each child on a second occasion using predominantly the same rater. Reliability was analysed by calculating Intra-Class correlation Coefficients, ICC(2,1), Percentage Exact Agreement (PEA) and Percentage Close Agreement (PCA) and measures...
of Minimal Detectable Change (MDC) were calculated.

**Results:** Thirty Aboriginal children (18 male, 12 female: mean age 8.8 years) were assessed at eight remote Fitzroy Valley communities. The inter-rater reliability for the BOT-2 Short Form score sheet outcomes ranged from 0.88 (95% CI, 0.77 – 0.94) to 0.92 (95% CI, 0.84 – 0.96) indicating excellent reliability. The test-retest reliability (median interval between tests being 45.5 days) for the BOT-2 Short Form score sheet outcomes ranged from 0.62 (95% CI, 0.34 – 0.80) to 0.73 (95% CI, 0.50 – 0.86) indicating fair to good reliability. The raw score MDC was 6.12.

**Conclusion:** The BOT-2 Short Form has acceptable reliability for use in remote Australian Aboriginal communities and will be useful in determining motor deficits in children exposed to alcohol prenatally. This is the first known study evaluating the reliability of the BOT-2 Short Form, either in the context of assessment for FASD or in Aboriginal children.

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61. **PRENATAL EXPOSURES AND ANTI-MULLERIAN HORMONE IN FEMALE ADOLESCENTS: THE AVON LONGITUDINAL STUDY OF PARENTS AND CHILDREN**

**ABSTRACT**

Given that the primordial ovarian follicular pool is established in utero, it may be influenced by parental characteristics and the intrauterine environment. Anti-Müllerian hormone (AMH) levels are increasingly recognized as a biomarker of ovarian reserve in females in adulthood and adolescence. We examined and compared associations of maternal and paternal prenatal exposures with AMH levels in adolescent (mean age, 15.4 years) female offspring (n = 1,399) using data from the Avon Longitudinal Study of Parents and Children, a United Kingdom birth cohort study that originated in 1991 and is still ongoing (data are from 1991-2008). The median AMH level was 3.67 ng/mL (interquartile range: 2.46-5.57). Paternal but not maternal smoking prior to and during pregnancy were inversely associated with AMH levels. No or irregular maternal menstrual cycles before pregnancy were associated with higher AMH levels in daughter during adolescence. High maternal gestational weight gain (top fifth versus the rest of the distribution) was associated with lower AMH levels in daughters. Parental age, body mass index, and alcohol intake during pregnancy, child's birth weight, and maternal parity and time to conception were not associated with daughters' AMH levels. Our results suggest that some parental preconceptual characteristics and environmental exposures while the child is in utero may influence the long-term ovarian development and function in female offspring.

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62. **EXPOSURE TO ETHANOL ON PRENATAL DAYS 19–20 INCREASES ETHANOL INTAKE AND PALATABILITY IN THE INFANT RAT: INVOLVEMENT OF KAPPA AND MU OPIOID RECEPTORS**

Elena Díaz-Cenzano, Mirari Gaztañaga, M. Gabriela Chotro
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* Correspondence to: M. Gabriela Chotro, E-mail: g.chotro@ehu.es

**ABSTRACT**

Prenatal exposure to ethanol on gestation Days 19–20, but not 17–18, increases ethanol acceptance in infant rats. This effect seems to be a conditioned response acquired prenatally, mediated by the opioid system, which could be stimulated by ethanol's pharmacological properties (mu-opioid receptors) or by a component of the amniotic fluid from gestation-day 20 (kappa-inducing factor). The latter option was evaluated administering non-ethanol chemosensory stimuli on gestation Days 19–20 and testing postnatal intake and palatability. However, prenatal exposure to anise or vanilla increased neither intake nor palatability of these tastants on postnatal Day 14. In experiment 2, the role of ethanol's pharmacological effect was tested by administering ethanol and selective antagonists of mu and kappa opioid receptors prenatally. Blocking the mu-opioid receptor system completely reversed the effects on intake and palatability, while antagonizing kappa receptors only partially reduced the effects on palatability. This suggests that the pharmacological effect of ethanol on the fetal mu opioid system is the appetitive reinforcer, which induces the prenatally conditioned preference detected in the preweanling period.

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http://onlinelibrary.wiley.com/doi/10.1002/dev.21162/abstract;jsessionid=09C7D45BEABAB836C465FD412AB790DD.f01t03

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63. **COMPLEX CARDIAC DEFECTS AFTER ETHANOL EXPOSURE DURING DISCRETE CARDIOGENIC EVENTS IN ZEBRAFISH: PREVENTION WITH FOLIC ACID**

Swapnalee Sarmah, James A. Marrs
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**ABSTRACT**

**Background:** Fetal alcohol spectrum disorder (FASD) describes a range of birth defects including various congenital heart defects (CHDs). Mechanisms of FASD-associated CHDs are not understood. Whether alcohol interferes with a single critical event or with multiple events in heart formation is not known.
**Results:** Our zebrafish embryo experiments showed that ethanol interrupts different cardiac regulatory networks and perturbs multiple steps of cardiogenesis (specification, myocardial migration, looping, chamber morphogenesis, and endocardial cushion formation). Ethanol exposure during gastrulation until cardiac specification or during myocardial midline migration did not produce severe or persistent heart development defects. However, exposure comprising gastrulation until myocardial precursor midline fusion or during heart patterning stages produced aberrant heart looping and defective endocardial cushions. Continuous exposure during entire cardiogenesis produced complex cardiac defects leading to severely defective myocardium, endocardium, and endocardial cushions. Supplementation of retinoic acid with ethanol partially rescued early heart developmental defects, but the endocardial cushions did not form correctly. In contrast, supplementation of folic acid rescued normal heart development, including the endocardial cushions.

**Conclusions:** Our results indicate that ethanol exposure interrupted divergent cardiac morphogenetic events causing heart defects. Folic acid supplementation was effective in preventing a wide spectrum of ethanol-induced heart developmental defects.


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64. PREVENTION OF FETAL ALCOHOL SYNDROME
Fröschl B, Brunner-Ziegler S, Wirl C.

**ABSTRACT**
The fetal alcohol syndrome (FAS) is the most avoidable handicap of newborns. It describes prenatal damages which result from the alcohol consumption of the mother. These can be: reduced body length and weight (pre- and postnatal), microcephaly, musculoskeletal, mental and statomotoric developmental retardations and impaired coordinative ability. There are preventive measures of which the efficiency is examined. Already, short counseling interviews, so-called short interventions, increase the abstinence of pregnant women.


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65. CHANGES IN ALCOHOL CONSUMPTION IN PREGNANT AUSTRALIAN WOMEN BETWEEN 2007 AND 2011
Cate M Cameron, PhD, MPH, BSoCwK(Hons), Senior Research Fellow\(^1,2\)
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Elizabeth Kendall, BA, DipPsych, PhD, Professor\(^1,2\) and Associate Director\(^3\)
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Correspondence: cate.cameron@griffith.edu.au
ABSTRACT
Objective: To describe the prevalence and distribution of alcohol consumption during pregnancy in an Australian population over a 5-year period.

Design, setting and participants: Cross-sectional repeated sample, trend analysis of aggregated and stratified alcohol consumption patterns during pregnancy. Pregnant women were enrolled from 2007 to 2011 in the Griffith Study of Population Health: Environments for Healthy Living, a birth cohort study being conducted in south-east Queensland and north-east New South Wales.

Main outcome measures: Sociodemographic and alcohol consumption data were self-reported at enrolment. Alcohol measures included alcohol consumption (any level) and high-risk alcohol consumption, both during pregnancy (at any stage) and after the first trimester of pregnancy.

Results: Of 2731 pregnant women for whom alcohol consumption data were available, a decrease in alcohol consumption was observed over the study period; 52.8% reported alcohol use in 2007 compared with 34.8% in 2011 (P < 0.001). The proportion of women who drank alcohol after the first trimester of pregnancy declined from 42.2% in 2007 to 25.8% in 2011. However, high-risk drinking patterns — at all or after the first trimester — did not change over the 5 years (P = 0.12). Low-level alcohol consumption was associated with older women (P < 0.001), more highly educated women (P = 0.01), and women from higher-income households (P < 0.001). In contrast, high-risk consumption after the first trimester was associated with lower levels of education (P = 0.011) and single-parent status (P = 0.001).

Conclusions: This study showed a steady and statistically significant decline in the proportion of women who reported drinking alcohol during pregnancy from 2007 to 2011. To further reduce these levels, we need broad public health messages for the general population and localised strategies for high-risk subpopulations.

Trial registration: Australian New Zealand Clinical Trials Registry ACTRN12610000931077.
ABSTRACT
Handwriting is a critical skill for school success. Children with fetal alcohol spectrum disorders (FASD) often present with fine motor and visual–motor impairments that can affect handwriting performance, yet handwriting skills have not been systematically investigated in this clinical group. This study aimed to comprehensively describe handwriting skills in 20 school-age children with FASD. Children were tested with the Process Assessment of the Learner, 2nd Edition (PAL–II), and the Visuomotor Precision subtest of NEPSY, a developmental neuropsychological assessment. Participants performed below average on PAL–II measures of handwriting legibility and speed and on NEPSY visual–motor precision tasks. In contrast, PAL–II measures of sensorimotor skills were broadly within the average range. Results provide evidence of functional handwriting challenges for children with FASD and suggest diminished visual–motor skills and increased difficulty as task complexity increases. Future research is needed to further describe the prevalence and nature of handwriting challenges in this population.

Read Full Article, http://ajot.aotapress.net/content/67/5/534.abstract

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67. PRENATAL ETHANOL EXPOSURE HAS SEX-SPECIFIC EFFECTS ON HIPPOCAMPAL LONG-TERM POTENTIATION
Sickmann HM, Patten AR, Morch K, Sawchuk S, Zhang C, Parton R, Szlavik L, Christie BR. Division of Medical Sciences, University of Victoria, Victoria, BC, Canada; Department of Drug Design and Pharmacology, Faculty of Health and Medical Sciences, University of Copenhagen, Denmark.

ABSTRACT
Alcohol consumption during pregnancy is deleterious to the developing brain of the fetus and leads to persistent deficits in adulthood. Long-term potentiation (LTP) is a biological model for learning and memory processes and previous evidence has shown that prenatal ethanol exposure (PNEE) affects LTP in a sex specific manner during adolescence. The objective of this study was to determine if there are sex specific differences in adult animals and to elucidate the underlying molecular mechanisms that contribute to these differences. Pregnant Sprague-Dawley dams were assigned to either; liquid ethanol, pair-fed or standard chow diet. In vivo electrophysiology was performed in the hippocampal dentate gyrus (DG) of adult offspring. LTP was induced by administering 400 Hz stimuli. Western blot analysis for glutamine synthetase (GS) and glutamate decarboxylase from tissue of the DG indicated that GS expression was increased following PNEE. Surprisingly, adult females did not show any deficit in N-methyl-d-aspartate (NMDA)-dependent LTP after PNEE. In contrast, males showed a 40% reduction in LTP. It was indicated that glutamine synthetase expression was increased in PNEE females, suggesting that altered excitatory neurotransmitter replenishment may serve as a compensatory mechanism. Ovariectomizing females did not influence LTP in control or PNEE animals, suggesting that circulating estradiol levels do not play a major role in maintaining LTP levels in PNEE females. These results demonstrate the sexually dimorphic effects of PNEE on the ability for the adult brain to elicit LTP in the DG. The mechanisms for these effects are not fully understood, but an increase in glutamine synthetase in females may underlie this phenomenon.


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68. PRENATAL ETHANOL EXPOSURE INCREASES BRAIN CHOLESTEROL CONTENT IN ADULT RATS
Barceló-Coblijn G, Wold LE, Ren J, Murphy EJ.
Department of Pharmacology, Physiology, and Therapeutics, School of Medicine and Health Sciences, University of North Dakota, 501 N. Columbia Rd, Room 3700, Grand Forks, ND, 58202-9037, USA.

ABSTRACT
Fetal alcohol syndrome is the most severe expression of the fetal alcohol spectrum disorders (FASD). Although alterations in fetal and neonate brain fatty acid composition and cholesterol content are known to occur in animal models of FASD, the persistence of these alterations into adulthood is unknown. To address this question, we determined the effect of prenatal ethanol exposure on individual phospholipid class fatty acid composition, individual phospholipid class mass, and cholesterol mass in brains from 25-week-old rats that were exposed to ethanol during gestation beginning at gestational day 2. While total phospholipid mass was unaffected, phosphatidylinositol and cardiolipin mass was decreased 14 and 43 %, respectively. Exposure to prenatal ethanol modestly altered brain phospholipid fatty acid composition, and the most consistent change was a significant 1.1-fold increase in total polyunsaturated fatty acids (PUFA), in the n-3/n-6 ratio, and in the 22:6n-3 content in ethanolamine glycerophospholipids and in phosphatidylserine. In contrast, prenatal ethanol consumption significantly increased brain cholesterol mass 1.4-fold and the phospholipid to cholesterol ratio was significantly increased 1.3-fold. These results indicate that brain cholesterol mass was significantly increased in adult rats exposed prenatally to ethanol, but changes in phospholipid mass and phospholipid fatty acid composition were extremely limited. Importantly, suppression of postnatal ethanol consumption was not sufficient to reverse the large increase in cholesterol observed in the adult rats.

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69. MATERNAL ALCOHOL-USE DISORDER IS ASSOCIATED WITH INCREASED RISK OF SUDDEN INFANT DEATH SYNDROME AND INFANT DEATH FROM OTHER CAUSES
Katrine Strandberg-Larsen
Section of Social Medicine, Department of Public Health, University of Copenhagen, Copenhagen K, Denmark
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Commentary on:

Implications for practice and research
- Women with an alcohol-use disorder should be identified and offered supportive antenatal care services and treatment.
• Alcohol-use disorder is associated with lifestyle and parenting styles that contribute to an increased risk of infant death; the prevention of and effective management of alcohol abuse has the potential of reducing infant deaths, particularly sudden infant death syndrome (SIDS).

• Future research should include alcohol-related diagnosis in fathers and compare associations between maternal and paternal alcohol-related diagnosis and infant mortality in order to disentangle environmental factors from direct intrauterine effects of alcohol consumption on infant and child development.

Read Full Article, http://ebn.bmj.com/content/early/2013/08/30/eb-2013-101376.extract

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70. USING AUTOPSY BRAIN TISSUE TO STUDY ALCOHOL-RELATED BRAIN DAMAGE IN THE GENOMIC AGE
Sutherland GT, Sheedy D, Kril JJ.
Discipline of Pathology, Sydney Medical School, The University of Sydney, Sydney, NSW, Australia.

ABSTRACT
The New South Wales Tissue Resource Centre at the University of Sydney, Australia, is one of the few human brain banks dedicated to the study of the effects of chronic alcoholism. The bank was affiliated in 1994 as a member of the National Network of Brain Banks and also focuses on schizophrenia and healthy control tissue. Alcohol abuse is a major problem worldwide, manifesting in such conditions as fetal alcohol syndrome, adolescent binge drinking, alcohol dependency, and alcoholic neurodegeneration. The latter is also referred to as alcohol-related brain damage (ARBD). The study of postmortem brain tissue is ideally suited to determining the effects of long-term alcohol abuse, but it also makes an important contribution to understanding pathogenesis across the spectrum of alcohol misuse disorders and potentially other neurodegenerative diseases. Tissue from the bank has contributed to 330 peer-reviewed journal articles including 120 related to alcohol research. Using the results of these articles, this review chronicles advances in alcohol-related brain research since 2003, the so-called genomic age. In particular, it concentrates on transcriptomic approaches to the pathogenesis of ARBD and builds on earlier reviews of structural changes (Harper et al. Prog Neuropsychopharmacol Biol Psychiatry 2003;27:951) and proteomics (Matsumoto et al. Expert Rev Proteomics 2007;4:539).


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71. PREVALENCE OF ALCOHOL USE BEFORE AND DURING PREGNANCY AND PREDICTORS OF DRINKING DURING PREGNANCY: A CROSS SECTIONAL STUDY IN SWEDEN
Skagerström J, Alehagen S, Häggström-Nordin E, Arestedt K, Nilsson P.
Department of Medical and Health Sciences, Division of Community Medicine, Linköping University, Linköping, SE-581 83, Sweden. janna.skagerstrom@liu.se.
ABSTRACT

Background: There is a paucity of research on predictors for drinking during pregnancy among women in Sweden and reported prevalence rates differ considerably between studies conducted at different antenatal care centres. Since this knowledge is relevant for preventive work the aim of this study was to investigate these issues using a multicenter approach.

Methods: The study was conducted at 30 antenatal care centers across Sweden from November 2009 to December 2010. All women in pregnancy week 18 or more with a scheduled visit were asked to participate in the study. The questionnaire included questions on sociodemographic data, alcohol consumption prior to and during the pregnancy, tobacco use before and during pregnancy, and social support.

Results: Questionnaires from 1594 women were included in the study. A majority, 84%, of the women reported alcohol consumption the year prior to pregnancy; about 14% were categorized as having hazardous consumption, here defined as a weekly consumption of > 9 standard drinks containing 12 grams of pure alcohol or drinking more than 4 standard drinks at the same occasion. Approximately 6% of the women consumed alcohol at least once after pregnancy recognition, of which 92% never drank more than 1 standard drink at a time. Of the women who were hazardous drinkers before pregnancy, 19% reduced their alcohol consumption when planning their pregnancy compared with 33% of the women with moderate alcohol consumption prior to pregnancy. Factors predicting alcohol consumption during pregnancy were older age, living in a large city, using tobacco during pregnancy, lower score for social support, stronger alcohol habit before pregnancy and higher score for social drinking motives.

Conclusions: The prevalence of drinking during pregnancy is relatively low in Sweden. However, 84% of the women report drinking in the year preceding pregnancy and most of these women continue to drink until pregnancy recognition, which means that they might have consumed alcohol in early pregnancy. Six factors were found to predict alcohol consumption during pregnancy. These factors should be addressed in the work to prevent alcohol-exposed pregnancies.


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Springer Link, Metabolic Brain Disease
December 2013, Volume 28, Issue 4, pp 667-676

72. EXPRESSION OF AUTOPHAGY AND UPR GENES IN THE DEVELOPING BRAIN DURING ETHANOL-SENSITIVE AND RESISTANT PERIODS
Alexander Alimov, Haiping Wang, Mei Liu, Jacqueline A. Frank, Mei Xu, Xiaoming Ou, Jia Luo

ABSTRACT

Fetal alcohol spectrum disorders (FASD) results from ethanol exposure to the developing fetus and is the leading cause of mental retardation. FASD is associated with a broad range of neurobehavioral deficits which may be mediated by ethanol-induced neurodegeneration in the developing brain. An immature brain is more susceptible to ethanol neurotoxicity. We hypothesize that the enhanced sensitivity of the immature brain to ethanol is due to a limited capacity to alleviate cellular stress. Using a third trimester equivalent mouse model of ethanol exposure, we demonstrated that subcutaneous injection of ethanol induced a wide-spread neuroapoptosis in postnatal day 4 (PD4) C57BL/6 mice, but had little effect on the brain of PD12 mice. We analyzed the expression profile of genes regulating apoptosis, and the pathways of ER stress response (also known as unfolded protein response, UPR) and autophagy during these ethanol-sensitive and resistant periods (PD4 versus PD12) using PCR microarray. The expression of pro-apoptotic genes, such as caspase-3, was much
higher on PD4 than PD12; in contrast, the expression of genes that regulate UPR and autophagy, such as atf6, atg4, atg9, atg10, beclin1, bnip3, cebpβ, ctbs, ctss, grp78, ire1α, lamp, lc3 perk, pik3c3, and sqstm1 was significantly higher on PD12 than PD4. These results suggest that the vulnerability of the immature brain to ethanol could result from high expression of pro-apoptotic proteins and a deficiency in the stress responsive system, such as UPR and autophagy.


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73. INFANT EMOTIONAL WITHDRAWAL: A PRECURSOR OF AFFECTIVE AND COGNITIVE DISTURBANCE IN FETAL ALCOHOL SPECTRUM DISORDERS

Molteno CD, Jacobson JL, Carter RC, Dodge NC, Jacobson SW. Department of Psychiatry and Mental Health, Faculty of Health Sciences, University of Cape Town, Cape Town, South Africa.

ABSTRACT

Background:
Our aim was to test the hypothesis that emotional withdrawal is an early indicator of affective disorder in infants heavily exposed prenatally to alcohol, which is independent of alcohol-related effects on mother-infant interaction and temperament and discriminated between children later diagnosed with fetal alcohol syndrome (FAS) and partial FAS (PFAS) and predicted cognitive and affective outcomes at 5 and 9 years.

Methods:
The sample consisted of Cape Coloured (mixed ancestry) infants, whose mothers were interviewed during pregnancy regarding their alcohol consumption using a timeline follow-back approach. Infant emotional withdrawal (n = 85) was assessed on the Alarm Distress Baby Scale at 6.5 months. Mother-infant interaction was evaluated from video recordings during free play and infant feeding at 6.5 months (n = 127). Infant temperament was assessed by maternal report on the EAS Temperament Survey at 13 months (n = 119). Sociodemographic and psychological correlates of maternal alcohol use and infant iron deficiency were examined as potential confounders. The children were diagnosed for FAS/PFAS by expert dysmorphologists at 5 years, cognitive and affective function at 5 and 9 years.

Results:
Prenatal alcohol exposure was associated with increased infant emotional withdrawal and decreased activity, but unrelated to mother-infant interaction or any other temperament measures. Children later diagnosed with FAS and PFAS at 5 years exhibited more emotional withdrawal and less responsivity and activity as infants. Infant withdrawal, responsivity, quality of interaction, and maternal sensitivity also predicted poorer IQ and affective response at 5 and 9 years. When all 4 infant affective measures were examined simultaneously in a regression analysis, only infant emotional withdrawal persisted as a significant predictor of 9-year IQ.

Conclusions:
This study is the first to document a direct effect of fetal alcohol exposure on emotional withdrawal in infancy. These data link prenatal alcohol to a specific aspect of infant affective function not attributable to mother-infant interaction, infant temperament, or other socioemotional aspects of the infant’s environment and identify infant emotional withdrawal as an early indicator of affective disturbance, particularly in children later diagnosed with FAS and PFAS.
74. CHRONIC EXCESSIVE ALCOHOL CONSUMPTION AND MALE FERTILITY: A CASE REPORT ON REVERSIBLE AZOOSPERMIA AND A LITERATURE REVIEW

Bruno Guthauser1,2,* , Florence Boitrelle2,3, Arnaud Plat4, Nicolas Thiercelin5 and Francois Vialard2,3

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ABSTRACT

Aims: The aim of this work was to report on a heavy drinker whose azoospermia was reversed after alcohol withdrawal. We also review the literature on links between alcohol consumption and azoospermia.

Method: This study is a clinical case report and a literature review.

Results: Two years after alcohol withdrawal, a child was born following assisted reproduction technique. Excessive alcohol consumption (i.e. more than 60 g a day) is strongly associated with azoospermia and this condition may be reversible after alcohol withdrawal.

Conclusions: Testicular biopsies should be countra-indicated for heavy drinkers, and in order to increase the chances of obtaining a pregnancy, alcohol abstinence should be encouraged in male with low-to-moderate alcohol intakes.

Read Full Article, http://alcalc.oxfordjournals.org/content/early/2013/08/21/alcalc.agt133.long

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75. ALCOHOL CONSUMPTION AMONG WOMEN

Irene Zelner, Gideon Koren

ABSTRACT

Alcohol (ethanol) consumption in pregnancy is the etiology of fetal alcohol spectrum disorder (FASD), a leading cause of congenital disability worldwide. Hence, any attempt to prevent or manage FASD must start from comprehensive understanding of alcohol consumption by women in general, and by women of reproductive years in particular. This review presents and synthesizes studies conducted worldwide on alcohol consumption by pregnant women, risk factors associated with gestational drinking, as well as doses and definitions of drinking behaviours.
76. **PRENATAL ALCOHOL EXPOSURE IS ASSOCIATED WITH ALTERED SUBCELLULAR DISTRIBUTION OF GLUCOCORTICOID AND MINERALOCORTICOID RECEPTORS IN THE ADOLESCENT MOUSE HIPPOCAMPAL FORMATION**

Kevin K. Caldwell, Samantha L. Goggin, Christina R. Tyler, Andrea M. Allan
Department of Neurosciences, School of Medicine, University of New Mexico Health Sciences Center, Albuquerque, New Mexico

**ABSTRACT**

**Background:** Accumulating evidence indicates that several of the long-term consequences of prenatal alcohol exposure (PAE) are the result of changes in the development and function of cortico-limbic structures, including the hippocampal formation. The glucocorticoid receptor (GR) and mineralocorticoid receptor (MR) are key regulators of hippocampal formation development, structure, and functioning and, thus, are potential mediators of PAE's effects on this brain region. In the present studies, we assessed the impact of PAE on components of corticosteroid signaling pathways in the mouse hippocampal formation.

**Methods:** Throughout pregnancy, mouse dams were offered either 10% (w/v) ethanol sweetened with 0.066% (w/v) saccharin (SAC) or 0.066% (w/v) SAC alone using a limited (4-hour) access, drinking-in-the-dark paradigm. The hippocampal formation was isolated from naïve postnatal day 40 to 50 offspring, and subcellular fractions were prepared. Using immunoblotting techniques, we measured the levels of GR, MR, 11-β-hydroxysteroid dehydrogenase 1 (11β-HSD1), and the FK506-binding proteins 51 (FKBP51, FKBP5) and 52 (FKBP52, FKBP4). Finally, we determined the effect of PAE on context discrimination, a hippocampal-dependent learning/memory task.

**Results:** PAE was associated with reduced MR and elevated GR nuclear localization in the hippocampal formation, whereas cytosolic levels of both receptors were not significantly altered. FKBP51 levels were reduced, while FKBP52 levels were unaltered, and 11β-HSD1 levels were increased in postnuclear fractions isolated from PAE mouse hippocampal formation. These neurochemical alterations were associated with reduced context discrimination.

**Conclusions:** The data support a model in which PAE leads to increased nuclear localization of GRs secondary to reductions in FKBP51 and increases in 11β-HSD1 levels in the adolescent mouse hippocampal formation. Persistent dysregulation of GR subcellular distribution is predicted to damage the hippocampal formation and may underlie many of the effects of PAE on hippocampal-dependent functioning.
77. A STUDY OF CORTICAL MORPHOLOGY IN CHILDREN WITH FETAL ALCOHOL SPECTRUM DISORDERS
François De Guio1*, Jean-François Mangin1, Denis Rivière1, Matthieu Perrot1, Christopher D. Molteno2, Sandra W. Jacobson2,3,4, Ernesta M. Meintjes4,5, Joseph L. Jacobson2,3,4
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ABSTRACT
Prenatal alcohol exposure is responsible for a broad range of brain structural malformations, which can be studied using magnetic resonance imaging (MRI). Advanced MRI methods have emerged to characterize brain abnormalities, but the teratogenic effects of alcohol on cortical morphology have received little attention to date. Twenty-four 9-year-old children with fetal alcohol spectrum disorders (9 with fetal alcohol syndrome, 15 heavy exposed nonsyndromal children) and 16 age-matched controls were studied to assess the effect of alcohol consumption during pregnancy on cortical morphology. An automated method was applied to 3D T1-weighted images to assess cortical gyrification using global and regional sulcal indices and two region-based morphological measurements, mean sulcal depth and fold opening. Increasing levels of alcohol exposure were related to reduced cortical folding complexity, even among children with normal brain size, indicating a reduction of buried cortical surface. Fold opening was the strongest anatomical correlate of prenatal alcohol intake, indicating a widening of sulci in all regions that were examined. These data identify cortical morphology as a suitable marker for further investigation of brain damage associated with prenatal alcohol exposure.

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78. ETHANOL EXPOSURE DISRUPTS EXTRAEMBRYONIC MICROTUBULE CYTOSKELETON AND EMBRYONIC BLASTOMERE CELL ADHESION, PRODUCING EPIBOLY AND GASTRULATION DEFECTS
Sarmah S, Muralidharan P, Curtis CL, McClintick JN, Buente BB, Holdgrafer DJ, Ogbeifun O, Olorungbounmi OC, Patino L, Lucas R, Department of Biology, Indiana University-Purdue University Indianapolis, 723 West Michigan Street, Indianapolis, IN 46202-5130, USA.

ABSTRACT
Fetal alcohol spectrum disorder (FASD) occurs when pregnant mothers consume alcohol, causing embryonic ethanol exposure and characteristic birth defects that include craniofacial, neural and cardiac defects. Gastrulation is a particularly sensitive developmental stage for teratogen exposure, and zebrafish is an outstanding model to study gastrulation and FASD.
Epiboly (spreading blastomere cells over the yolk cell), prechordal plate migration and convergence/extension cell movements are sensitive to early ethanol exposure. Here, experiments are presented that characterize mechanisms of ethanol toxicity on epiboly and gastrulation. Epiboly mechanisms include blastomere radial intercalation cell movements and yolk cell microtubule cytoskeleton pulling the embryo to the vegetal pole. Both of these processes were disrupted by ethanol exposure. Ethanol effects on cell migration also indicated that cell adhesion was affected, which was confirmed by cell aggregation assays. E-cadherin cell adhesion molecule expression was not affected by ethanol exposure, but E-cadherin distribution, which controls epiboly and gastrulation, was changed. E-cadherin was redistributed into cytoplasmic aggregates in blastomeres and dramatically redistributed in the extraembryonic yolk cell. Gene expression microarray analysis was used to identify potential causative factors for early development defects, and expression of the cell adhesion molecule protocadherin-18a (pcdh18a), which controls epiboly, was significantly reduced in ethanol exposed embryos. Injecting pcdh18a synthetic mRNA in ethanol treated embryos partially rescued epiboly cell movements, including enveloping layer cell shape changes. Together, data show that epiboly and gastrulation defects induced by ethanol are multifactorial, and include yolk cell (extraembryonic tissue) microtubule cytoskeleton disruption and blastomere adhesion defects, in part caused by reduced pcdh18a expression.

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79. EYEBLINK CONDITIONING: A NON-INVASIVE BIOMARKER FOR NEURODEVELOPMENTAL DISORDERS
Reeb-Sutherland BC, Fox NA.
Department of Psychology, DM 256, Florida International University, 11200 SW 8th Street, Miami, FL, 33199, USA, besuther@fiu.edu.

ABSTRACT
Eyeblink conditioning (EBC) is a classical conditioning paradigm typically used to study the underlying neural processes of learning and memory. EBC has a well-defined neural circuitry, is non-invasive, and can be employed in human infants shortly after birth making it an ideal tool to use in both developing and special populations. In addition, abnormalities in the cerebellum, a region of the brain highly involved in EBC, have been implicated in a number of neurodevelopmental disorders including autism spectrum disorders (ASDs). In the current paper, we review studies that have employed EBC as a biomarker for several neurodevelopmental disorders including fetal alcohol syndrome, Down syndrome, fragile X syndrome, attention deficit/hyperactivity disorder, dyslexia, specific language impairment, and schizophrenia. In addition, we discuss the benefits of using such a tool in individuals with ASD.

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80. ALCOHOL CONSUMPTION IN PREGNANCY: RESULTS FROM THE GENERAL PRACTICE SETTING
Ni Shúilleabháin A, Barry J, Kelly A, O'Kelly F, Darker C, O'Dowd T.
Abstract

BACKGROUND: There is no established safe level of alcohol consumption in pregnancy. Studies from Ireland have consistently shown lower abstention and higher binge drinking rates in pregnancy than other countries, indicating a high potential for foetal alcohol-related disorders. There has been little research on alcohol in pregnancy in primary care.

AIMS: To determine the prevalence of alcohol consumption amongst pregnant women attending their GP for antenatal care, and to compare this to use in the year prior to conception.

METHODS: Prospective cross-sectional study was carried out in fifteen teaching practices in the greater Dublin area. Women were recruited at their antenatal visits. Data were gathered by self-completed questionnaire in the practice, or researcher-administered telephone questionnaire. The questionnaire was based on the AUDIT, a WHO-validated data collection instrument designed for use in primary care.

RESULTS: Two hundred and forty valid questionnaires were returned (80 % recruitment rate). Alcohol intake and binge drinking levels were much lower during pregnancy compared to the year prior to pregnancy (p < 0.001). There was a marked reduction in the prevalence of alcohol use in pregnancy compared to previous research. Over 97 % drink no more than once a week, including almost two-thirds of women who abstain totally from alcohol in pregnancy. Non-pregnant Irish women drink alcohol more frequently, and with higher rates of binge drinking, than women of other nationalities.

CONCLUSIONS: Primary care is a suitable setting to research alcohol use in pregnancy. Alcohol use in pregnancy in Ireland has decreased markedly compared to previous research from this jurisdiction.


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81. DETERMINANTS OF NOVEL OBJECT AND LOCATION RECOGNITION DURING DEVELOPMENT

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ABSTRACT

In the novel object recognition (OR) paradigm, rats are placed in an arena where they encounter two sample objects during a familiarization phase. A few minutes later, they are returned to the same arena and are presented with a familiar object and a novel object. The object location recognition (OL) variant involves the same familiarization procedure but during testing one of the familiar objects is placed in a novel location. Normal adult rats are able to perform both the OR and OL tasks, as indicated by enhanced exploration of the novel vs. the familiar test item. Rats with hippocampal lesions perform the OR but not OL task indicating a role of spatial memory in OL [1]. Recently, these tasks have been used to study the ontogeny of spatial memory but the literature has yielded conflicting results [2,3]. The current experiments add to this literature by: (1) behaviorally characterizing these paradigms in postnatal day (PD) 21, 26 and 31-day-old rats; (2) examining the role of NMDA systems in OR vs. OL; and (3) investigating the effects of neonatal alcohol exposure on both tasks. Results
indicate that normal-developing rats are able to perform OR and OL by PD21, with greater novelty exploration in the OR task at each age. Second, memory acquisition in the OR but not OL task requires NMDA receptor function in juvenile rats. Lastly, neonatal alcohol exposure does not disrupt performance in either task. Implications for the ontogeny of incidental spatial learning and its disruption by developmental alcohol exposure are discussed.


82. LOSS OF MOTONEURONS IN THE VENTRAL COMPARTMENT OF THE RAT HYPOGLOSSAL NUCLEUS FOLLOWING EARLY POSTNATAL EXPOSURE TO ALCOHOL
Stettner GM, Kubin L, Volgin DV.
Department of Animal Biology, School of Veterinary Medicine, University of Pennsylvania, Philadelphia, PA, USA. Electronic address: georg.stettner@med.uni-goettingen.de.

ABSTRACT
Perinatal alcohol exposure (AE) has multiple detrimental effects on cognitive and various behavioral outcomes, but little is known about its impact on the autonomic functions. In a rat model of fetal alcohol spectrum disorders (FASD), we investigated neurochemical and neuroanatomical alterations in two brainstem nuclei, the hypoglossal nucleus (XIIIn) and the dorsal nucleus of the vagus nerve (Xdn). One group of male Sprague-Dawley rats (n=6) received 2.625 g/kg ethanol intragastrically twice daily on postnatal days (PD) 4-9, a period equivalent to the third trimester of human pregnancy, and another group (n=6) was sham-intubated. On PD 18-19, the rats were perfused and medullary sections were immunohistochemically processed for choline acetyltransferase (ChAT) or two aminergic receptors that mediate excitatory drive to motoneurons, α₁-adrenergic (α₁-R) and serotonin 2A (5-HT(2A)-R), and c-Fos. Based on ChAT labeling, AE rats had reduced numbers of motoneurons in the ventral XIIIn (XIIIn-v; 35.4±1.3 motoneurons per side and section vs. 40.0±1.2, p=0.022), but not in the dorsal XIIIn or Xdn. Consistent with ChAT data, both the numbers of α₁-R-labeled motoneurons in the XIIIn-v and the area of the XIIIn-v measured using 5-HT(2A)-R staining were significantly smaller in AE rats (19.7±1.5 vs. 25.0±1.4, p=0.031 and 0.063 mm² ±0.002 vs. 0.074±0.002, p=0.002, respectively). Concurrently, both 5-HT(2A)-R and c-Fos staining tended to be higher in AE rats, suggesting an increased activation. Thus, postnatal AE causes motoneuronal loss in the XIIIn-v. This may compromise upper airway control and contribute to increased risk of upper airway obstructions and sudden infant death in FASD victims.


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83. MATERNAL ALCOHOL CONSUMPTION PRODUCING FETAL ALCOHOL SPECTRUM DISORDERS (FASD): QUANTITY, FREQUENCY, AND TIMING OF DRINKING
Philip A, May a, b, Jason Blankenship b, Anna-Susan Marais b, J. Phillip Gossage b, Wendy O. Kalberg b, Belinda Joubert f, Marise Cloete f, Ronel Barnard f, Marlene De Vries f, Julie Haskena, Luther K. Robinson d, Colleen M. Adnams a, David Buckley b, Melanie Manning f, Charles D.H. Parry c, d, H. Eugene Hoyme h, Barbara Tabachnick k, Soraya Seedat c
ABSTRACT

Background: Concise, accurate measures of maternal prenatal alcohol use are needed to better understand fetal alcohol spectrum disorders (FASD).

Methods: Measures of drinking by mothers of children with specific FASD diagnoses and mothers of randomly-selected controls are compared and also correlated with physical and cognitive/behavioral outcomes.

Results: Measures of maternal alcohol use can differentiate maternal drinking associated with FASD from that of controls and some from mothers of alcohol-exposed normals. Six variables that combine quantity and frequency concepts distinguish mothers of FASD children from normal controls. Alcohol use variables, when applied to each trimester and three months prior to pregnancy, provide insight on critical timing of exposure as well. Measures of drinking, especially bingeing, correlate significantly with increased child dysmorphology and negative cognitive/behavioral outcomes in children, especially low non-verbal IQ, poor attention, and behavioral problems. Logistic regression links (p < .001) first trimester drinking (vs. no drinking) with FASD, elevating FASD likelihood 12 times; first and second trimester drinking increases FASD outcomes 61 times; and drinking in all trimesters 65 times. Conversely, a similar regression (p = .008) indicates that drinking only in the first trimester makes the birth of a child with an FASD 5 times less likely than drinking in all trimesters.

Conclusions: There is significant variation in alcohol consumption both within and between diagnostic groupings of mothers bearing children diagnosed within the FASD continuum. Drinking measures are empirically identified and correlated with specific child outcomes. Alcohol use, especially heavy use, should be avoided throughout pregnancy.


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ABSTRACT

**Study question**: How variable is the length of human pregnancy, and are early hormonal events related to gestational length?

**Summary answer**: Among natural conceptions where the date of conception (ovulation) is known, the variation in pregnancy length spanned 37 days, even after excluding women with complications or preterm births.

**What is known already**: Previous studies of length of gestation have either estimated gestational age by last menstrual period (LMP) or ultrasound (both imperfect measures) or included pregnancies conceived through assisted reproductive technology.

**Study design, size, duration**: The Early Pregnancy Study was a prospective cohort study (1982-85) that followed 130 singleton pregnancies from unassisted conception to birth, with detailed hormonal measurements through the conception cycle; 125 of these pregnancies were included in this analysis.

**Participants/materials, setting, methods**: We calculated the length of gestation beginning at conception (ovulation) in 125 naturally conceived, singleton live births. Ovulation, implantation and corpus luteum (CL) rescue pattern were identified with urinary hormone measurements. We accounted for events that artificially shorten the natural length of gestation (Cesarean delivery or labor induction, i.e. 'censoring') using Kaplan-Meier curves and proportional hazards models. We examined hormonal and other factors in relation to length of gestation. We did not have ultrasound information to compare with our gold standard measure.

**Main results and the role of chance**: The median time from ovulation to birth was 268 days (38 weeks, 2 days). Even after excluding six preterm births, the gestational length range was 37 days. The coefficient of variation was higher when measured by LMP (4.9%) than by ovulation (3.7%), reflecting the variability of time of ovulation. Conceptions that took longer to implant also took longer from implantation to delivery ($P = 0.02$). CL rescue pattern (reflecting ovarian response to implantation) was predictive ($P = 0.006$); pregnancies with a rapid progesterone rise were longer than those with delayed rise (a 12-day difference in the median gestational length). Mothers with longer gestations were older ($P = 0.02$), had longer pregnancies in other births ($P < 0.0001$) and were heavier at birth ($P = 0.01$). We did not see an association between the length of gestation and several factors that have been associated with gestational length in previous studies: body mass index, alcohol intake, parity or offspring sex.

**Limitations, reasons for caution**: The sample size was small and some exposures were rare, reducing power to detect weak associations.

**Wider implications of the findings**: Human gestational length varies considerably even when measured exactly (from ovulation). An individual woman's deliveries tend to occur at similar gestational ages. Events in the first 2 weeks after conception are predictive of subsequent pregnancy length, and may suggest pathways underlying the timing of delivery.

**Study funding/competing interest**: This research was supported by the Intramural Research Program of the NIH, National Institute of Environmental Health Sciences. None of the authors has any conflict of interest to declare.


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85. **AN EXAMINATION OF THE SOCIAL DETERMINANTS OF HEALTH AS FACTORS RELATED TO HEALTH, HEALING AND PREVENTION OF FOETAL ALCOHOL SPECTRUM DISORDER IN A NORTHERN CONTEXT--THE BRIGHTENING OUR HOME FIRES PROJECT, NORTHWEST TERRITORIES, CANADA**

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**ABSTRACT**

**Objective:** The Brightening Our Home Fires (BOHF) project was conceptualized as an exploratory project to examine the issue of the prevention of foetal alcohol spectrum disorder (FASD) from a women's health perspective in the Northwest Territories (NT). While dominant discourse suggests that FASD is preventable by abstention from alcohol during pregnancy, a broader perspective would indicate that alcohol and pregnancy is a far more complex issue, that is, bound in location, economics, social and cultural views of health. This project was prevention focused and a social determinant of health (SDH) perspective informed this research.

**Methods:** The BOHF project was a qualitative research project using a participatory action research framework to examine women's health and healing in the north. The methodology utilized was Photovoice. Women were provided training in digital photography and given cameras to use and keep. The primary research question utilized was: What does health and healing look like for you in your community? Women described their photos, individually or in groups around this central topic. This research was FASD informed, and women participants were aware this was an FASD prevention funded project whose approach focused on a broader context of health and lived experience.

**Results:** This project drew 30 participants from: Yellowknife, Lutsel 'ke, Behchokö and Ulukhaktok. These four different communities across the NT represented Dene and Inuit culture. The qualitative data analysis offered themes of importance to women's health in the north including: land and tradition; housing; poverty; food; family; health, mental health and trauma, and travel. Photovoice provides a non-threatening way to engage in dialogue on complex health and social issues.

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86. **PRENATAL ALCOHOL EXPOSURE AMONG ALASKA NATIVE/AMERICAN INDIAN INFANTS**

Khan BA, Robinson RF, Smith JJ, Dillard DA.
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**ABSTRACT**

**Background:** Recent reports indicate a decline in rates of Fetal Alcohol Syndrome (FAS) among Alaska Native and American Indian (AN/AI) infants. Nevertheless, AN/AI infants remain disproportionately impacted by the effects of prenatal alcohol exposure.

**Methods:** AN/AI pregnant women in their 3rd trimester completed a questionnaire on demographic data and the amount and frequency of their alcohol consumption in the month prior to conception and during pregnancy. Differences across demographics and trimesters were tested with the Chi-square, Fisher's exact or McNemar's test as appropriate.

www.nofas-uk.org
Results: Of the 125 participants, 56% (n = 71) reported no alcohol consumption in the 1st through 3rd trimesters of pregnancy; 30% (n = 38) of the 125 participants also reported no alcohol consumption in the month before pregnancy. Of the 43% (n = 54) who reported consuming alcohol during pregnancy (1st, 2nd and/or 3rd trimester), most (35%) reported alcohol use only in the 1st trimester. Binge drinking in the 1st or 2nd trimester was reported amongst 20% (n = 25) of participants with an additional 18% (n = 29) reporting binge drinking in the month prior to pregnancy. Women who reported pre-conception binge drinking were significantly more likely to report binge drinking during their 1st trimester (p < 0.0001) and 2nd trimester (p < 0.0001). A history of tobacco use (p = 0.0403) and cigarette smoking during pregnancy (p < 0.0001) were also associated with binge drinking during pregnancy.

Conclusion: Among study participants, reported use of alcohol was primarily limited to pre-conception and the 1st trimester, with a dramatic decrease in the 2nd and 3rd trimesters. Prevention programmes, such as the Alaska FAS Prevention Project, may have contributed to observed decreases in the 2nd and 3rd trimesters. Additional study and focus on pre-conception, the 1st trimester and binge drinking, as well as tobacco use might augment Fetal Alcohol Spectrum Disorder prevention efforts.


87. PRENATAL ETHANOL (ETOH) EXPOSURE ALTERS THE SENSITIVITY OF THE ADULT DENTATE GYRUS TO ACUTE ETOH EXPOSURE
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ABSTRACT
Background: Prenatal ethanol (EtOH) exposure results in a spectrum of structural, cognitive, and behavioral abnormalities, collectively termed "fetal alcohol spectrum disorders" (FASDs). The hippocampal formation, an area of the brain strongly linked with learning and memory, is particularly vulnerable to the teratogenic effects of EtOH. Prenatal EtOH exposure can lead to long-lasting impairments in the ability to process spatial information, as well as produce long-lasting deficits in the ability of animals to exhibit long-term potentiation (LTP), a biological model of learning and memory processing. These deficits also have the ability to facilitate EtOH and/or other drug abuse later in life. This study sought to determine prenatal EtOH exposure altered the effects of acute EtOH application on synaptic plasticity.

Methods: Prenatal EtOH exposure was modeled using a liquid diet where dams were given 1 of 3 diets: (i) a liquid diet containing EtOH (35.5% EtOH-derived calories), (ii) a liquid diet, isocaloric to the EtOH diet, but with maltose-dextrin substituting for the EtOH-derived calories, and (iii) an ad libitum diet of standard rat chow. Extracellular recordings from transverse brain slices (350 μm) prepared from 50- to 70-day-old rats, following prenatal EtOH exposure (gestational day 1 to 21). LTP was examined in the dentate gyrus following acute EtOH exposure (0, 20, or 50 mM) in these slices.

Results: Prenatal EtOH exposure attenuated LTP in the adult dentate gyrus. In control offspring, acute application of EtOH in adulthood attenuated (20 mM) or blocked (50 mM) LTP. Conversely, the effect of acute EtOH application on LTP was not as pronounced in prenatal EtOH offspring.

Conclusions: Prenatal EtOH exposure alters the sensitivity of the adult dentate gyrus to acute EtOH application producing a long-lasting tolerance to the inhibitory effects of EtOH.
This decreased sensitivity may provide a mechanism promoting the formation of drug-associated memories and help explain the increased likelihood of developing an alcohol dependency often observed in individuals with FASDs.


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Wiley Online Library - Alcoholism: Clinical and Experimental Research
Early View (Online Version of Record published before inclusion in an issue)
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88. SENSORY-MOTOR DEFICITS IN CHILDREN WITH FETAL ALCOHOL SPECTRUM DISORDER ASSESSED USING A ROBOTIC VIRTUAL REALITY PLATFORM

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ABSTRACT

Background:
Fetal alcohol spectrum disorder (FASD) is associated with a large number of cognitive and sensory-motor deficits. In particular, the accurate assessment of sensory-motor deficits in children with FASD is not always simple and relies on clinical assessment tools that may be coarse and subjective. Here we present a new approach: using robotic technology to accurately and objectively assess motor deficits of children with FASD in a center-out reaching task.

Methods:
A total of 152 typically developing children and 31 children with FASD, all aged between 5 and 18 were assessed using a robotic exoskeleton device coupled with a virtual reality projection system. Children made reaching movements to 8 peripheral targets in a random order. Reach trajectories were subsequently analyzed to extract 12 parameters that had been previously determined to be good descriptors of a reaching movement, and these parameters were compared for each child with FASD to a normative model derived from the performance of the typically developing population.

Results:
Compared with typically developing children, the children with FASD were found to be significantly impaired on most of the parameters measured, with the greatest deficits found in initial movement direction error. Also, children with FASD tended to fail more parameters than typically developing children: 95% of typically developing children failed fewer than 3 parameters compared with 69% of children with FASD. These results were particularly pronounced for younger children.

Conclusions:
The current study has shown that robotic technology is a sensitive and powerful tool that provides increased specificity regarding the type of motor problems exhibited by children with FASD. The high frequency of motor deficits in children with FASD suggests that interventions aimed at stimulating and/or improving motor development should routinely be considered for
this population.


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89. IMPACT OF COMBINED PRENATAL ETHANOL AND PRENATAL STRESS EXPOSURE ON ANXIETY AND HIPPOCAMPAL-SENSITIVE LEARNING IN ADULT OFFSPRING
Staples MC, Rosenberg MJ, Allen NA, Porch MW, Savage DD. Department of Neurosciences, University of New Mexico Health Sciences Center, Albuquerque, New Mexico.

ABSTRACT

Background: Prenatal ethanol (EtOH) and prenatal stress have both been independently shown to induce learning deficits and anxiety behavior in adult offspring. However, the interactive effects of these 2 developmental teratogens on behavioral outcomes have not been systematically evaluated.

Methods: We combined an established moderate prenatal EtOH consumption paradigm where Long-Evans rat dams voluntarily consume either a 0 or 5% EtOH solution in 0.066% saccharin water (resulting in a mean peak maternal serum EtOH concentration of 84 mg/dl) with a novel prenatal stress paradigm. Pregnant rats were exposed to 3% 2,3,5-trimethyl-3-thiazoline (TMT) for 20 minutes a day on gestational days 13, 15, 17, and 19. Adult female offspring were evaluated for anxiety-like behavior using an elevated plus-maze and hippocampal-sensitive learning using a 2-trial trace conditioning (TTTC) task.

Results: TMT exposure produced a threefold increase in maternal serum corticosterone compared to nonexposed, unhandled controls. Neither prenatal exposure paradigm, either alone or in combination, altered maternal weight gain, EtOH consumption, maternal care of litters, litter size, pup birth weight, or pup weight gain up to weaning. Offspring exposed to prenatal stress displayed significant increases in anxiety-like behavior in the elevated plus-maze in terms of open arm entries and time spent on the open arms, with no significant effect of prenatal EtOH exposure and no interaction of the 2 prenatal exposures. Performance in a TTTC task revealed a significant effect of prenatal EtOH exposure on freezing behavior on the testing day, with no significant effect of prenatal stress exposure and no interaction of the 2 prenatal exposures.

Conclusions: While each prenatal exposure independently produced different behavioral outcomes, the results indicate that there is no significant interaction of prenatal EtOH and prenatal stress exposures on learning or anxiety at the exposure levels employed in this dual exposure paradigm. Subsequent studies will examine whether similar outcomes occur in male offspring and whether other measures of anxiety or learning are differentially impacted by these prenatal exposure paradigms.


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90. PRENATAL ALCOHOL EXPOSURE RESULTS IN LONG-TERM SEROTONIN NEURON DEFICITS IN FEMALE RATS: MODULATORY ROLE OF OVARIAN STEROIDS
Sliwowska JH, Song HJ, Bodnar T, Weinberg J.
Department of Cellular and Physiological Sciences, University of British Columbia, Vancouver, British Columbia, Canada; Laboratory of Neurobiology, Institute of Zoology, Poznan University of Life Sciences, Poznan, Poland.

ABSTRACT
Background: Previous studies on male rodents found that prenatal alcohol exposure (PAE) decreases the number of serotonin immunoreactive (5-HT-ir) neurons in the brainstem. However, data on the effects of PAE in females are lacking. In light of known sex differences in responsiveness of the 5-HT system and known effects of estrogen (E2) and progesterone (P4) in the brain, we hypothesized that sex steroids will modulate the adverse effects of PAE on 5-HT neurons in adult females.

Methods: Adult females from 3 prenatal groups (Prenatal alcohol-exposed [PAE], Pair-fed [PF], and ad libitum-fed Controls [C]) were ovariectomized (OVX), with or without hormone replacement, or underwent Sham OVX. 5-HT-ir cells were examined in key brainstem areas.

Results: Our data support the hypothesis that PAE has long-term effects on the 5-HT system of females and that ovarian steroids have a modulatory role in these effects. Intact (Sham OVX) PAE females had marginally lower numbers of 5-HT-ir neurons in the dorsal raphe nucleus of the brainstem compared with PF and C females. This marginal difference became significant following removal of hormones by OVX. Replacement with E2 restored the number of 5-HT-ir neurons in PAE females to control levels, while P4 reversed the effects of E2. Importantly, despite these differential responses of the 5-HT system to ovarian steroids, there were no differences in E2 and P4 levels among prenatal treatment groups.

Conclusions: These data demonstrate long-term, adverse effects of PAE on the 5-HT system of females, as well as differential sensitivity of PAE compared with control females to the modulatory effects of ovarian steroids on 5-HT neurons. Our findings have important implications for understanding sex differences in 5-HT dysfunction in depression/anxiety disorders and the higher rates of these mental health problems in individuals with fetal alcohol spectrum disorder.


91. DIFFUSION MRI OF THE DEVELOPING CEREBRAL CORTICAL GRAY MATTER CAN BE USED TO DETECT ABNORMALITIES IN TISSUE MICROSTRUCTURE ASSOCIATED WITH FETAL ETHANOL EXPOSURE
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Department of Behavioral Neuroscience, Oregon Health & Science University, Portland OR, USA; Advanced Imaging Research Center, Oregon Health & Science University, Portland OR, USA.

Abstract
Fetal alcohol spectrum disorders (FASDs) comprise a wide range of neurological deficits that result from fetal exposure to ethanol (EtOH), and are the leading cause of environmentally related birth defects and mental retardation in the western world. One aspect of diagnostic and therapeutic intervention strategies that could substantially improve our ability to combat
this significant problem would be to facilitate earlier detection of the disorders within individuals. Light microscopy-based investigations performed by several laboratories have previously shown that morphological development of neurons within the early-developing cerebral cortex is abnormal within the brains of animals exposed to EtOH during fetal development. We and others have recently demonstrated that diffusion MRI can be of utility for detecting abnormal cellular morphological development in the developing cerebral cortex. We therefore assessed whether diffusion tensor imaging (DTI) could be used to distinguish the developing cerebral cortices of ex vivo rat pup brains born from dams treated with EtOH (EtOH; 4.5g/kg, 25%) or calorie-matched quantities of maltose/dextrin (M/D) throughout gestation. Water diffusion and tissue microstructure were investigated using DTI (fractional anisotropy, FA) and histology (anisotropy index, AI), respectively. Both FA and AI decreased with age, and were higher in the EtOH than the M/D group at postnatal ages (P)0, P3, and P6. Additionally, there was a significant correlation between FA and AI measurements. These findings provide evidence that disruptions in cerebral cortical development induced by EtOH exposure can be revealed by water diffusion anisotropy patterns, and that these disruptions are directly related to cerebral cortical differentiation.


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92. RISK OF SPINA BIFIDA AND MATERNAL CIGARETTE, ALCOHOL, AND COFFEE USE DURING THE FIRST MONTH OF PREGNANCY
Corey M. Benedume, Mahsa M. Yazdy, Allen A. Mitchelle and Martha M. Werler
Slone Epidemiology Center at Boston University, 1010 Commonwealth Ave. Boston, MA 02215, USA

ABSTRACT
This study was conducted to assess the association between the risks of spina bifida (SB) in relation to cigarette, alcohol, and caffeine consumption by women during the first month of pregnancy. Between 1988–2012, this multi-center case-control study interviewed mothers of 776 SB cases and 8,756 controls about pregnancy events and exposures. We evaluated cigarette smoking, frequency of alcohol drinking, and caffeine intake during the first lunar month of pregnancy in relation to SB risk. Logistic regression models were used to calculate adjusted odds ratios and 95% confidence intervals. Levels of cigarette smoking (1–9 and ≥10/day), alcohol intake (average ≥4 drinks/day) and caffeine intake (<1, 1, and ≥2 cups/day) were not likely to be associated with increased risk of SB. Further, results were similar among women who ingested less than the recommended amount of folic acid (400 μg/day).

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93. REDUCED EXPRESSION OF BRAIN CANNABINOIDS RECEPTOR 1 (CNR1) IS COUPLED WITH AN INCREASED COMPLEMENTARY MICRO-RNA (MIR-26B) IN A MOUSE MODEL OF FETAL ALCOHOL SPECTRUM DISORDERS
Stringer RL, Laufer BI, Kleiber ML, Singh SM.
Department of Biology, Molecular Genetics Unit, Western University, London, ON N6A 5B7, Canada. ssingh@uwo.ca.

ABSTRACT
Background: Prenatal alcohol exposure is known to result in fetal alcohol spectrum disorders, a continuum of physiological, behavioural, and cognitive phenotypes that include increased risk for anxiety and learning-associated disorders. Prenatal alcohol exposure results in life-long disorders that may manifest in part through the induction of long-term gene expression changes, potentially maintained through epigenetic mechanisms.

Findings: Here we report a decrease in the expression of Canabinoid receptor 1 (Cnr1) and an increase in the expression of the regulatory microRNA miR-26b in the brains of adult mice exposed to ethanol during neurodevelopment. Furthermore, we show that miR-26b has significant complementarity to the 3'-UTR of the Cnr1 transcript, giving it the potential to bind and reduce the level of Cnr1 expression.

Conclusions: These findings elucidate a mechanism through which some genes show long-term altered expression following prenatal alcohol exposure, leading to persistent alterations to cognitive function and behavioural phenotypes observed in fetal alcohol spectrum disorders.


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94. THE ASSOCIATION OF MILD, MODERATE, AND BINGE PRENATAL ALCOHOL EXPOSURE AND CHILD NEUROPSYCHOLOGICAL OUTCOMES: A META-ANALYSIS
Flak AL, Su S, Bertrand J, Denny CH, Kesmodel US, Cogswell ME.
Department of Epidemiology, Rollins School of Public Health, Emory University, Atlanta, Georgia; Oak Ridge Institute for Science and Education, Oak Ridge, Tennessee; National Center on Birth Defects and Developmental Disabilities, Centers for Disease Control and Prevention, Atlanta, Georgia.

ABSTRACT
Background: The objective of this review is to evaluate the literature on the association between mild, moderate, and binge prenatal alcohol exposure and child neurodevelopment.

Methods: Meta-analysis with systematic searches of MEDLINE (1970 through August 2012), EMBASE (1988 through August 2012), and PsyclINFO® (1970 through August 2012) and examination of selected references.

Results: From 1,593 articles, we identified 34 presenting data from cohort studies that met our inclusion criteria. Information on study population, outcomes, measurement instruments, timing and quantification of alcohol exposure, covariates, and results was abstracted. Outcomes included academic performance, attention, behavior, cognition, language skills, memory, and visual and motor development. The quality of each article was assessed by 2 researchers using the Newcastle-Ottawa Scale. Based on 8 studies of 10,000 children aged 6 months through 14 years, we observed a significant detrimental association between any
binge prenatal alcohol exposure and child cognition (Cohen's d [a standardized mean difference score] -0.13; 95% confidence interval [CI], -0.21, -0.05). Based on 3 high-quality studies of 11,900 children aged 9 months to 5 years, we observed a statistically significant detrimental association between moderate prenatal alcohol exposure and child behavior (Cohen's d -0.15; 95% CI, -0.28, -0.03). We observed a significant, albeit small, positive association between mild-to-moderate prenatal alcohol exposure and child cognition (Cohen's d 0.04; 95% CI, 0.00, 0.08), but the association was not significant after post hoc exclusion of 1 large study that assessed mild consumption nor was it significant when including only studies that assessed moderate alcohol consumption. None of the other completed meta-analyses resulted in statistically significant associations between mild, moderate, or binge prenatal alcohol exposure and child neuropsychological outcomes.

Conclusions: Our findings support previous findings suggesting the detrimental effects of prenatal binge drinking on child cognition. Prenatal alcohol exposure at levels less than daily drinking might be detrimentally associated with child behavior. The results of this review highlight the importance of abstaining from binge drinking during pregnancy and provide evidence that there is no known safe amount of alcohol to consume while pregnant.


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Development, dev.biologists.org

95. PDGFRA PROTECTS AGAINST ETHANOL-INDUCED CRANIOFACIAL DEFECTS IN A ZEBRAFISH MODEL OF FASD
Neil McCarthy1, Leah Wetherill2, C. Ben Lovely1, Mary E. Swartz1, Tatiana M. Foroud2 and Johann K. Eberhart1,*

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ABSTRACT

Human birth defects are highly variable and this phenotypic variability can be influenced by both the environment and genetics. However, the synergistic interactions between these two variables are not well understood. Fetal alcohol spectrum disorders (FASD) is the umbrella term used to describe the wide range of deleterious outcomes following prenatal alcohol exposure. Although FASD are caused by prenatal ethanol exposure, FASD are thought to be genetically modulated, although the genes regulating sensitivity to ethanol teratogenesis are largely unknown. To identify potential ethanol-sensitive genes, we tested five known craniofacial mutants for ethanol sensitivity: cyp26b1, gata3, pdgfra, smad5 and smoothened. We found that only platelet-derived growth factor receptor alpha (pdgfra) interacted with ethanol during zebrafish craniofacial development. Analysis of the PDGF family in a human FASD genome-wide dataset links PDGFRA to craniofacial phenotypes in FASD, prompting a mechanistic understanding of this interaction. In zebrafish, untreated pdgfra mutants have cleft palate due to defective neural crest cell migration, whereas pdgfra heterozygotes develop normally.

Ethanol-exposed pdgfra mutants have profound craniofacial defects that include the loss of the palatal skeleton and hypoplasia of the pharyngeal skeleton. Furthermore, ethanol treatment revealed latent haploinsufficiency, causing palatal defects in ~62% of pdgfra
heterozygotes. Neural crest apoptosis partially underlies these ethanol-induced defects in pdgfra mutants, demonstrating a protective role for Pdgfra. This protective role is mediated by the PI3K/mTOR pathway.

Collectively, our results suggest a model where combined genetic and environmental inhibition of PI3K/mTOR signaling leads to variability within FASD.

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96. LOW-TO-MODERATE ALCOHOL CONSUMPTION DURING PREGNANCY AND CHILD DEVELOPMENT--MOVING BEYOND OBSERVATIONAL STUDIES
Gray R.
National Perinatal Epidemiology Unit, University of Oxford, Oxford, UK. ron.gray@npeu.ox.ac.uk

COMMENT ON
The effects of low to moderate alcohol consumption and binge drinking in early pregnancy on behaviour in 5-year-old children: a prospective cohort study on 1628 children. [BJOG. 2013]


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97. EFFECT OF PRENATAL ALCOHOL EXPOSURE ON BONY CRANIOFACIAL DEVELOPMENT: A MOUSE MICROCT STUDY
Department of Radiology and Imaging Sciences, IU School of Medicine, Indianapolis, IN 46202, USA. shenli@iu.edu

ABSTRACT
Craniofacial bone dysmorphology is an important but under-explored potential diagnostic feature of fetal alcohol spectrum disorders. This study used longitudinal MicroCT 3D imaging to examine the effect of prenatal alcohol exposure on craniofacial bone growth in a mouse model. C57BL/6J dams were divided into 3 groups: alcohol 4.2% v/v in PMI® liquid diet (ALC), 2 weeks prior to and during pregnancy from embryonic (E) days 7-E16; pair-fed controls (PF), isocalorically matched to the ALC group; chow controls (CHOW), given ad libitum chow and water. The MicroCT scans were performed on pups on postnatal days 7 (P7) and P21. The volumes of the neurocranium (volume encased by the frontal, parietal, and occipital bones) and the viscerocranium (volume encased by the mandible and nasal bone), along with total skull bone volume, head size, and head circumference were evaluated using general linear models and discriminant analyses. The pups in the alcohol-treated group, when compared to the chow-fed controls (ALC vs CHOW) and the isocaloric-fed controls (ALC vs PF), showed differences in head size and circumference at P7 and P21, the total skull volume and parietal bone volume at P7, and volume of all the tested bones except nasal at P21. There was a growth trend of ALC < CHOW and ALC < PF. While covarying for gender and
head size or circumference, the treatment affected the total skull and mandible at P7 (ALC > CHOW), and the total skull, parietal bone, and occipital bone at P21 (ALC < CHOW, ALC < PF). While covarying for the P7 measures, the treatment affected only the 3 neurocranial bones at P21 (ALC < CHOW, ALC < PF). Discriminant analysis sensitively selected between ALC and CHOW (AUC = 0.967), between ALC and PF (AUC = 0.995), and between PF and CHOW (AUC = 0.805). These results supported our hypothesis that craniofacial bones might be a reliable and sensitive indicator for the diagnosis of prenatal alcohol exposure. Significantly, we found that the neurocranium (upper skull) was more sensitive to alcohol than the viscerocranium (face).

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98. CANADIAN CHILDREN AND YOUTH IN CARE: THE COST OF FETAL ALCOHOL SPECTRUM DISORDER
Svetlana Popova, Shannon Lange, Larry Burd, Jürgen Rehm

ABSTRACT
Background: A high prevalence of prenatal alcohol exposure has been reported among children in care and thus, the risk of fetal alcohol spectrum disorder (FASD) in this population is high.

Objective: The purpose of the current study was to estimate the number of children (0–18 years) in care with FASD and to determine the associated cost by age group, gender, and province/territory in Canada in 2011.

Methods: The prevalence of children in care by province/territory was obtained from the Canadian Child Welfare Research Portal, and the number of children in care with FASD for each province/territory was estimated from available epidemiological studies. In order to calculate the total cost per province/territory, the cost per individual per day, by age group, was applied to the respective number of children in care with FASD.

Results: The estimated number of children in care with FASD ranged from 2,225 to 7,620, with an annual cost of care ranging from 57.9 to 198.3 million Canadian dollars (CND). The highest overall cost (29.5 to 101.1 million CND) was for 11–15 year-olds.

Conclusion: The study findings can be used to demonstrate the substantial economic burden that FASD places on the child welfare system. Attention towards the needs of this population and prevention efforts to reduce FASD incidence in Canada, and other countries are urgently needed.

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MAGNETIC RESONANCE MICROSCOPY-BASED ANALYSES OF THE NEUROANATOMICAL EFFECTS OF GESTATIONAL DAY 9 ETHANOL EXPOSURE IN MICE

Parnell SE, Holloway HT, O'Leary-Moore SK, Dehart DB, Paniaqua B, Oguz I, Budin F, Styner MA, Johnson GA, Sulik KK.

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ABSTRACT

Animal model-based studies have shown that ethanol exposure during early gestation induces developmental stage-specific abnormalities of the face and brain. The exposure time-dependent variability in ethanol's teratogenic outcomes is expected to contribute significantly to the wide spectrum of effects observed in humans with fetal alcohol spectrum disorder (FASD). The work presented here employs a mouse FASD model and magnetic resonance microscopy (MRM; high resolution magnetic resonance imaging) in studies designed to further our understanding of the developmental stage-specific defects of the brain that are induced by ethanol. At neurulation stages, i.e. at the beginning of gestational day (GD) 9 and again 4 hours later, time-mated C57Bl/6J dams were intraperitoneally administered 2.9 g/kg ethanol or vehicle. Ethanol-exposed fetuses were collected on GD 17, processed for MRM analysis, and results compared to comparably staged controls. Linear and volume measurements as well as shape changes for numerous individual brain regions were determined. GD 9 ethanol exposure resulted in significantly increased septal region width, reduction of cerebellar volume, and enlargement of all of the ventricles. Additionally, the results of shape analyses showed that many areas of the ethanol-exposed brains including the cerebral cortex, hippocampus and right striatum were significantly misshapen. These data demonstrate that ethanol can induce dysmorphology that may not be obvious based on volumetric analyses alone, highlight the asymmetric aspects of ethanol-induced defects, and add to our understanding of ethanol's developmental stage-dependent neuroteratogenesis.

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INVOLVING CONSUMERS AND THE COMMUNITY IN THE DEVELOPMENT OF A DIAGNOSTIC INSTRUMENT FOR FETAL ALCOHOL SPECTRUM DISORDERS IN AUSTRALIA


Telethon Institute for Child Health Research, Centre for Child Health Research, The University of Western Australia, PO Box 855, West Perth, Western Australia 6872, Australia. hjones@ichr.uwa.edu.au

ABSTRACT

Background: Australia's commitment to consumer and community participation in health and medical research has grown over the past decade. Participatory research models of engagement are the most empowering for consumers.
Methods: As part of a project to develop a diagnostic instrument for fetal alcohol spectrum disorders (FASD) in Australia (FASD Project), the Australian FASD Collaboration (Collaboration), including a consumer advocate and two consumer representatives, was established. On completion of the FASD Project an on-line survey of Collaboration members was conducted to assess their views on consumer involvement. Women in the community were also invited to participate in Community Conversations to discuss real life situations regarding communications with health professionals about alcohol and pregnancy. Community Conversation feedback was analysed qualitatively and attendees were surveyed about their views of the Community Conversation process.

Results: The on-line survey was completed by 12 members of the Collaboration (71%). Consumer and community participation was considered important and essential, worked well, and was integral to the success of the project. The 32 women attending the Community Conversations generated 500 statements that made reference to prevention, how information and messages are delivered, and appropriate support for women. Nearly all the attendees at the Community Conversations (93%) believed that they had an opportunity to put forward their ideas and 96% viewed the Community Conversations as a positive experience.

Conclusions: The successful involvement of consumers and the community in the FASD Project can be attributed to active consumer and community participation, which included continued involvement throughout the project, funding of participation activities, and an understanding of the various contributions by the Collaboration members.


101. GLUCOSE METABOLISM DISORDER IS A RISK FACTOR IN ETHANOL EXPOSURE INDUCED MALFORMATION IN EMBRYONIC BRAIN
Tan RR, Li YF, Zhang XT, Huang YH, Wu YP, Ouyang SH, Tsoi B, Yi RN, Yang X, Kurihara H, He RR.
Pharmacy College, Jinan University, Guangzhou 510632, China.

ABSTRACT
Prenatal exposure to ethanol has been reported to cause developmental defects in the brain. During brain development, a sufficient energy source is deemed essential and glucose is regarded as the primary energy source for neurons. In this study, the impact of ethanol on embryonic malformation and cerebral glucose metabolism in developing embryo was investigated. Different doses of ethanol (0, 10, 20, 40 mg/egg) were administrated to chicken embryos after 36 h incubation. Embryonic brain weight was found significantly decreased. Moreover, we observed an obvious reduction of neurofilament expression in the central nervous system (CNS) by immunostaining assay. All the above indicated that ethanol exposure caused obvious CNS damages and resulted malformations in the developing brain. Mechanism research showed that cerebral glucose and lactic acid contents, activities of hexokinase, pyruvate kinase and lactic dehydrogenase were decreased dose dependently. Meanwhile, mRNA levels of glucose transporter 1, glucose transporter 3 and insulin-like growth factor I in the brain demonstrated a significant decrease in gene expression after ethanol exposure. These results suggested that glucose metabolism disorder is an important risk factor in ethanol exposure induced malformation in embryonic brain.

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102. DIMENSIONS OF PARENTAL ALCOHOL USE/PROBLEMS AND OFFSPRING TEMPERAMENT, EXTERNALIZING BEHAVIORS, AND ALCOHOL USE/PROBLEMS
Kendler KS, Gardner CO, Edwards A, Hickman M, Heron J, Macleod J, Lewis G, Dick DM. Virginia Institute of Psychiatric and Behavioral Genetics, Virginia Commonwealth University School of Medicine, Richmond, Virginia; Department of Psychiatry, Virginia Commonwealth University School of Medicine, Richmond, Virginia; Department of Human and Molecular Genetics, Virginia Commonwealth University School of Medicine, Richmond, Virginia.

ABSTRACT
Background: Alcohol consumption (AC) and alcohol problems (AP) are complex traits. How many factors reflecting parental AC and AP are present in the large prospectively followed Avon Longitudinal Study of Parents and Children cohort? Would these factors be uniquely associated with various temperamental and alcohol-related outcomes in the children?

Methods: We factor-analyzed multiple items reflecting maternal and paternal AC and AP measured over a 12-year period from before the birth of the child (n = 14,093 families). We examined, by linear regression controlling for socioeconomic status, the relationship between scales derived from these factors and offspring early childhood temperament, externalizing traits, and adolescent AC and AP (ns ranging from 9,732 to 3,454).

Results: We identified 5 coherent factors: typical maternal AC, maternal AC during pregnancy, maternal AP, paternal AC, and paternal AP. In univariate analyses, maternal and paternal AC and AP were modestly and significantly associated with low shyness, sociability, hyperactivity, and conduct problems in childhood and early adolescence; delinquent behavior at age 15; and AC and AP at ages 15 and 18. AC and AP at age 18 were more strongly predicted by parental factors than at age 15. Maternal AC during pregnancy uniquely predicted externalizing traits at ages 4, 13, and 15.

Conclusions: Parental AC and AP are complex multidimensional traits that differ in their association with a range of relevant measures in their children. Controlling for background AC and AP, self-reported levels of maternal AC during pregnancy uniquely predicted externalizing behaviors in childhood and adolescence.

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103. ALCOHOL EXPOSURE IN UTERO INCREASES SUSCEPTIBILITY TO PROSTATE TUMORIGENESIS IN RAT OFFSPRING
Murugan S, Zhang C, Mootahedzadeh S, Sarkar DK. Endocrine Program, Department of Animal Sciences, Rutgers, The State University of New Jersey, New Brunswick, New Jersey.

ABSTRACT
Background: Prenatal alcohol exposure has been shown to increase offspring susceptibility to some chemical carcinogens. Whether prenatal exposure to alcohol makes the offspring more susceptible to the development of prostate cancer is not known. Therefore, we determined whether any functional abnormalities and increased cancer susceptibility exist in

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the prostate of fetal alcohol-exposed male rats during the adult period.

**Methods:** Pregnant rats were fed with a liquid diet containing alcohol (alcohol-fed [AF]), or pair-fed with isocaloric liquid diet (PF) or ad libitum fed with rat chow (ad lib-fed). Male offspring of these rats were given N-Nitroso-N-methylurea and testosterone to induce prostate neoplasia or left untreated. Around 6 to 8 months of age, the prostates of these animals were processed for determination of biochemical changes and histopathologies.

**Results:** Prostates of noncarcinogen treated animals that were alcohol exposed during the prenatal period demonstrated inflammatory cell infiltration and epithelial atypia and increased number of proliferative cells in the ventral lobe of this gland, but the prostate of control animal showed normal cytoarchitecture. In addition, prenatal alcohol-exposed rats showed decreased levels of cell-cell adhesion marker and increased estrogenic activity in the ventral prostate. Prenatally ethanol (EtOH)-exposed rats, when treated with carcinogen and testosterone, showed histological evidence for high-grade prostatic intraepithelial neoplasia (PIN) primarily in the ventral prostate, whereas control animals showed only low-grade PIN. Prenatally EtOH-exposed rats treated with carcinogen and testosterone also showed increased number of proliferative cells and androgen receptor with concomitant decreased levels of tumor suppressor proteins in the ventral prostate.

**Conclusions:** These results suggest for the first time that prenatal EtOH exposures induce histophysiological changes in the prostate as well as it increases the susceptibility of the prostate to develop neoplasia during adulthood.

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**104. EARLY FETAL BINGE ALCOHOL EXPOSURE PREDICTS HIGH BEHAVIORAL SYMPTOM SCORES IN 5.5-YEAR-OLD CHILDREN**
Alvik A, Aalen OO, Lindemann R.
Division of Mental Health and Addiction, Institute of Clinical Medicine University of Oslo, Oslo, Norway; Child and Adolescent Mental Health Research Unit, Oslo University Hospital, Oslo, Norway.

**ABSTRACT**

**Background:** Fetal binge alcohol exposure has been associated with neurobehavioral and cognitive symptoms. This study explored whether binge drinking mainly before recognition of pregnancy predicted high symptom scores on the Strengths and Difficulties Questionnaire (SDQ) in 5.5-year-old children.

**Methods:** In a population-based, longitudinal study representative of pregnant women in Oslo, Norway, questionnaires were answered at 17 and 30 weeks of pregnancy, 6 months after term, and at child age 5.5 years (n = 1,116, constituting 66% of the original cohort). Logistic regression analyses identified factors predicting high SDQ scores, and multiple regression analyses identified direct effects on the SDQ Total.

**Results:** Binge exposure (≥5 standard units per occasion [SUpo]) during pregnancy week 0 to 6, that is, 0 to 4 weeks after conception, predicted scores in the Abnormal and Borderline range on the SDQ in 5.5-year-olds, after adjusting for other confounding variables. Very early binge exposure less often than once a week predicted high symptom scores on the SDQ Total (p = 0.05) and Hyperactivity/Inattention (significant), while exposure at least once a week demonstrated a 3- to 5-fold significant increase in high symptom scores on Total,

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Emotional, and Conduct problems. Reporting ≥8 SuPo had stronger predictive power than reporting 5 to 7 SuPo. The results were not explained by participants reporting major lifetime depression. Other predictive factors, although weaker, were maternal symptoms of depression and anxiety during the child's infancy. High education (mother and father), high income (maternal partner), higher child birth weight, and child female sex reduced the likelihood of high SDQ symptom scores. Path analysis demonstrated early binge exposure to have a direct effect on the SDQ Total score.

Conclusions: Binge drinking up to 4 weeks after conception had a strong and direct predictive effect on SDQ symptom scores in 5.5-year-olds. These results strongly support the advice to avoid binge drinking when planning pregnancy.


105. A RANDOMIZED TRIAL COMPARING TELEPHONE VERSUS IN-PERSON BRIEF INTERVENTION TO REDUCE THE RISK OF AN ALCOHOL-EXPOSED PREGNANCY

ABSTRACT
Brief, effective interventions are needed to reduce the risk of an alcohol-exposed pregnancy in women who drink and do not use effective contraception. The Healthy Choices study compared telephone and in-person administration of a brief intervention. In addition to indicators of alcohol use and effective contraception, compliance with the intervention was examined. Women between the ages of 18 and 44 who were drinking above recommended levels and not using effective contraception were randomly assigned to either a telephone (n=68) or in-person (n=63) brief (two sessions) intervention. Overall, participants showed small but significant reductions in alcohol use and larger increases in effective use of contraception. Risk of alcohol-exposed pregnancy was thus significantly reduced, largely due to improved contraception with minor reductions in alcohol use. There was no significant difference in success of the intervention between the two conditions (telephone versus in-person). These findings suggest telephone-based brief intervention may be equally successful and cost-effective in reducing the risk of an alcohol-exposed pregnancy and thus fetal alcohol syndrome.


106. PREVALENCE OF PRESCRIPTION AND ILLICIT DRUGS IN PREGNANCY-ASSOCIATED NON-NATURAL DEATHS OF FLORIDA MOTHERS, 1999-2005
ABSTRACT
Abuse of prescription and illicit drugs has been rapidly increasing. This study examines the prevalence of drug use in the non-natural deaths of pregnant or recently pregnant women. Records from Florida’s Pregnancy Associated Mortality Review conducted between 1999 and 2005 (n = 415) were linked to 385 toxicology reports obtained from Florida medical examiners’ offices. The final study sample consisted of 169 drug-positive, pregnancy-associated non-natural deaths. Of these, 86 were positive for both blood and urine, 64 were positive for blood only and five for urine only, and the remainder were positive for some other specimen. Among these deaths, 91 cases (54%) involved prescription drugs, 78 cases (46%) involved illicit drugs, and 69 cases (41%) involved alcohol. Opioids constituted the majority of deaths associated with prescription drugs. Substantial co-use of opioids and benzodiazepines was seen. Pregnant or recently pregnant women may have more interactions with healthcare providers, which may present more opportunities for intervention and prevention.
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107. CD24 EXPRESSION IDENTIFIES TERATOGEN-SENSITIVE FETAL NEURAL STEM CELL SUBPOPULATIONS: EVIDENCE FROM DEVELOPMENTAL ETHANOL EXPOSURE AND ORTHOTOPIC CELL TRANSFER MODELS
Joseph D. Tingling, Shameena Bake, Rhonda Holgate, Jeremy Rawlings, Phillips P. Nagsuk, Jayashree Chandrasekharan, Sarah L. Schneider, Rajesh C. Miranda

ABSTRACT
Background: Ethanol is a potent teratogen. Its adverse neural effects are partly mediated by disrupting fetal neurogenesis. The teratogenic process is poorly understood, and vulnerable neurogenic stages have not been identified. Identifying these is a prerequisite for therapeutic interventions to mitigate effects of teratogen exposures.

Methods: We used flow cytometry and qRT-PCR to screen fetal mouse-derived neurosphere cultures for ethanol-sensitive neural stem cell (NSC) subpopulations, to study NSC renewal and differentiation. The identity of vulnerable NSC populations was validated in vivo, using a maternal ethanol exposure model. Finally, the effect of ethanol exposure on the ability of vulnerable NSC subpopulations to integrate into the fetal neurogenic environment was assessed following ultrasound guided, adoptive transfer.

Results: Ethanol decreased NSC mRNAs for c-kit, Musashi-1 and GFAP. The CD24+ NSC population, specifically the CD24+CD15+ double-positive subpopulation, was selectively decreased by ethanol. Maternal ethanol exposure also resulted in decreased fetal forebrain CD24 expression. Ethanol pre-exposed CD24+ cells exhibited increased proliferation, and deficits in cell-autonomous and cue-directed neuronal differentiation, and following orthotopic transplantation into naïve fetuses, were unable to integrate into neurogenic niches. CD24-depleted cells retained neurosphere regeneration capacity, but following ethanol exposure, generated increased numbers of CD24+ cells relative to controls.

Conclusions: Neuronal lineage committed CD24+ cells exhibit specific vulnerability, and ethanol exposure persistently impairs this population’s cell-autonomous differentiation capacity. CD24+ cells may additionally serve as quorum sensors within neurogenic niches; their loss, leading to compensatory NSC activation, perhaps depleting renewal capacity. These data collectively advance a mechanistic hypothesis for teratogenesis leading to microencephaly.
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108. NEUROPSYCHOLOGICAL REHABILITATION IN ALCOHOL-RELATED BRAIN DAMAGE: A SYSTEMATIC REVIEW
Jenny Svanberg¹,*, and Jonathan J. Evans²
¹ Forth Valley Substance Misuse Service, Stirling Community Hospital, Stirling FK8 2AU, UK
² Institute of Health and Wellbeing, Glasgow University, Glasgow, UK
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ABSTRACT
Aims: The evidence base for rehabilitating alcohol-related brain damage (ARBD) is still in its infancy. The aim of this review was to collate evidence of intervention studies for ARBD and Wernicke–Korsakoff syndrome (WKS), to offer some indication of methodological quality, and to suggest directions for future research in this area.

Methods: A comprehensive search strategy resulted in systematic review of 16 studies investigating neurorehabilitation of cognitive impairment relating to ARBD.

Results: Most studies addressed rehabilitation of the memory impairments associated with Korsakoff's syndrome, although one study seeking to remediate executive functioning impairment was also included. Three studies outlining service models or approaches were included, with the aim of generating advances in service development for this population.

Conclusion: The reviewed studies were of varying methodology, allowing only tentative conclusions. However, the available evidence suggested benefits of a number of memory rehabilitation strategies. Options for practice are suggested.

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109. NEONATAL ETHANOL EXPOSURE RESULTS IN DOSE-DEPENDENT IMPAIRMENTS IN THE ACQUISITION AND TIMING OF THE CONDITIONED EYEBLINK RESPONSE AND ALTERED CEREBELLAR INTERPOSITUS NUCLEUS AND HIPPOCAMPAL CA1 UNIT ACTIVITY IN ADULT RATS
Lindquist DH, Sokoloff G, Milner E, Steinmetz JE.
Department of Psychology, The Ohio State University, Columbus, OH 43210, USA.
lindquist.40@osu.edu

ABSTRACT
Exposure to ethanol in neonatal rats results in reduced neuronal numbers in the cerebellar cortex and deep nuclei of juvenile and adult animals. This reduction in cell numbers is correlated with impaired delay eyeblink conditioning (EBC), a simple motor learning task in which a neutral conditioned stimulus (CS; tone) is repeatedly paired with a co-terminating unconditioned stimulus (US; periorbital shock). Across training, cell populations in the interpositus (IP) nucleus model the temporal form of the eyeblink-conditioned response (CR). The hippocampus, though not required for delay EBC, also shows learning-dependent increases in CA1 and CA3 unit activity. In the present study, rat pups were exposed to 0, 3, 4, or 5 mg/kg/day of ethanol during postnatal days (PD) 4-9. As adults, CR acquisition and timing were assessed during 6 training sessions of delay EBC with a short (280 ms)
interstimulus interval (ISI; time from CS onset to US onset) followed by another 6 sessions with a long (880 ms) ISI. Neuronal activity was recorded in the IP and area CA1 during all 12 sessions. The high-dose rats learned the most slowly and, with the moderate-dose rats, produced the longest CR peak latencies over training to the short ISI. The low dose of alcohol impaired CR performance to the long ISI only. The 3E (3 mg/kg/day of ethanol) and 5E (5 mg/kg/day of ethanol) rats also showed slower-than-normal increases in learning-dependent excitatory unit activity in the IP and CA1. The 4E (4 mg/kg/day of ethanol) rats showed a higher rate of CR production to the long ISI and enhanced IP and CA1 activation when compared to the 3E and 5E rats. The results indicate that binge-like ethanol exposure in neonatal rats induces long-lasting, dose-dependent deficits in CR acquisition and timing and diminishes conditioning-related neuronal excitation in both the cerebellum and hippocampus.

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111. POTENTIAL IMPACTS OF THE ALBERTA FETAL ALCOHOL SPECTRUM DISORDER SERVICE NETWORKS ON SECONDARY DISABILITIES: A COST-BENEFIT ANALYSIS
Nguyen Xuan Thanh, Jessica Moffatt, Philip Jacobs, Anderson W Chuck, Egon Jonsson
Publication – PubMed
18th July 2013

ABSTRACT
Objectives: To estimate the break-even effectiveness of the Alberta Fetal Alcohol Spectrum Disorder (FASD) Service Networks in reducing occurrences of secondary disabilities associated with FASD.

Methods: The secondary disabilities addressed within this study include crime, homelessness, mental health problems, and school disruption (for children) or unemployment (for adults). We used a cost-benefit analysis approach where benefits of the service networks were the cost difference between the two approaches: having the 12 service networks and having no service network in place, across Alberta. We used a threshold analysis to estimate the break-even effectiveness (i.e. the effectiveness level at which the service networks became cost-saving).

Results: If no network was in place throughout the province, the secondary disabilities would cost $22.85 million (including $8.62 million for adults and $14.24 million for children) per year. Given the cost of network was $6.12 million per year, the break-even effectiveness was estimated at 28% (range: 25% to 32%).

Discussion: Although not all benefits associated with the service networks are included, such as the exclusion of the primary benefit to those experiencing FASD, the benefits to FASD caregivers, and the preventative benefits, the economic and social burden associated with secondary disabilities will “pay-off” if the effectiveness of the program in reducing secondary disabilities is 28%.

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http://www.jptcp.com/pubmed.php?articleId=418

112. OMEGA-3 FATTY ACIDS CAN REVERSE THE LONG-TERM DEFICITS IN HIPPOCAMPAL SYNAPTIC PLASTICITY CAUSED BY PRENATAL ETHANOL EXPOSURE
Patten AR, Sickmann HM, Dyer RA, Innis SM, Christie BR.
Division of Medical Sciences, Island Medical Program, University of Victoria, Victoria, British Columbia, Canada.

ABSTRACT
Fetal alcohol spectrum disorders result in long-lasting neurological deficits including decreases in synaptic plasticity and deficits in learning and memory. In this study we examined the effects of prenatal ethanol exposure on hippocampal synaptic plasticity in male and female Sprague-Dawley rats. Furthermore, we looked at the capacity for postnatal dietary intervention to rescue deficits in synaptic plasticity. Animals were fed an omega-3 enriched diet from birth until adulthood (PND55-70) and in vivo electrophysiology was performed by stimulating the medial perforant path input to the dentate gyrus and recording field excitatory post-synaptic potentials. LTP was induced by administering bursts of five 400 Hz pulses as a theta-patterned train of stimuli (200 ms inter-burst interval). Ethanol-exposed adult males, but
not females, exhibited a significant reduction in LTP. This deficit in male animals was completely reversed with an omega-3 enriched diet. These results demonstrate that omega-3 fatty acids can have benefits following prenatal neuropathological insults and may be a viable option for alleviating some of the neurological deficits associated with FASD.

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113. INADEQUATE INTAKE OF NUTRIENTS ESSENTIAL FOR NEURODEVELOPMENT IN CHILDREN WITH FETAL ALCOHOL SPECTRUM DISORDERS (FASD)
Fuglestad AJ, Fink BA, Eckerle JK, Boys CJ, Hoecker HL, Kroupina MG, Zeisel SH, Georgieff MK, Wozniak JR.
University of Minnesota Twin Cities, United States.

ABSTRACT
This study evaluated dietary intake in children with fetal alcohol spectrum disorders (FASD). Pre-clinical research suggests that nutrient supplementation may attenuate cognitive and behavioral deficits in FASD. Currently, the dietary adequacy of essential nutrients in children with FASD is unknown. Dietary data were collected as part of a randomized, double-blind controlled trial of choline supplementation in FASD. Participants included 31 children with FASD, ages 2.5-4.9 years at enrollment. Dietary intake data was collected three times during the nine-month study via interview-administered 24-hour recalls with the Automated Self-Administered 24-hour Recall. Dietary intake of macronutrients and 17 vitamins/minerals from food was averaged across three data collection points. Observed nutrient intakes were compared to national dietary intake data of children ages 2-5 years (What we Eat in America, NHANES 2007-2008) and to the Dietary Reference Intakes. Compared to the dietary intakes of children in the NHANES sample, children with FASD had lower intakes of saturated fat, vitamin D, and calcium. The majority (>50%) of children with FASD did not meet the Recommended Dietary Allowance (RDA) or Adequate Intake (AI) for fiber, n-3 fatty acids, vitamin D, vitamin E, vitamin K, choline, and calcium. This pattern of dietary intake in children with FASD suggests that there may be opportunities to benefit from nutritional intervention. Supplementation with several nutrients, including choline, vitamin D, and n-3 fatty acids, has been shown in animal models to attenuate the cognitive deficits of FASD. These results highlight the potential of nutritional clinical trials in FASD.

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114. PREDICTORS OF ANTENATAL ALCOHOL USE AMONG AUSTRALIAN WOMEN: A PROSPECTIVE COHORT STUDY
Priority Research Centre for Gender, Health and Ageing, University of Newcastle, Callaghan, NSW, Australia.

ABSTRACT
Objective: To identify predictors of antenatal alcohol consumption among women who

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usually consume alcohol.

**Design:** Prospective cohort study.

**Setting:** Australian Longitudinal Study on Women's Health (ALSWH).

**Population or sample:** A total of 1969 women sampled from the ALSWH 1973-78 cohort.

**Methods:** Women were included if they were pregnant in 2000, 2003, 2006 or 2009. The relationship between antenatal alcohol consumption and sociodemographics, reproductive health, mental health, physical health, health behaviours, alcohol guidelines and healthcare factors was investigated using a multivariate logistic regression model.

**Main outcome measures:** Alcohol use during pregnancy.

**Results:** Most (82.0%) women continued to drink alcohol during pregnancy. Women were more likely to drink alcohol during pregnancy if they had consumed alcohol on a weekly basis before pregnancy (odds ratio [OR] 1.47; 95% confidence interval [95% CI] 1.13-1.90), binge drank before pregnancy (OR 2.28; 95% CI 1.76-2.94), or if they were pregnant while alcohol guidelines recommended low alcohol versus abstinence (OR 1.60; 95% CI 1.26-2.03). Drinking during pregnancy was less likely if women had a Health Care Card (OR 0.63; 95% CI 0.45-0.88) or if they had ever had fertility problems (OR 0.64; 95% CI 0.48-0.86).

**Conclusions:** Most Australian women who drank alcohol continued to do so during pregnancy. Prepregnancy alcohol consumption was one of the main predictors of antenatal alcohol use. Alcohol guidelines, fertility problems and Health Care Card status also impacted antenatal alcohol consumption.


115. **ASSESSING IMPULSIVITY IN PREPUBERTAL MALE RATS: A NOVEL DEVICE AND METHOD TO ASSESS MOTOR AND COGNITIVE IMPULSIVITY**

Juárez J, Muñoz-Villegas P, Guerrero-Álvarez A, Flores-Ocampo P. Laboratorio de Farmacología y Conducta, Instituto de Neurociencias, CUCBA, Universidad de Guadalajara, Guadalajara, Jalisco, México. jjuarez@cencar.udg.mx

**ABSTRACT**

The use of animal models in studies of impulsivity has made valuable contributions to our understanding of this behavioral trait as it relates to disorders such as attention deficit hyperactivity disorder. The objective of this work was to develop a paradigm that would make it possible to evaluate both motor and cognitive impulsivity using the same device after a short training period. The operant behavior demanded in this device consists in having rats cross a bridge after receiving a signal to obtain a reward that is available on a goal platform in a Wait-to-Go-signal task, or in crossing a bridge after the animals make a choice between two alternatives in a Delay-discounting task. To test this device and method, a study was conducted using an animal model of dopaminergic dysfunction produced by prenatal alcohol treatment (which has been shown to cause attention deficits and alterations of impulsivity in adult rats). Compared with controls, prepubertal male rats treated prenatally with alcohol showed both higher cognitive and higher motor impulsivity as assessed by the parameters used. Although attention changes proved not to be dependent on prenatal treatment, they were sensitive to the task performed. The device and methods introduced herein thus
constitute useful instruments for evaluating impulsivity. Their significant advantages include a short investment in training time, and the ability to assess different types of impulsivity from the vantage point of distinct theoretical perspectives.


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116. IN UTERO DRUG AND ALCOHOL EXPOSURE IN INFANTS BORN TO MOTHERS PRESCRIBED MAINTENANCE METHADONE
McGlone L, Mactier H, Hassan H, Cooper G.
Neonatal Unit, Royal Hospital for Sick Children, , Glasgow, UK.

ABSTRACT
Aims: To describe the prevalence of in utero alcohol and illicit drug exposure in infants born to mothers prescribed methadone in pregnancy, and to compare the accuracy of maternal interview with infant toxicology.

Methods: Urine and meconium samples were collected from 56 infants born to mothers prescribed methadone during pregnancy and a confidential interview conducted soon after delivery. Samples were screened for drugs of misuse and meconium samples analysed for the presence of fatty acid ethyl esters (FAEEs) to detect prenatal alcohol exposure.

Results: 91% of infants had been exposed to illicit drugs in utero, including opiates (73%), benzodiazepines (70%) and cannabinoids (59%). 47% of infants had elevated FAEEs. Meconium was more sensitive at detecting in utero drug exposure than urine toxicology (p<0.01 for opiates, benzodiazepines, cannabinoids) or maternal interview (p=0.03 for opiates, p<0.01 for cannabinoids).

Conclusions: The majority of infants born to mothers prescribed methadone during pregnancy are exposed to polysubstance misuse, and almost one-half additionally exposed to excess alcohol.


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Published online July 8, 2013

117. PRENATAL ALCOHOL EXPOSURE AND EDUCATIONAL ACHIEVEMENT IN CHILDREN AGED 8–9 YEARS
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Centre for Population Health Research, Curtin University, Perth, Australia;
Division of Population Sciences, Telethon Institute for Child Health Research, West Perth, Australia;
ABSTRACT
Objective: This study examines the relationships between the dose, pattern, and timing of prenatal alcohol exposure and achievement in reading, writing, spelling, and numeracy in children aged 8 to 9 years.

Methods: Data from a randomly selected, population-based birth cohort of infants born to non-Indigenous women in Western Australia between 1995 and 1997 (n = 4714) (Randomly Ascertained Sample of Children born in Australia’s Largest State Study cohort) were linked to the Western Australian Midwives’ Notification System and the Western Australian Literacy and Numeracy Assessment statewide education testing program. The records for 86% (n = 4056) of the cohort were successfully linked with education records when the children were aged 8 to 9 years. The associations between prenatal alcohol exposure and achievement of national benchmarks in school numeracy, reading, spelling, and writing tests and nonattendance for the tests was examined. Logistic regression was used to generate adjusted odds ratios (aOR) and 95% confidence intervals (CI), adjusting for potential confounding factors. The referent group included children of mothers who previously drank alcohol but who abstained during pregnancy.

Results: Children were twice as likely not to achieve the benchmark for reading after heavy prenatal alcohol exposure during the first trimester (aOR 2.26; 95% CI 1.10–4.65) and for writing when exposed to occasional binge drinking in late pregnancy (aOR 2.35; 95% CI 1.04–5.43). Low-moderate prenatal alcohol exposure was not associated with academic underachievement.

Conclusions: The type of learning problems expressed depends on the dose, pattern, and timing of prenatal alcohol exposure.

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PubMed, Law Hum Behav. 2013 Jul 8. [Epub ahead of print]

118. EVALUATING THE PSYCHOLEGAL ABILITIES OF YOUNG OFFENDERS WITH FETAL ALCOHOL SPECTRUM DISORDER
McLachlan K, Roesch R, Viljoen JL, Douglas KS.

ABSTRACT
Individuals with a diagnosis of fetal alcohol spectrum disorder (FASD) experience a range of physical, cognitive, and behavioral deficits thought to interfere with their ability to competently navigate the arrest, interrogation, and trial process. This study examined the psycholegal abilities of young offenders with FASD, including their understanding and appreciation of Miranda rights, and adjudication capacities (factual knowledge of criminal procedure, appreciation of the nature and object of the proceedings, ability to participate in a defense and communicate with counsel). Two groups of young offenders (50 with FASD and 50 without prenatal alcohol exposure) completed Grisso’s Instruments for Assessing Understanding and Appreciation of Miranda rights and the Fitness Interview Test-Revised to assess overall rates of impairment in youth with FASD, as well as differences between the groups. Potentially important predictors of psycholegal abilities were also evaluated. Results indicated the majority of young offenders with FASD (90%) showed impairment in at least one psycholegal ability, and rates of impairment were significantly higher than the comparison group. However, considerable within-group variability was observed. IQ and reading comprehension emerged as robust predictors of participants’ psycholegal abilities, while the FASD diagnosis emerged as an additional predictor of psycholegal impairment.

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differentiated participants’ scores on the FIT-R. These findings underscore the importance of individualized and comprehensive forensic assessments of psycholegal abilities in this population when warranted. Additional system level strains for this population are discussed, including problems in approaching competency remediation, and the potentially growing need for accommodation and forensic assessments in the face of limited financial and professional resources in legal settings.

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119. MICRODELETION 11Q13.1.Q13.2 IN A PATIENT PRESENTING WITH DEVELOPMENTAL DELAY, FACIAL DYSMORPHISM, AND ESOPHAGEAL ATRESIA: POSSIBLE ROLE OF THE GSTP1 GENE IN ESOPHAGUS MALFORMATION
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ABSTRACT

Background: Esophageal atresia is a major congenital malformation characterized by a complete interruption of the esophageal continuity. It is frequently observed in associations and syndromes. As an isolated finding, it has a multifactorial etiology whose genetic factors are poorly known. Recently, the GST family, especially the GSTM1 null genotype (but not the GSTP1 polymorphism I105V), has been associated with esophageal atresia. These enzymes play a role in phase II detoxification of xenobiotics. Here we present the clinical and molecular findings observed in a patient suggesting that the loss of the GSTP1 allele might predispose to this malformation.

Case: We describe a patient presenting with esophageal atresia associated with developmental delay and facial dysmorphism, whose mother used tobacco and alcohol during the first 2 months of her pregnancy. Microdeletion/microduplication analysis was performed using comparative genomic hybridization and a 180K Agilent array. It detected a de novo 2 Mb chromosome 11q13.1.q13.2 deletion.

Conclusion: The deleted chromosomal segment includes the GSTP1 gene. We hypothesize that the deletion of one GSTP1 allele (an isoform highly expressed in embryonic tissues), associated with specific environmental factors, such as tobacco and alcohol, could cause the esophageal atresia observed in our patient.

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120. EXPLORING THE POTENTIAL TO USE DATA LINKAGE FOR INVESTIGATING THE RELATIONSHIP BETWEEN BIRTH DEFECTS AND PRENATAL ALCOHOL EXPOSURE
O'Leary CM, Elliott EJ, Nassar N, Bower C.
Centre for Population Health Research, Curtin University, Perth, Western Australia. colleen.oleary@curtin.edu.au

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ABSTRACT

Background: This study explores the potential of data linkage to investigate the proportion of birth defects classified as alcohol-related (ARBD) by the Institutes of Medicine (IOM) that are attributable to maternal alcohol-use disorder.

Methods: Maternal alcohol-use disorder was identified using International Classification of Diseases (9th and 10th revision) codes for alcohol-related diagnoses recorded on record-linked Western Australian health, mental health, and/or drug and alcohol datasets 1983 to 2007. Children of these mothers (n=23,859) were compared with a randomly selected cohort of children born to mothers without an alcohol diagnosis, frequency-matched by maternal age, Aboriginal status, and child's birth year (n=61,370). Birth defects were identified through linkage with the Western Australian Register of Developmental Anomalies and defects with chromosomal causes were excluded. Associations between overall and individual IOM-designated ARBD and a maternal alcohol-related diagnosis recorded "during pregnancy" or "any" diagnosis (before/during/after pregnancy) was assessed using multivariate logistic regression to generate odds ratios and 95% confidence intervals. Population-attributable fractions were calculated for significant results using total population numbers.

Results: There was a significant association between maternal alcohol-related diagnoses recorded during pregnancy and ARBD (adjusted odds ratio, 3.14; 95% confidence interval, 2.49-3.96), with an attributable fraction of 0.57%. "Any" maternal alcohol diagnosis demonstrated a higher attributable fraction for ARBD (1.53%), with the highest attributable fractions for microcephaly (7.31%), ptosis (3.75%), atrial septal defect (2.86%), and conotruncal heart defects (2.01%).

Conclusion: Research using linked, population-based administrative health data has the potential to advance knowledge of ARBD. Routine collection and recording of alcohol use during pregnancy for all pregnant women is required and would enhance this methodology.


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121. PROMOTING ABSTINENCE FROM ALCOHOL DURING PREGNANCY: IMPLICATIONS FROM FORMATIVE RESEARCH

France KE, Donovan RJ, Henley N, Bower C, Elliott EJ, Payne JM, D’Antoine H, Bartu AE. School of Marketing, Tourism and Leisure, Edith Cowan University, Joondalup, Australia.

ABSTRACT

This research developed messages to promote abstinence from alcohol during pregnancy and identified elements that enhance message persuasiveness. An exploratory phase was conducted in 2009 that comprised four focus groups with 23 women in Western Australia and elicited beliefs and attitudes on alcohol use during pregnancy and motivations for behavior change. Four television concepts were subsequently developed and appraised in five focus groups with 31 participants using standard advertising pretesting questions. The implications for campaigns addressing prenatal alcohol exposure and further research are noted and limitations discussed. Funding was received from Healthway and the National Health and Medical Research Council.


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122. COMMENTARY ON DAY AND COLLEAGUES : THE ASSOCIATION BETWEEN PRENATAL ALCOHOL EXPOSURE AND BEHAVIOR AT 22 YEARS OF AGE--ADVERSE EFFECTS OF RISKY PATTERNS OF DRINKING AMONG LOW TO MODERATE ALCOHOL-USING PREGNANT WOMEN
Jacobson SW, Carter RC, Jacobson JL.
Department of Psychiatry and Behavioral Neurosciences, Wayne State University School of Medicine, Detroit, MI 48201, USA. sandra.jacobson@wayne.edu

ABSTRACT
Day and colleagues have presented the first data showing that the behavioral effects of low to moderate prenatal alcohol exposure seen in childhood and adolescence persist into adulthood. Using the Achenbach Adult Self-Report, they found dose-dependent effects of prenatal exposure on internalizing, externalizing, and attention problems that persist in young adults and, thus, appear to be permanent. To date, few studies have attempted to identify thresholds at which prenatal alcohol exposure is harmful, although the animal literature suggests that even 1 to 2 binge episodes can result in adverse effects in the offspring. Four prospective longitudinal studies have reported adverse effects at what can be characterized as moderate exposure levels based on NIAAA criteria, but moderate drinking women often concentrate their alcohol use on 1 to 2 days per week, thereby engaging in binge drinking. In this study, binge drinking was not a strong predictor of adverse outcome when average daily dose was held constant, a conclusion that the authors note runs "counter to studies that have reported that binge drinking has a greater effect." This inconsistency may be due to the difficulty of allocating variance that is shared (overlapping) between average daily dose and binge drinking (i.e., dose/occasion). Data from laboratory animal studies, in which dosage can be manipulated experimentally, demonstrate that a higher dose per occasion, the key feature of binge drinking, leads to more severe adverse effects. Day and colleagues' findings of adverse effects at low levels of exposure provides clear evidence that there is no safe level of drinking during pregnancy and that, even at low levels, drinking results in irreversible behavioral impairment. On the other hand, given the evidence from the animal and most human studies, it is important for all women who drink during pregnancy, even at light to moderate levels, to recognize that minimizing their intake per occasion and refraining from binge drinking can reduce risk to the fetus.

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123. RISK FACTORS FOR DEVELOPING ATOPIC DERMATITIS
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COPSAC (COpenhagen Prospective Studies on Asthma in Childhood), Gentofte University Hospital, Ledreborg Alle 34, 2820 Gentofte, Denmark. c.giwerman@hotmail.com

ABSTRACT
The aim of this thesis was to investigate possible risk factors affecting the development of AD. AD is a frequent disease among children and has a substantial impact on the lives of both the child and its family. A better understanding of the disease would enable better treatment, prevention and information to the families involved. Previous risk factor studies have been hampered by an unsuitable study design and/or difficulties in standardization when diagnosing AD, which limit their conclusions. In paper I, we conducted a traditional cross-
sectional analysis testing 40 possible risk factors for developing AD at 3 years of age. Our data suggested a strong heredity of AD and confirmed the risk associated with the non-functional FLG allele mutations after adjustments for confounders. Besides this mother’s dermatitis and father's allergic rhinitis were found to increase the risk of AD. Perinatal exposure to dog was the only environmental exposure that significantly reduced the disease manifestation, suggesting other, yet unknown environmental factors affecting the increasing prevalence of AD in children. Length at birth was shown to be inversely associated with the risk of later developing AD. This traditional risk factor analysis led to two borderline significant results: duration of exclusive breastfeeding and mother's alcohol intake during the 3rd trimester. Since these possible two risk factors could neither be rejected nor accepted, we decided to do two in-depth studies, further investigating these, using longitudinal data information and data analysis instead of the traditional cross-sectional approach (paper II & III). In paper II, we investigated the risk of developing AD and wheezy symptoms until age 2 years depending on duration of breastfeeding. We found an increased risk of AD, but a protective effect on wheezy disorders in infancy from exclusive breastfeeding. The effect of exclusive breastfeeding on the risk of development of AD was significant after adjustment for demographics, FLG variants R501X and 2282del4 status, parent’s AD and pets at home (RR 2.09, 95% CI 1.15-3.80, p=0.016). In addition, there was a significant effect of duration of exclusive breastfeeding (p=0.043), as the relative risk of AD was increased in proportion to increased duration of breastfeeding. The risk associated with exclusive breastfeeding was not explained by the fatty acid composition of mother's milk, though a trend showed higher risk of AD if mother's milk had low concentrations of n-3 fatty acids. In paper III, we found that alcohol intake during pregnancy was associated with a significantly higher risk of developing AD in the offspring, with the effect persisting throughout the whole 7 years follow-up period (HR 1.44, 95% CI 1.05-1.99, p=0.024). The increased risk was still significant after confounder adjustment for mother's education, AD and smoking habits during the 3rd trimester. There was no association between alcohol intake during pregnancy and other atopic endpoints (wheeze episodes, asthma, allergic rhinitis, blood eosinophil count, total IgE, sensitization, cord blood IgE and nasal eosinophilia). However, the underlying explanation was not clear. The thesis is based on data collected as part of the ongoing COPSAC cohort. The cohort is a longitudinal, prospective birth cohort following 411 children born to mothers with asthma. This selection of high-risk children restricts the interpretation of the results and they cannot necessarily be expanded to apply to the general population.


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124. ALCOHOL USE DISORDERS AND HOSPITAL-ACQUIRED INFECTIONS IN WOMEN UNDERGOING CESAREAN DELIVERY

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Division of Pulmonary Disease and Critical Care Medicine, Department of Internal Medicine, Virginia Commonwealth University, Richmond, Virginia 23298, USA.

ABSTRACT

Objective: To determine the risk of hospital-acquired infection in women with alcohol use disorders undergoing cesarean delivery.

Methods: Using the Nationwide Inpatient Sample, we conducted a retrospective cohort study of women undergoing cesarean delivery from 2002 to 2010. Women with a diagnosis of alcohol use disorder were compared with women without alcohol use disorders. Hospital-acquired infections include surgical site infection, endometritis, urinary tract infection, sepsis, and pneumonia.
Results: A total of 12,081 women with alcohol use disorders were identified and matched with 11,960 women without alcohol use disorders. Women with alcohol use disorders were more likely to have development of urinary tract infection and sepsis. By multivariable analyses, women with alcohol use disorders had higher odds of hospital-acquired infections (odds ratio 2.2, 95% confidence interval [CI] 1.9-2.7; P=2×10; 397 among those with alcohol use disorders and 179 among those without alcohol use disorders; number needed to harm 55). Length of stay was longer in women with alcohol use disorders, but this was unexplained by hospital-acquired infection (3.3 days; 95% CI 3.2-3.3 compared with 3.1 days; 95% CI 3.0-3.1; P=4×10).

Conclusion: Women with alcohol use disorders undergoing cesarean delivery have increased risk of hospital-acquired infections. Interventions aimed at decreasing alcohol use disorders during pregnancy may reduce maternal and fetal complications.

Level of evidence: II.


125. DEPRESSIVE SYMPTOMS MODERATE TREATMENT RESPONSE TO BRIEF INTERVENTION FOR PREVENTION OF ALCOHOL EXPOSED PREGNANCY

Penberthy JK, Hook JN, Hettema J, Farrell-Carnahan L, Ingersoll K. Center for Addiction Research and Education, Department of Psychiatry and Neurobehavioral Sciences, University of Virginia School of Medicine, P.O. Box 800623, Charlottesville, VA 22908, USA. jkp2n@virginia.edu

ABSTRACT
The previously published randomized controlled trial, EARLY, tested the efficacy of a motivational interviewing (MI) plus feedback condition against a video information (VI) condition and an informational brochure (IB) condition in reducing drinking and/or increasing contraception effectiveness, and found that drinking and rates of effective contraception improved in all conditions. In this reanalysis of the data from EARLY, potential moderating effects of depressive, global distress, and anxiety symptoms in response to the three brief interventions to reduce alcohol exposed pregnancy risk were examined. Women with higher levels of depression at baseline reported greater improvements in the MI plus feedback condition versus the VI and IB conditions with depression moderating both drinking behavior and contraceptive effectiveness. Global distress moderated only drinking behavior in the MI plus feedback but not other groups and anxiety was not a moderator of outcome in any of the intervention groups. Depressed or distressed women at risk for AEP may benefit from an AEP risk reduction intervention that incorporates interaction with a treatment provider versus educational information provided via video or written materials.

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126. CHRONIC ETHANOL EXPOSURE DURING DEVELOPMENT: DISTURBANCES OF BREATHING AND ADAPTATION

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**ABSTRACT**
The effects of prenatal exposure to some drugs of abuse, such as nicotine, on breathing function have been clearly established. However, the case of alcohol (ethanol), the most widely consume drug of abuse, remains unknown. Prenatal ethanol consumption in humans may lead to fetal alcohol syndrome and although the effect of chronic prenatal ethanol exposure (CPEE) on cognitive function is frequently studied, nothing is known about CPEE’s effects on breathing as compared with other drugs of abuse. The role of nicotine for example, in human neonatal pathology, such as sudden infant death syndrome, is acknowledged today, whereas the full scope of CPEE’s role is only recently emerging. Here, we review preclinical investigations on the effects of CPEE on breathing in different animal models, including possible mechanisms of adaptation to CPEE. These recent preclinical studies shed new light on a widely used drug of abuse and should facilitate the understanding of the danger posed by alcohol consumption during pregnancy.


**127. SUPPRESSION AND EPIGENETIC REGULATION OF MIR-9 CONTRIBUTES TO ETHANOL TERATOLOGY: EVIDENCE FROM ZEBRAFISH AND MURINE FETAL NEURAL STEM CELL MODELS**
Pappalardo-Carter DL, Balaraman S, Sathyan P, Carter ES, Chen WJ, Miranda RC.
Department of Neuroscience and Experimental Therapeutics, Texas A&M Health Science Center, College of Medicine, Bryan, Texas.

**ABSTRACT**
**Background:** Fetal alcohol exposure produces multiorgan defects, making it difficult to identify underlying etiological mechanisms. However, recent evidence for ethanol (EtOH) sensitivity of the miRNA miR-9 suggests one mechanism, whereby EtOH broadly influences development. We hypothesized that loss of miR-9 function recapitulates aspects of EtOH teratology.

**Methods:** Zebrafish embryos were exposed to EtOH during gastrulation, or injected with anti-miR-9 or nonsense control morpholinos during the 2-cell stage of development and collected between 24 and 72 hours postfertilization (hpf). We also assessed the expression of developmentally important, and known miR-9 targets, FGFR-1, FOXP2, and the nontargeted transcript, MECP2. Methylation at CpG islands of mammalian miR-9 genes was assessed in fetal murine neural stem cells (mNSCs) by methylation-specific PCR, and miRNA processing assessed by qRT-PCR for pre-miR-9 transcripts.

**Results:** EtOH treatment and miR-9 knockdown resulted in similar cranial defects including microcephaly. Additionally, EtOH transiently suppressed miR-9, as well as FGFR-1 and FOXP2, and alterations in miR-9 expression were correlated with severity of EtOH-induced teratology. In mNSCs, EtOH increased CpG dinucleotide methylation at the miR-9-2 locus and accumulation of pre-miR-9-3.

**Conclusions:** EtOH exerts regulatory control at multiple levels of miR-9 biogenesis. Moreover, early embryonic loss of miR-9 function recapitulated the severe range of teratology associated with developmental EtOH exposure. EtOH also disrupts the relationship between miR-9 and target gene expression, suggesting a nuanced relationship between EtOH and miRNA regulatory networks in the developing embryo. The implications of these data for the expression and function of mature miR-9 warrant further investigation.
128. PRENATAL ALCOHOL EXPOSURE AND CHILDHOOD ATOPIC DISEASE: A MENDELIAN RANDOMIZATION APPROACH
Centre for Primary Care and Public Health, Barts and The London School of Medicine and Dentistry, London, United Kingdom. Electronic address: s.shaheen@qmul.ac.uk.

ABSTRACT
Background: Alcohol consumption in western pregnant women is not uncommon and could be a risk factor for childhood atopic disease. However, reported alcohol intake may be unreliable, and associations are likely to be confounded.

Objective: We aimed to study the relation between prenatal alcohol exposure and atopic phenotypes in a large population-based birth cohort with the use of a Mendelian randomization approach to minimize bias and confounding.

Methods: In white mothers and children in the Avon Longitudinal Study of Parents and Children (ALSPAC) we first analyzed associations between reported maternal alcohol consumption during pregnancy and atopic outcomes in the offspring measured at 7 years of age (asthma, wheezing, hay fever, eczema, atopy, and total IgE). We then analyzed the relation of maternal alcohol dehydrogenase (ADH)1B genotype (rs1229984) with these outcomes (the A allele is associated with faster metabolism and reduced alcohol consumption and, among drinkers, would be expected to reduce fetal exposure to ethanol).

Results: After controlling for confounders, reported maternal drinking in late pregnancy was negatively associated with childhood asthma and hay fever (adjusted odds ratio [OR] per category increase in intake: 0.91 [95% CI, 0.82-1.01] and 0.87 [95% CI, 0.78-0.98], respectively). However, maternal ADH1B genotype was not associated with asthma comparing carriers of A allele with persons homozygous for G allele (OR, 0.98 [95% CI, 0.66-1.47]) or hay fever (OR, 1.11 [95% CI, 0.71-1.72]), nor with any other atopic outcome.

Conclusion: We have found no evidence to suggest that prenatal alcohol exposure increases the risk of asthma or atopy in childhood.

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ABSTRACT
Audio computer-assisted survey instrument (ACASI) has been shown to decrease under-reporting of socially undesirable behaviours, but has not been evaluated in pregnant women at risk of HIV acquisition in Brazil. We assigned HIV-negative pregnant women receiving routine antenatal care at in Porto Alegre, Brazil and their partners to receive a survey regarding high-risk sexual behaviours and drug use via ACASI (n = 372) or face-to-face (FTF) (n = 283) interviews. Logistic regression showed that compared with FTF, pregnant women interviewed via ACASI were significantly more likely to self-report themselves as single (14% versus 6%), having >5 sexual partners (35% versus 29%), having oral sex (42% versus 35%), using intravenous drugs (5% versus 0), smoking cigarettes (23% versus 16%), drinking alcohol (13% versus 8%) and using condoms during pregnancy (32% versus 17%). Therefore, ACASI may be a useful method in assessing risk behaviours in pregnant women, especially in relation to drug and alcohol use.


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exposure at 18 weeks, adjusted OR 1.25 (95% CI 1.06 to 1.48). Similar results were found for both paternal and postnatal maternal alcohol exposure. A Mendelian-randomization approach was used to estimate the association between maternal genotype and offspring balance using the non-synonymous variant rs1229984*A (ADH1B) to proxy for lower maternal alcohol consumption; no strong associations were found between this genotype/proxy and offspring balance.

**Conclusions:** No evidence was found to indicate that moderate maternal alcohol consumption in this population sample had an adverse effect on offspring balance at age 10. An apparent beneficial effect of higher total maternal alcohol consumption on offspring balance appeared likely to reflect residual confounding.

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**131. ACETALDEHYDE REINFORCEMENT AND MOTOR REACTIVITY IN NEWBORNS WITH OR WITHOUT A PRENATAL HISTORY OF ALCOHOL EXPOSURE**
March SM, Culleré ME, Abate P, Hernández JI, Spear NE, Molina JC. Laboratorio de Alcohol, Ontogenia y Desarrollo, Instituto de Investigación Médica Mercedes y Martín Ferreyra Córdoba, Argentina; Facultad de Psicología, Universidad Nacional de Córdoba, Cátedra Psicobiología Experimental Córdoba, Argentina.

**ABSTRACT**
Animal models have shown that early ontogeny seems to be a period of enhanced affinity to ethanol. Interestingly, the catalase system that transforms ethanol (EtOH) into acetaldehyde (ACD) in the brain, is more active in the perinatal rat compared to adults. ACD has been found to share EtOH’s behavioral effects. The general purpose of the present study was to assess ACD motivational and motor effects in newborn rats as a function of prenatal exposure to EtOH. Experiment 1 evaluated if ACD (0.35 μmol) or EtOH (0.02 μmol) supported appetitive conditioning in newborn pups prenatally exposed to EtOH. Experiment 2 tested if prenatal alcohol exposure modulated neonatal susceptibility to ACD’s motor effects (ACD dose: 0, 0.35 and 0.52 μmol). Experiment 1 showed that EtOH and ACD supported appetitive conditioning independently of prenatal treatments. In Experiment 2, latency to display motor activity was altered only in neonates prenatally treated with water and challenged with the highest ACD dose. Prenatal EtOH experience results in tolerance to ACD’s motor activity effects. These results show early susceptibility to ACD’s appetitive effects and attenuation of motor effects as a function of prenatal history with EtOH, within a stage in development where brain ACD production seems higher than later in life.

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132. LOW-DOSE THYROXINE ATTENUATES AUTISM-ASSOCIATED ADVERSE EFFECTS OF FETAL ALCOHOL IN MALE OFFSPRING’S SOCIAL BEHAVIOR AND HIPPOCAMPAL GENE EXPRESSION

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ABSTRACT

Background: Fetal alcohol spectrum disorder (FASD) is characterized by neurodevelopmental anomalies manifesting in cognitive and behavioral deficits in the offspring with diverse severities. Social behavior is affected in FASD, and these deficits overlap with those of autism spectrum disorder (ASD). Identifying some of the molecular characteristics related to ASD in an animal model of FASD could ultimately provide details on the underlying molecular mechanisms of both disorders that could lead to novel treatments.

Methods: Pregnant Sprague–Dawley rats received the following diets: control (C; ad libitum standard laboratory chow), nutritional control pair-fed (PF), ethanol (EtOH), or an EtOH diet supplemented with 0.3, 1.5, or 7.5 mg thyroxine (T₄)/l in the diet. Social behavior and memory were tested in the adult offspring. Plasma total T₄, free T₃ (fT₃), and thyroid-stimulating hormone (TSH) levels were measured. Hippocampal expression of Gabrb3, Ube3a, Nr2b, Rasgrf1, and Dio3 were measured by RT-qPCR and protein levels of Mecp2 and Slc25a12 by Western blotting.

Results: Adult male offspring of EtOH dams showed elevated fT₃ and low TSH levels. Adult male, but not female, offspring of EtOH dams exhibited social behavior and memory deficits. Expression of autism candidates, Gabrb3, Ube3a, Mecp2, and Slc25a12, was significantly increased in the hippocampus of male offspring of EtOH dams. Hippocampal Nr2b and Dio3 were also increased, while Rasgrf1 was decreased in the same population. Peripheral thyroid function, social behavioral deficits, and altered expression of the above genes were normalized by simultaneous administration of 0.3 mg/l T₄ in the EtOH diet.

Conclusions: Our data suggest that social interaction deficits of FASD share molecular mechanism with ASD by showing altered hippocampal expression of several ASD candidate genes. Social interaction deficits as well as the gene expression changes in the offspring of EtOH-consuming dams can be reversed by low dose of thyroid hormone supplementation to the mothers.


133. MODERATE-LEVEL PRENATAL ALCOHOL EXPOSURE ENHANCES ACOUSTIC STARTLE MAGNITUDE AND DISRUPTS PREPULSE INHIBITION IN ADULT RHESUS MONKEYS

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ABSTRACT

Background: Prenatal alcohol exposure can contribute to a wide range of neurodevelopmental impairments in children and adults including behavioral and neuropsychiatric disorders. In rhesus monkeys, we examined whether moderate-level prenatal alcohol exposure would alter acoustic startle responses and prepulse inhibition (PPI) of the acoustic startle. PPI is a highly quantifiable measure of inhibitory neural processes or sensorimotor gating associated with neuropsychiatric disorders.

Methods: Acoustic startle and PPI of the acoustic startle were tested in 37 adult rhesus monkeys (Macaca mulatta) from 4 experimental conditions: (i) moderate-level prenatal alcohol-exposed, (ii) prenatally stressed, (iii) moderate-level prenatal alcohol-exposed + prenatally stressed, and (iv) sucrose controls.

Results: Prenatal alcohol-exposed monkeys showed a higher magnitude of acoustic startle response and disrupted PPI compared with monkeys not exposed to alcohol prenatally. Monkeys in all conditions showed higher hypothalamic-pituitary-adrenocortical (HPA) axis responses after undergoing the startle procedure, but HPA responses were unrelated to startle response magnitude, latency, or PPI.

Conclusions: Finding altered PPI in monkeys prenatally exposed to a moderate dose of alcohol suggests that reduced sensorimotor gating is 1 effect of prenatal alcohol exposure. Because reduced sensorimotor gating is observed in many neuropsychiatric disorders, sensorimotor gating deficits could be an aspect of the comorbidity between fetal alcohol spectrum disorder and mental health conditions.


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Results: A total of 175 new mothers had 287 hospital admissions with the principal or stay AUD diagnoses during the study period in NSW. Of the 287 admissions, 181 admissions (63.07%) were reported for an alcohol-related disorder as the principal diagnosis. The hospital admission rate for AUD was 1.76/1,000 person-years (PY) (95% CI: 1.45 to 2.07) during the 6 months prepregnancy. The rate decreased to 0.49/1,000 PY (95% CI: 0.36 to 0.63) during pregnancy and to 0.82/1,000 PY (95% CI: 0.67 to 0.97) in the first year after birth. Women who smoked during pregnancy, lived in a remote area and were younger than 25 years, were more likely to be admitted to hospital with AUD diagnoses. Women in the middle disadvantaged quintile and born in other countries were less likely to be admitted to hospital with AUD diagnoses.

Conclusions: Hospital admission for AUD decreased significantly in pregnancy and the first year postpartum compared to the prepregnancy period.


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135. FEASIBILITY AND PROMISE OF A REMOTE-DELIVERED PRECONCEPTION MOTIVATIONAL INTERVIEWING INTERVENTION TO REDUCE RISK FOR ALCOHOL-EXPOSED PREGNANCY

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3 Department of Physical Medicine & Rehabilitation, Virginia Commonwealth University, Richmond, Virginia.
4 College of William and Mary, Williamsburg, Virginia.

ABSTRACT

Background: Alcohol-exposed pregnancy (AEP) is a leading cause of birth defects. Effective face-to-face preconception interventions based on motivational interviewing (MI) exist and should be translated into remote formats for maximum public health impact. This study investigated the feasibility and promise of a one-session, remote-delivered, preconception, MI-based AEP intervention (EARLY Remote) for non–treatment-seeking community women.

Subjects and Methods: This was a single-arm, prospective pilot intervention study. All participants received the intervention via telephone and mail. Feasibility of remote-delivery methods, treatment engagement, treatment credibility, MI treatment integrity, and therapeutic alliance were examined. Outcomes were 3- and 6-month drinks per drinking day (DDD), rate of unreliable contraception, and proportion of women at risk for AEP due to continued risk drinking and no or unreliable contraception use.
**Results:** Feasibility of remote delivery was established; participants were engaged by the intervention and rated it as credible. Integrity to MI and therapeutic alliance were good. Both DDD and rate of unreliable contraception decreased significantly over time. Proportions of women who drank at risk levels, used unreliable or no contraception, and/or were at risk for AEP in the past 90 days decreased significantly from baseline to 6 months.

**Conclusions:** Remote delivery was feasible, and the translated remote intervention may reduce AEP risk. Refinement of EARLY Remote may facilitate its placement within a spectrum of effective MI-based preconception AEP interventions as part of a stepped-care approach. EARLY Remote may have an important role within a stepped-care model for dissemination to geographically disperse women at risk for AEP. This could result in substantial public health impact through reduction of AEP on a larger scale.

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http://online.liebertpub.com/doi/abs/10.1089/tmj.2012.0247

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**ABSTRACT**

Diffusion tensor imaging (DTI) of brain development in fetal alcohol spectrum disorders (FASD) has revealed structural abnormalities, but studies have been limited by the use of cross-sectional designs. Longitudinal scans can provide key insights into trajectories of neurodevelopment within individuals with this common developmental disorder. Here we evaluate serial DTI and T1-weighted volumetric MRI in a human sample of 17 participants with FASD and 27 controls aged 5-15 years who underwent 2-3 scans each, ~2-4 years apart (92 scans total). Increases of fractional anisotropy and decreases of mean diffusivity (MD) were observed between scans for both groups, in keeping with changes expected of typical development, but mixed-models analysis revealed significant age-by-group interactions for three major white matter tracts: superior longitudinal fasciculus and superior and inferior fronto-occipital fasciculus. These findings indicate altered developmental progression in these frontal-association tracts, with the FASD group notably showing greater reduction of MD between scans. ΔMD is shown to correlate with reading and receptive vocabulary in the FASD group, with steeper decreases of MD in the superior fronto-occipital fasciculus and superior longitudinal fasciculus between scans correlating with greater improvement in language scores. Volumetric analysis revealed reduced total brain, white, cortical gray, and deep gray matter volumes and fewer significant age-related volume increases in the FASD group, although age-by-group interactions were not significant. Longitudinal DTI indicates delayed white matter development during childhood and adolescence in FASD, which may underlie persistent or worsening behavioral and cognitive deficits during this critical period.

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ABSTRACT

Background: Individuals with Fetal Alcohol Spectrum Disorder (FASD) constitute a special population that may be at particularly high risk for substance use. The purpose of the current study was to estimate the utilization of specialized addiction treatment services (SATS) and the associated cost, as a part of the total cost of health care associated with FASD in Canada.

Methods: The current study was a modeling study. Data on SATS by lifetime mental disorder status were obtained from the Drug and Alcohol Treatment Information System (DATIS) in Ontario, Canada for 2010/11. The number of clients with FASD who received SATS in Ontario in 2010/11 was estimated, assuming that approximately 37% (confidence interval: 21.6%-54.5%) of individuals with FASD abuse or are addicted to alcohol and/or drugs and that their utilization rate of SATS is the same as those for people with a lifetime mental disorder. The data from DATIS was then extrapolated to the total Canadian population.

Results: The cost of SATS for clients with FASD in Canada in 2010/11 ranged from $1.65 million Canadian dollars (CND) to $3.59 million CND, based on 5,526 outpatient visits and 9,529 resident days. When the sensitivity analysis was performed the cost of SATS ranged from $979 thousand CND to $5.34 million CND.

Conclusions: Special attention must be paid to at-risk groups of individuals such as those with FASD, in order to reduce the likelihood of the development of co-morbid substance abuse problems, and thus, reducing the overall burden on Canadian society.


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were mated and exposed to either 10% (v/v) ethanol or water for the first 8 days of gestation (GD 0-8), and then offered water for the rest of gestation. Early developmental milestone achievement was assessed in offspring at postnatal days (P) 7, 14 and 21. Adult offspring underwent a comprehensive battery of behavioural tests to examine a range of behavioural domains including locomotion, exploration, anxiety, social behaviour, learned helplessness, sensorimotor gating, and nociception, as well as spatial memory in a water maze. Ethanol-exposed mice had similar postnatal developmental trajectories to water-exposed mice. However, the ethanol-exposed mice showed increased hyperlocomotion at P 14, 21 and 70 (p<0.05). Increased exploration and heightened motivation were also observed in adult mice. Furthermore, ethanol-exposed mice showed a significant improvement in memory in the water maze. The main findings were that mice had persistent and long lasting alterations in behaviour, including hyperactivity and enhanced spatial memory. These data suggest that even moderate dose ethanol exposure in early gestation has long term consequences on brain function and behaviour in mice.

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139. MONITORING POPULATION LEVELS OF ALCOHOL CONSUMPTION IN PREGNANT WOMEN: A CASE FOR USING BIOMARKERS
Shipton D, Tappin D, Sherwood R, Mactier H, Aitken D, Crossley J.
Paediatric Epidemiology and Community Health Unit, Department of Child Health, Division of Developmental Medicine, University of Glasgow, Glasgow, United Kingdom.
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ABSTRACT
A challenge to biochemically monitoring alcohol consumption in pregnancy is the prohibitive costs of collecting thousands of blood samples. This pilot study looks at the feasibility of using residual samples to monitor chronic and acute alcohol consumption in pregnancy. Residual anomalies screening samples (n = 150, 2006/7) were tested for carbohydrate-deficient transferrin (CDT, chronic marker) and ethyl glucuronide (EtG, acute marker). Valid readings were obtained for CDT but not EtG. These results pave the way for a larger representative study, to provide, for the first time, a national biochemical baseline estimate of chronic alcohol consumption in the pregnant population.

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140. SONIC HEDGEHOG EXPRESSION IS DISRUPTED FOLLOWING IN OVO ETHANOL EXPOSURE DURING EARLY CHICK EYE DEVELOPMENT
Brennan D, Giles S.
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ABSTRACT

The eye is particularly sensitive to ethanol's teratogenic effects. Our previous work, using a chick embryo model system, has shown that ethanol acts rapidly to perturb vital processes of early eye development producing defects of the lens and retina. Ethanol-induced disruption of the midline ventral telencephalon, a key site for expression of ocular morphogens such as sonic hedgehog (Shh), was further established. Consequently, in this study we have examined the effects of ethanol on the Shh pathway during the period of optic vesicle/optic cup formation. Chick embryos were injected in ovo with 125μL of a 20% ethanol solution directly into the yolk-sac at HH-stage 7, resulting in peak ethanol uptake of 0.294g/dL. Subsequent molecular analysis at 12, 24 and 48h post-treatment revealed that ethanol had no affect on Shh transcription, while, a significant reduction in the expression of the active signalling Shh protein was found. Surprisingly, none of the downstream Shh pathway members (Ptc, Gli1 and Gli3) were significantly altered by ethanol exposure. Overall, our results indicate that ethanol's disruption of Shh may be mediated through some alternative mechanism independent of the classical signalling pathway. However, the precise role of Shh in relation to ethanol teratogenicity continues to be debated. Thus, in conclusion, our findings are discussed in relation to the varied and often conflicting reports of ethanol-induced Shh perturbation found in the literature.


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141. A POTENTIAL MOLECULAR TARGET FOR MORPHOLOGICAL DEFECTS OF FETAL ALCOHOL SYNDROME: KIR2.1

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ABSTRACT

Fetal alcohol spectrum disorder (FASD) is a developmental disorder that affects up to 0.2% of births. FASD comprises severe cognitive and structural birth defects including cleft lip/palate, small jaw, wide-set eyes, dental abnormalities, digit abnormalities, small head, and short stature. Strict counseling guidelines stress abstaining from alcohol during pregnancy, but the prevalence of FASD persists. The lack of a convincing molecular target has hindered FASD research and treatment. Interestingly, mutations in an inwardly rectifying potassium channel, Kir2.1, cause a similar constellation of birth defects as in FASD. In other words, FASD phenocopies the traits conveyed by Kir2.1 mutations. Furthermore, alcohol directly binds to and modulates Kir2.1. Substantial evidence now suggests that alcohol targets Kir2.1 to cause the birth defects associated with FASD. This review compiles clinical, genetic, biochemical, electrophysiological, and molecular evidence that identifies Kir2.1 as a molecular target for FASD development and possibly therapeutic treatment.


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142. PRENATAL SUBSTANCE EXPOSURE: NEUROBIOLOGIC ORGANIZATION AT 1 MONTH

Conradt E, Sheinkopf SJ, Lester BM, Tronick E, LaGasse LL, Shankaran S, Bada H, Bauer
ABSTRACT

Objective: To examine the autonomic nervous system and neurobehavioral response to a sustained visual attention challenge in 1-month-old infants with prenatal substance exposure.

Study design: We measured heart rate, respiratory sinus arrhythmia, and neurobehavior during sustained visual orientation tasks included in the Neonatal Intensive Care Unit Network Neurobehavioral Scale in 1129 1-month-old infants with prenatal substance exposure. Four groups were compared: infants with prenatal cocaine and opiate exposure, infants with cocaine exposure, infants with opiate exposure, and infants with exposure to other substances (ie, alcohol, marijuana, and tobacco).

Results: The infants with prenatal exposure to both cocaine and opiates had the highest heart rates and lowest levels of respiratory sinus arrhythmia during a sustained visual attention challenge compared with the other 3 groups. Infants with prenatal cocaine and opiate exposure had poorer quality of movement and more hypertonicity during the Neonatal Intensive Care Unit Network Neurobehavioral Scale examination. They also had more nonoptimal reflexes and stress/abstinence signs compared with infants with prenatal exposure to cocaine only and those with prenatal exposure to alcohol, tobacco, and marijuana.

Conclusion: Problems with arousal regulation were identified in infants with prenatal substance exposure. Autonomic dysregulation has been implicated as a mechanism by which these difficulties occur. Our results suggest that infants with prenatal exposure to both cocaine and opiates have the greatest autonomic response to the challenge of a sustained visual attention task, possibly putting these infants at risk for problems associated with physiologic and behavioral regulation, a necessary prerequisite for early learning.


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143. EMBRYONIC CATALASE PROTECTS AGAINST ETHANOL-INITIATED DNA OXIDATION AND TERATOGENESIS IN ACATALASEMIC AND TRANSGENIC HUMAN CATALASE-EXPRESSING MICE

Miller L, Shapiro AM, Wells PG.
Department of Pharmacology and Toxicology, Faculty of Medicine, University of Toronto, Toronto, Ontario, Canada.

ABSTRACT

Reactive oxygen species (ROS) are implicated in fetal alcohol spectrum disorders (FASD) caused by alcohol (ethanol, EtOH). Although catalase detoxifies hydrogen peroxide, embryonic catalase activity is only about 5% of maternal levels. To determine the roles of ROS and embryonic catalase in FASD, pregnant mice with enhanced (expressing human catalase, hCat) or deficient (acatalasemic, aCat) catalase activity, or their respective wild-type (WT) controls, were treated ip on gestational day 9 with 4 or 6g/kg EtOH or its saline vehicle, and embryos and fetuses were, respectively, evaluated for oxidatively damaged DNA and structural anomalies. Untreated hCat and aCat dams had, respectively, more and less offspring than their WT controls. hCat progenies were protected from all EtOH fetal anomalies at the low dose (p < .01) and from reduced head diameter and resorptions at the high dose (p < .001). Conversely, aCat progenies were more sensitive to dose-dependent EtOH fetal
anomalies (p < .001) and exhibited a 50% increase in maternal lethality (p < .05) at the high dose. Maternal pretreatment of aCat mice with polyethylene glycol-conjugated catalase (PEG-Cat) reduced EtOH fetal anomalies (p < .001). EtOH-initiated embryonic DNA oxidation was reduced in hCat and WT mice pretreated with PEG-Cat and enhanced in aCat mice. Plasma concentrations of EtOH in catalase-altered mice were similar to controls, precluding a pharmacokinetic basis for altered EtOH teratogenesis. Endogenous embryonic catalase, despite its low level, is an important embryoprotective enzyme for EtOH teratogenesis and a likely determinant of individual risk.


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144. CONSUMPTION OF MEDICATIONS, ALCOHOL AND SMOKING IN PREGNANCY AND ASSESSMENT OF TERATOGENIC RISKS
Rocha RS, Bezerra SC, Lima JW, Costa Fda S. Universidade Estadual do Ceará (UECE), Fortaleza-CE, Brasil. bekinharocha@hotmail.com

ABSTRACT
Medications, alcohol and smoking can cause fetal damage. A cross-sectional study was conducted with 326 mothers of the Fortaleza General Hospital to evaluate the use of drugs, alcohol and smoking during pregnancy and its relation to teratogenic potential in different population characteristics, between 2006 and 2007. Postpartum women who had their babies in the research site were included and those whose babies were not admitted as hospital inpatients were excluded. Chi-square tests and t-tests were used in the analysis, with a p value <0.05 considered significant. 96.6% of the mothers took medications (2.8 drugs/pregnancy) and self-medication occurred in 11.3% of the cases. Single women took more drugs with high teratogenic potential (p=0.037). 11 cases of fetal malformation were observed, five of them were exposed to high teratogenic risks. Smoking occurred in 11.3% and alcohol use in 16%. Being single was found to be a risk factor for exposure to high teratogenic potential. Quality of prenatal care and other sociodemographic variables weren't related to exposure to teratogenic risks.


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145. ALCOHOL INTAKE IN LACTATING WOMEN ASSISTED IN A UNIVERSITY HOSPITAL
Nascimento AL, de Souza AF, de Amorim AC, Leitão MB, Maio R, Burgos MG. Departamento de Nutrição, Universidade Federal de Pernambuco (UFPE), Recife, PE, Brasil. nutrianaluisa@gmail.com

ABSTRACT
Objective: To determine the prevalence of alcohol intake and the degree of alcohol-related risk among nursing mothers attended at the Child Care Service of Hospital das Clínicas of Universidade Federal de Pernambuco, Brazil.

Methods: A cross-sectional study was carried out with 157 nursing mothers enrolled in the
Child Care Program of the university hospital. A questionnaire was administered addressing demographic and socioeconomic variables, type and duration of breastfeeding, smoking habits and consumption of foods considered as appetizers. The Alcohol Use Disorders Identification Test (AUDIT C) was applied for assessing alcohol consumption in the previous 12 months. Pearson's chi-square test and Fisher's exact test were used for statistical analysis.

**Results:** Twelve percent of the nursing mothers reported consuming alcoholic beverages, 100% of whom were classified as being at low risk for alcohol use disorders. The frequency of nursing mothers who consumed appetizers during alcohol consumption was 100%, the most common of which was cheese - 18 (95%).

**Conclusions:** The prevalence of alcohol intake was low in the nursing mothers analyzed. The users exhibited a low risk for alcohol disorders and a high frequency of the consumption of appetizers during alcohol consumption.


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146. FETAL ALCOHOL SPECTRUM DISORDERS AND FETAL ALCOHOL SYNDROME: THE STATE OF THE ART AND NEW DIAGNOSTIC TOOLS
Memo L, Gnoato E, Caminiti S, Pichini S, Tarani L.
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**ABSTRACT**
Ethanol consumption during pregnancy is a widespread problem which is increasing in the generation of young women. Gestational alcohol consumption causes fetal exposure to this teratogen and is associated with the onset of fetal alcohol spectrum disorders (FASD) including fetal alcohol syndrome (FAS). FASD and FAS can lead to several physical, cognitive and behavioral disabilities, whose early diagnosis is of primary importance to perform primary prevention with total abstinence from alcohol during pregnancy and secondary prevention in newborns and children for a proper follow up to reduce risk of secondary consequences. In recent years significant efforts have been made to understand the underlying mechanisms of this disease and to identify objective biological and instrumental diagnostic tools, such as exposure biomarkers in neonatal meconium and advanced magnetic resonance imaging. Nonetheless, further studies are still needed to implement our knowledge on fetal effects of ethanol, and multidisciplinary actions are necessary to raise awareness among women of childbearing age about the danger of consuming even small amounts of ethanol during pregnancy.


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147. MATERNAL FACTORS PREDICTING COGNITIVE AND BEHAVIORAL CHARACTERISTICS OF CHILDREN WITH FETAL ALCOHOL SPECTRUM DISORDERS

May, Philip A; Tabachnick, Barbara G; Gossage, J. Phillip; Kalberg, Wendy O; Marais, Anna-Susan; Robinson, Luther K; Manning, Melanie A; Blankenship, Jason; Buckley, David; Hoyme, H. Eugene; Adnams, Colleen M

ABSTRACT

Objective: To provide an analysis of multiple predictors of cognitive and behavioral traits for children with fetal alcohol spectrum disorders (FASDs).

Method: Multivariate correlation techniques were used with maternal and child data from epidemiologic studies in a community in South Africa. Data on 561 first-grade children with fetal alcohol syndrome (FAS), partial FAS (PFAS), and not FASD and their mothers were analyzed by grouping 19 maternal variables into categories (physical, demographic, childbearing, and drinking) and used in structural equation models (SEMs) to assess correlates of child intelligence (verbal and nonverbal) and behavior.

Results: A first SEM using only 7 maternal alcohol use variables to predict cognitive/behavioral traits was statistically significant (B = 3.10, p < .05) but explained only 17.3% of the variance. The second model incorporated multiple maternal variables and was statistically significant explaining 55.3% of the variance. Significantly correlated with low intelligence and problem behavior were demographic (B = 3.83, p < .05) (low maternal education, low socioeconomic status [SES], and rural residence) and maternal physical characteristics (B = 2.70, p < .05) (short stature, small head circumference, and low weight). Childbearing history and alcohol use composites were not statistically significant in the final complex model and were overpowered by SES and maternal physical traits.

Conclusions: Although other analytic techniques have amply demonstrated the negative effects of maternal drinking on intelligence and behavior, this highly controlled analysis of multiple maternal influences reveals that maternal demographics and physical traits make a significant enabling or disabling contribution to child functioning in FASD.


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148. DETECTION OF ILLICIT DRUGS IN URINE IN THE DIVISION OF NEONATOLOGY, HOSPITAL MOLAS IN LA PAMPA

Villarreal M, Ré S.
Servicio de Neonatología, Hospital Dr. Lucio Molas, Santa Rosa, La Pampa, Argentina. marvillarreal@cpenet.com.ar

ABSTRACT

There are few studies on the use of illicit drugs during pregnancy with a variable prevalence depending on the year, maternal age, region and diagnostic methods. Mothers’ and newborn infants’ urine samples were tested for illegal drugs in cases where the mother reported consumption, lack of antenatal care and neonatal signs and symptoms, from 2009 to 2011. A rapid strip test for simultaneous qualitative detection of multiple drugs and metabolites in urine
was used. In 19 out of 39 (49%) cases in which urine samples were collected, an illicit drug was detected in the mother and/or the newborn infant. Cocaine was the most frequently detected drug. There was a high coexistence of social and familiar risk factors, smoking (84%) and alcohol consumption (47%).

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149. IMPAIRED ODOR IDENTIFICATION IN CHILDREN WITH HISTORIES OF HEAVY PRENATAL ALCOHOL EXPOSURE
Bower E, Szajer J, Mattson SN, Riley EP, Murphy C.
San Diego State University/University of California, San Diego Joint Doctoral Program in Clinical Psychology, San Diego, CA 92120-4913, USA.

ABSTRACT
Prenatal alcohol exposure can lead to behavioral and cognitive impairments across multiple domains. Many of the brain regions impacted by prenatal alcohol exposure are also linked with olfactory processing, and odor identification deficits have been documented in certain neurological disorders associated with these brain regions. As odor identification following prenatal alcohol exposure is not well studied, we compared odor identification in children with prenatal exposure to alcohol (AE) to typically developing controls (CON) (N = 16/group). It was hypothesized that children in the AE group would perform more poorly than children in the CON group on the San Diego Odor Identification Test, an identification test of 8 common household odorants. Children exposed to alcohol during prenatal development were significantly impaired in olfactory identification (M = 5.95, SE = 0.37) compared to typically developing controls (M = 7.24, SE = 0.37). These findings confirmed the hypothesis that prenatal exposure to alcohol is associated with odor identification deficits, and suggest that further research is warranted to identify the mechanisms underlying these deficits, the integrity of brain areas that are involved, and to determine whether olfactory performance might contribute to better identification of children at risk for behavioral and cognitive deficits.

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150. NUANCED BUT SIGNIFICANT: HOW ETHANOL PERTURBS AVIAN CRANIAL NEURAL CREST CELL ACTIN CYTOSKELETON, MIGRATION AND PROLIFERATION
Oyedele OO, Kramer B.
School of Anatomical Sciences, Faculty of Health Sciences, University of the Witwatersrand Johannesburg, 7 York Road, Parktown 2193, South Africa. olusegun.oyedele@ubc.ca

ABSTRACT
Children with fetal alcohol syndrome (FAS) display striking craniofacial abnormalities. These features are proposed to result from perturbations in the morphology and function of cranial neural crest cells (cNCCs), which contribute significantly to the craniofacial complex. While certain pathways by which this may occur have been suggested, precise teratogenic
mechanisms remain intensely investigated, as does the question of the teratogenic dose. The present study focused on examining how avian cNCC actin cytoskeleton, migratory distance, and proliferation are affected ex vivo by exposure to ethanol concentrations that simulate maternal intoxication. Chick cNCCs were cultured in 0.2% and 0.4% v/v ethanol. Distances migrated by both ethanol-treated and control cells at 24 and 48 h were recorded. Following phalloidin immunocytochemistry, treated and control cNCCs were compared morphologically and quantitatively. Apoptosis and proliferation in control versus treated cNCCs were also studied. Chick cNCCs cultured in ethanol lost their spindle-like shapes and their ordered cytoskeleton. There was a significant stage-dependent effect on cNCC migration at 24 h (p = 0.035), which was greatest at stage 10 (HH). Ethanol treatment for 48 h revealed a significant main effect for ethanol, chiefly at the 0.4% level. There was also an interaction effect between ethanol dose and stage of development (stage 9 HH). Actin microfilament disruption was quantitatively increased by ethanol at the doses studied while cNCC proliferation was increased but not significantly. Ethanol had no effect on cNCC apoptosis. At ethanol levels likely to induce human FAS, avian cNCCs exhibit various subtle, potentially significant changes in morphology, migration, and proliferation, with possible consequences for fated structures.


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association between SGA and low-alcohol exposure but adds to evidence of a dose-response relationship with significant risks observed at binge drinking levels.

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152. ALCOHOL AND NMDA RECEPTOR: CURRENT RESEARCH AND FUTURE DIRECTION
Chandrasekar R.
Department of Biochemistry and Biotechnology Core Facility, Kansas State University Manhattan, KS, USA.

ABSTRACT
The brain is one of the major targets of alcohol actions. Most of the excitatory synaptic transmission in the central nervous system is mediated by N-methyl-D-aspartate (NMDA) receptors. However, one of the most devastating effects of alcohol leads to brain shrinkage, loss of nerve cells at specific regions through a mechanism involving excitotoxicity, oxidative stress. Earlier studies have indicated that chronic exposure to ethanol both in vivo and in vitro, increases NR1 and NR2B gene expression and their polypeptide levels. The effect of alcohol and molecular changes on the regulatory process, which modulates NMDAR functions including factors altering transcription, translation, post-translational modifications, and protein expression, as well as those influencing their interactions with different regulatory proteins (downstream effectors) are incessantly increasing at the cellular level. Further, I discuss the various genetically altered mice approaches that have been used to study NMDA receptor subunits and their functional implication. In a recent countable review, epigenetic dimension (i.e., histone modification-induced chromatin remodeling and DNA methylation, in the process of alcohol related neuroadaptation) is one of the key molecular mechanisms in alcohol mediated NMDAR alteration. Here, I provide a recount on what has already been achieved, current trends and how the future research/studies of the NMDA receptor might lead to even greater engagement with many possible new insights into the neurobiology and treatment of alcoholism.

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PLOS One, Published: May 28, 2013DOI: 10.1371/journal.pone.0063794

153. ETHANOL DIVERTS EARLY NEURONAL DIFFERENTIATION TRAJECTORY OF EMBRYONIC STEM CELLS BY DISRUPTING THE BALANCE OF LINEAGE SPECIFIERS
Rosa Sánchez-Alvarez, Saurabh Gayen, Rajanikanth Vadigepalli, Helen Anni

ABSTRACT
Background: Ethanol is a toxin responsible for the neurodevelopmental deficits of Fetal Alcohol Spectrum Disorders (FASD). Recent evidence suggests that ethanol modulates the protein expression of lineage specifier transcription factors Oct4 (Pou5f1) and Sox2 in early stages of mouse embryonic stem (ES) cell differentiation. We hypothesized that ethanol induced an imbalance in the expression of Oct4 and Sox2 in early differentiation, that dysregulated the expression of associated and target genes and signaling molecules and
diverted cells from neuroectodermal (NE) formation.

**Methodology/Principal Findings:** We showed modulation by ethanol of 33 genes during ES cell differentiation, using high throughput microfluidic dynamic array chips measuring 2,304 real time quantitative PCR assays. Based on the overall gene expression dynamics, ethanol drove cells along a differentiation trajectory away from NE fate.

These ethanol-induced gene expression changes were observed as early as within 2 days of differentiation, and were independent of cell proliferation or apoptosis. Gene expression changes were correlated with fewer βIII-tubulin positive cells of an immature neural progenitor phenotype, as well as a disrupted actin cytoskeleton were observed. Moreover, Tuba1a and Gapdh housekeeping genes were modulated by ethanol during differentiation and were replaced by a set of ribosomal genes with stable expression.

**Conclusions/Significance:** These findings provided an ethanol-response gene signature and pointed to the transcriptional dynamics underlying lineage imbalance that may be relevant to FASD phenotype.


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**154. TOBACCO AND ALCOHOL DURING PREGNANCY: PREVALENCE AND DETERMINANTS IN GENEVA IN 2008**

Dupraz J, Graff V, Barasche J, Etter JF, Boulvain M.

University of Geneva, Faculty of Medicine, Switzerland. [djuls@bluewin.ch](mailto:djuls@bluewin.ch)

**ABSTRACT**

**Objectives:** To describe alcohol and tobacco consumption during pregnancy in women giving birth in a public hospital in Geneva, to evaluate risk factors related to these consumptions and to explore the influence of close relatives on the consumption habits of pregnant women.

**Methods:** A cross-sectional questionnaire survey after delivery in 207 women in the maternity ward of the Geneva University Hospitals in 2008. We used retrospective self-reports of smoking during pregnancy (including temporary smoking), smoking during the entire pregnancy and alcohol drinking during pregnancy (even a single glass).

**Results:** The proportion of smokers decreased from 31.2% before pregnancy to 21.7% during pregnancy (temporary smoking included), and 9.2% of women smoked continuously until delivery. Major factors associated with tobacco use were living alone, living with a smoker and tobacco consumption of the husband/partner in the presence of the pregnant woman. Regarding alcohol consumption, 62.7% of the participants reported drinking (even occasionally) before pregnancy, and 36.3% of the women drank at least one glass of alcohol during pregnancy. The alcohol consumption of the husband/partner and invitations to drink from other people were associated with alcohol consumption during pregnancy.

**Conclusions:** Among women delivering in a public hospital, tobacco and alcohol consumption during pregnancy was important and significantly influenced by the habits and attitude of close relatives. The involvement of relatives in health promotion interventions should be addressed.


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155. MATERNAL ALCOHOL USE INCREASES RISK OF INFANT MORTALITY
Burd L.
University of North Dakota, Grand Forks, North Dakota, USA.

ABSTRACT
No Abstract Available.

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156. EMODIN PREVENTS ETHANOL-INDUCED DEVELOPMENTAL ANOMALIES IN CULTURED MOUSE FETUS THROUGH MULTIPLE ACTIVITIES
Yon JM, Lin C, Oh KW, Baek HS, Lee BJ, Yun YW, Nam SY.
College of Veterinary Medicine and Research Institute of Veterinary Medicine, Chungbuk National University, Cheongju, Korea.

ABSTRACT
Background: Maternal alcohol ingestion on pregnant period causes fetal alcohol syndrome including psychological and behavioral problems, and developmental abnormality. In this study, we investigated the effect of emodin, an active anthraquinone component found in the roots and bark of the genus Rhamnus (Buckthorn), on ethanol-induced teratogenesis during embryonic organogenesis.

Methods: We cultured mouse embryos on embryonic day 8.5 for 2 days with ethanol (5 μl/3 ml) and/or emodin (1×10(-5) and 1×10(-4) μg/ml) using a whole embryo culture system and then investigated the developmental evaluation, superoxide dismutase (SOD) activity, and expression patterns of cytoplasmic SOD (SOD1), mitochondrial SOD (SOD2), cytosolic glutathione peroxidase (cGPx), tumor necrosis factor-α (TNF-α), caspase 3, and hypoxia inducible factor 1α (HIF-1α).

Results: Morphological parameters, including growth in yolk sac and fetal head, body length, and development of the central nervous system, circulation system, sensory organs, skeletal system, and limbs in embryos exposed to ethanol were significantly decreased compared to those of the normal control group, but co-treatment with emodin (1 × 10(-5) and 1 × 10(-4) μg/ml) significantly improved these parameters. Furthermore, the reduced levels of SOD activity, and SOD1, SOD2, cGPx, and HIF-1α and the increased gene levels of TNF-α and caspase-3 due to ethanol exposure were significantly restored by cotreatment with emodin.

Conclusions: This study revealed that cotreatment with emodin significantly prevented teratogenesis induced by ethanol, not only by modulating hypoxia and antioxidant enzymes, but also by attenuating the enhanced levels of TNF-α and caspase 3 in cultured embryos. Therefore, emodin may be an effective preventive agent for ethanol-induced teratogenesis.

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157. ETHANOL ALTERS PROLIFERATION AND DIFFERENTIATION OF NORMAL AND CHROMOSOMALLY ABNORMAL HUMAN EMBRYONIC STEM CELL-DERIVED NEUROSPHERAES
Malini Krishnamoorthy1,10,†, Brian A. Gerwe7,9,†, Christopher D. Scharer2, Vanita Sahasranaman6, Carmen D. Eilertson5, Rachel J. Nash6,10, Sümayra Naz Usta10, Shasmin Kelly5, Matthew Rose5, Rene Peraza5, Jagan Arumugham10, Bethany Stewart1, Steven L. Stice6, Rodney J. Nash1,3,5,10,*
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ABSTRACT
Ethanol is a powerful substance and, when consumed during pregnancy, has significant psychoactive and developmental effects on the developing fetus. These abnormalities include growth retardation, neurological deficits, and behavioral and cognitive deficiencies, commonly referred to as fetal alcohol spectrum disorder. The effect of ethanol has been reported to affect cellular development on the embryonic level, however, not much is known about mutations contributing to the influence of ethanol. The purpose of our study was to determine if mutation contribute to changes in differentiation patterning, cell-cycle regulatory gene expression, and DNA methylation in human embryonic stem cells after ethanol exposure. We exposed human embryonic stem cells (with and without know DNA mutations) to a low concentration (20 mM) of ethanol and measured neurosphere proliferation and differentiation, glial protein levels, expression of various cell-cycle genes, and DNA methylation. Ethanol altered cell-cycle gene expression between the two cell lines; however, gene methylation was not affected in ether lines.

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158. PRENATAL ALCOHOL EXPOSURE ALTERS THE CEREBRAL CORTEX PROTEOME IN WEANLING RATS
Canales L, Gambrell C, Chen J, Neal RE.
Department of Environmental and Occupational Health Sciences, School of Public Health and Information Sciences, University of Louisville, Louisville, KY 40292, USA.

ABSTRACT
Maternal consumption of alcohol during pregnancy impairs neurodevelopment in offspring. Utilizing a rodent model of continuous moderate dose alcohol exposure throughout gestation [gestation day 1 (GD1)-GD22; BAC ~70 mg/dL], the impact of developmental alcohol exposure on juvenile cerebral cortex protein abundances was determined. At weaning, cerebral cortex tissue was collected from pups for 2D SDS-PAGE based proteome analysis with statistical analysis by Partial Least Squares-Discriminant Analysis (PLS-DA). Gestational alcohol exposure increased the abundance of post-translationally modified forms of cytoskeletal proteins and the abundance of proteins within the small molecule biochemistry (includes glucose metabolism) pathway and proteosome processing pathways though ubiquitin conjugating enzymes and chaperones were decreased in abundance. In weanling offspring exposed prenatally to alcohol, alterations in cytoskeletal protein post-translational modifications were noted. Increased abundance of proteins from the small molecule biochemistry pathway, which includes glucose metabolism, and proteosome processing pathways were also noted. Decreased abundances of ubiquitin conjugating enzyme and chaperone protein were noted in the cerebral cortex of these offspring.

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159. RISKY SUBSTANCE EXPOSURE DURING PREGNANCY: A PILOT STUDY FROM LEBANESE MOTHERS
Rachidi S, Awada S, Al-Hajje A, Bawab W, Zein S, Saleh N, Salameh P.
Laboratory of Epidemiological and Clinical Research, Lebanese University, Beirut, Lebanon.

ABSTRACT
Background: The harmful effects of medication and licit substance use during pregnancy may potentially constitute a major public health concern. Our study aims to assess risky exposure of Lebanese pregnant women to drugs, tobacco, caffeine, and alcohol, and to determine their effect on postnatal outcomes.

Methods: Women at term were addressed after delivery in five university hospitals of Beirut and Mount Lebanon between February and June 2012. A standardized questionnaire was administered to them. Moreover, medical files of both mothers and their respective newborns were checked to confirm information given by mothers, and to assess the health outcome of the babies.

Results: Among the interviewed 350 women, active and passive smoking of tobacco (cigarette or water pipe), and consumption of category C, D, and X drugs were common during pregnancy in Lebanon; they were shown to negatively affect the neonatal outcome in multivariate analyses: they significantly decreased Apgar scores and increased the risk of underweight and medical complications of babies (P < 0.05).

Conclusion: Our study demonstrated that Lebanese women were exposed during pregnancy to multiple medications and licit substances that affected the neonates’ health. Our findings
have implications for clinical obstetric practice and prevention programs in Lebanon. Efforts should be made to decrease exposure to harmful substances during pregnancy.

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160. IS MILD-MODERATE DRINKING IN PREGNANCY HARMLESS? NEW EXPERIMENTAL EVIDENCE TO THE OPPOSITE
Justin Chan, Gideon Koren

ABSTRACT
During the last decade a growing number of studies have failed to detect adverse neurodevelopmental effects of mild –to moderate maternal drinking in the exposed child, supporting a climate that “some drinking in pregnancy is OK”. A recent experimental study in sheep, mimicking conditions of moderate drinking in the third trimester of pregnancy, provides powerful evidence that there are serious lifelong risks to fetal exposure to alcohol. These should serve as an alarm call to those who legitimize mild-moderate maternal drinking based on incomplete data

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161. ADDICTIONS AND PREGNANCY : HOW TO RUIN A PREGNANCY
Emonts P, Masson V, Chantraine F, Kridelka F, Nisolle M. Universite de Liege, Chef de Clinique, Service de Gynecologie-Obstetrique, CHU de Liege, site NDB et CHR Citadelle, Belgique. patrick.emonts@chu.ulg.ac.be

ABSTRACT
Pregnant women are well aware that any addiction during pregnancy can be harmful to the child. In spite of this knowledge, many continue to smoke, to drink alcohol, to consume illicit drugs or to absorb medicines because these dependences are particularly strong. Tobacco, alcohol, cocaine and ecstasy represent the most dangerous substances as regards foetal damage. The period of pregnancy is the optimal moment to stop these addictions. It is therefore essential to raise awareness among the general public, policy makers, and physicians of the fact that addictions during pregnancy cause a disparity in terms of future health and life expectancy of the unborn child.

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162. OPTIC NERVE HYPOPLASIA
Kaur S, Jain S, Sodhi HB, Rastogi A, Kamlesh.
Department of Ophthalmology, Guru Nanak Eye Center, New Delhi, India.

ABSTRACT
Optic nerve hypoplasia (ONH) is a congenital anomaly of the optic disc that might result in moderate to severe vision loss in children. With a vast number of cases now being reported, the rarity of ONH is obviously now refuted. The major aspects of ophthalmic evaluation of an infant with possible ONH are visual assessment, fundus examination, and visual electrophysiology. Characteristically, the disc is small, there is a peripapillary double-ring sign, vascular tortuosity, and thinning of the nerve fiber layer. A patient with ONH should be assessed for presence of neurologic, radiologic, and endocrine associations. There may be maternal associations like premature births, fetal alcohol syndrome, maternal diabetes. Systemic associations in the child include endocrine abnormalities, developmental delay, cerebral palsy, and seizures. Besides the hypoplastic optic nerve and chiasm, neuroimaging shows abnormalities in ventricles or white- or gray-matter development, septo-optic dysplasia, hydrocephalus, and corpus callosum abnormalities. There is a greater incidence of clinical neurologic abnormalities in patients with bilateral ONH (65%) than patients with unilateral ONH. We present a review on the available literature on the same to urge caution in our clinical practice when dealing with patients with ONH. Fundus photography, ocular coherence tomography, visual field testing, color vision evaluation, neuroimaging, endocrinology consultation with or without genetic testing are helpful in the diagnosis and management of ONH.

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163. LONG-LASTING NEURAL CIRCUIT DYSFUNCTION FOLLOWING DEVELOPMENTAL ETHANOL EXPOSURE
Sadrian B, Wilson DA, Saito M.
Department of Child and Adolescent Psychiatry, New York University Langone School of Medicine, One Park Avenue, Eighth Floor, New York, NY 10128, USA; Nathan Kline Institute, 140 Old Orangeburg Road, Orangeburg, NY 10962, USA.

ABSTRACT
Fetal Alcohol Spectrum Disorder (FASD) is a general diagnosis for those exhibiting long-lasting neurobehavioral and cognitive deficiencies as a result of fetal alcohol exposure. It is among the most common causes of mental deficits today. Those impacted are left to rely on advances in our understanding of the nature of early alcohol-induced disorders toward human therapies. Research findings over the last decade have developed a model where ethanol-induced neurodegeneration impacts early neural circuit development, thereby perpetuating subsequent integration and plasticity in vulnerable brain regions. Here we review our current knowledge of FASD neuropathology based on discoveries of long-lasting neurophysiological effects of acute developmental ethanol exposure in animal models. We discuss the important balance between synaptic excitation and inhibition in normal neural network function, and relate the significance of that balance to human FASD as well as related disease states. Finally, we postulate that excitation/inhibition imbalance caused by early ethanol-induced neurodegeneration results in perturbed local and regional network signaling and therefore neurobehavioral pathology.

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164. ASSOCIATIONS BETWEEN MULTIVITAMIN SUPPLEMENT USE AND ALCOHOL CONSUMPTION BEFORE PREGNANCY: PREGNANCY RISK ASSESSMENT MONITORING SYSTEM, 2004 TO 2008

Weiss LA, Chambers CD.
Division of Dysmorphology and Teratology, Department of Pediatrics, University of California, San Diego, La Jolla, California.

ABSTRACT
Background: Approximately 50 to 70% of childbearing-aged women consume alcohol and up to 23% of pregnancies have some level of prenatal alcohol exposure.

Methods: Using data from the Pregnancy Risk Assessment Monitoring System from 2004 to 2008, 111,644 women who completed questions relating to periconceptional alcohol use and multivitamin supplement use were included in the study. This study explored associations between periconceptional alcohol use and multivitamin supplementation use. Weighted multivariable logistic regression was used to explore associations, adjusting for maternal education, maternal ethnicity, maternal age, household income, and parity.

Results: During the periconceptional period, a dose-dependent association was found where women who consumed alcohol (≤3 drinks/wk, odds ratio [OR] = 0.76; 4 to 6 drinks/wk, OR = 0.60; 7 to 13 drinks/wk, OR = 0.49; ≥14 drinks/wk, OR = 0.39) and binged on alcohol (1 time, OR = 0.76; 2 to 3 times, OR = 0.66; 4 to 5 times, OR = 0.56; ≥6 times, OR = 0.50) were significantly less likely to take a multivitamin supplement compared with those that did not consume alcohol.

Conclusions: These findings emphasize the importance of periconceptional multivitamin supplement use, especially among alcohol-consuming women of childbearing age.


165. LONG-LASTING ALTERATIONS TO DNA METHYLATION AND NCRNAS COULD UNDERLIE THE EFFECTS OF FETAL ALCOHOL EXPOSURE IN MICE

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ABSTRACT
Fetal alcohol spectrum disorders (FASDs) are characterized by life-long changes in gene
expression, neurodevelopment and behavior. What mechanisms initiate and maintain these changes are not known, but current research suggests a role for alcohol-induced epigenetic changes. In this study we assessed alterations to adult mouse brain tissue by assaying DNA cytosine methylation and small non-coding RNA (ncRNA) expression, specifically the microRNA (miRNA) and small nucleolar RNA (snoRNA) subtypes. We found long-lasting alterations in DNA methylation as a result of fetal alcohol exposure, specifically in the imprinted regions of the genome harboring ncRNAs and sequences interacting with regulatory proteins. A large number of major nodes from the identified networks, such as Pten signaling, contained transcriptional repressor CTCF-binding sites in their promoters, illustrating the functional consequences of alcohol-induced changes to DNA methylation. Next, we assessed ncRNA expression using two independent array platforms and quantitative PCR. The results identified 34 genes that are targeted by the deregulated miRNAs. Of these, four (Pten, Nmnat1, Slitrk2 and Otx2) were viewed as being crucial in the context of FASDs given their roles in the brain. Furthermore, ~20% of the altered ncRNAs mapped to three imprinted regions (Snrpn-Ube3a, Dlk1-Dio3 and Sfmbt2) that showed differential methylation and have been previously implicated in neurodevelopmental disorders. The findings of this study help to expand on the mechanisms behind the long-lasting changes in the brain transcriptome of FASD individuals. The observed changes could contribute to the initiation and maintenance of the long-lasting effect of alcohol.

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166. IN VITRO FERTILIZATION OUTCOMES AND ALCOHOL CONSUMPTION IN AT-RISK DRINKERS: THE EFFECTS OF A RANDOMIZED INTERVENTION
Rossi BV, Chang G, Berry KF, Hornstein MD, Missmer SA.
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ABSTRACT
Background and objectives: Women’s use of alcohol in pregnancy is associated with an increased risk of fetal loss and birth defects. Also, alcohol use in women decreases the success of infertility treatment, such as in vitro fertilization (IVF). Our goal was to determine if there were differences in IVF outcomes and alcohol use parameters among at-risk drinkers randomized to a brief intervention (BI) versus assessment only (AO).

Methods: We conducted a randomized controlled trial to determine the effect of BI or AO among at-risk drinkers on IVF. We studied 37 women (AO = 21; BI = 16).

Results: While the BI group had a significantly greater decrease in the number of drinks/drinking day compared to the AO group (p = .04), there were no differences in the likelihood of implantation failure, chemical pregnancy, spontaneous abortion, preterm birth, or live birth.

Conclusions: BI and AO contributed to a decrease in alcohol use and did not demonstrate differences in IVF outcomes. A larger study may confirm these preliminary findings.

Scientific significance: Our results will assist care providers in treating alcohol use in pregnancy in an effective way, such that IVF cycles and the chance of pregnancy are optimized.

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167. SCREENING FOR SUBSTANCE ABUSE IN PREGNANCY
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sdj4924@aol.com

ABSTRACT
Several states have proposed laws that urine drug screening be performed as a part of qualifying for public assistance. At least one state (Florida) has passed such a law, and several other states are considering similar laws. The Oklahoma Commission on Children and Youth created a committee to study laws and policies regarding the use of illegal drugs while pregnant. To get a better understanding of drug screening and pregnancy, 151 consecutive obstetrical patients receiving Medicaid were screened at their initial obstetrical visit by verbal and written questionnaire’s concerning the use of alcohol, nicotine, and other illicit/dangerous drugs; in addition a urine drug screen for the use of illicit or dangerous drugs was performed. The patient histories regarding the use of dangerous or illicit substances was reviewed and compared with the urine drug screens performed at the same visit. The authors note that when studied the incidence of substance abuse has been similar in patient population receiving public assistance and patient populations with traditional insurance. Oklahoma is one of 13 states with laws requiring mandatory reporting of substance abuse in pregnancy or the exposure of the newborn to illicit substances.

168. THE IMPACT OF RAISING A CHILD WITH FASD UPON CARERS: FINDINGS FROM A MIXED METHODOLOGY STUDY IN THE UK
Raja Mukherjee, Elizabeth Wray
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Matthew Commers, Maastricht University, the Netherlands
Sheila Hollins, St George’s University of London, UK
Leopold Curfs, Maastricht University, the Netherlands

ABSTRACT
Research suggests that caring for a child with Fetal Alcohol Spectrum Disorders (FASD) creates unique challenges for carers. To investigate this, three focus groups and education sessions, attended by 66 people, were held in the UK. Knowledge about FASD and its impact on families was evaluated using the focus groups, the Parental Stress Index and knowledge questionnaire. Eight broad themes were identified from thematic analysis of the focus groups. The findings suggest more support is needed for carers of children with FASD, especially as carers grow older. The implication for current practice should be further evaluated in this group.

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169. BURDEN OF FETAL ALCOHOL SYNDROME IN A RURAL WEST COAST AREA OF SOUTH AFRICA
Olivier L, Urban M, Chersich M, Temmerman M, Viljoen D.
Foundation for Alcohol-Related Research, Cape Town, South Africa. urban@sun.ac.za

ABSTRACT
Background: Fetal alcohol syndrome (FAS) is common in parts of South Africa; rural residence is a frequently cited risk factor. We conducted a FAS school prevalence survey of an isolated rural community in a West Coast village of Western Cape Province, so obtaining the first directly measured rate, focusing specifically on a South African rural area, of FAS and partial FAS (PFAS).

Methods: The study area (Aurora village), a community of about 2 500 people in a grain-producing region, has one primary school. All learners were eligible for study inclusion. Initial anthropometry screening was followed by a diagnostic stage entailing examination by a dysmorphologist for features of FAS, neurodevelopmental assessment, and an interview assessing maternal alcohol consumption.

Results: Of 160 learners screened, 78 (49%) were screen-positive, of whom 63 (81%) were clinically assessed for FAS. The overall FAS/PFAS rate among the screened learners was 17.5% (95% confidence interval 12.0 - 24.2%), with 16 (10.0%) children having FAS and 12 (7.5%) PFAS. High rates of stunting, underweight and microcephaly were noted in all learners, especially those with FAS or PFAS. Five (18%) mothers of affected children were deceased by the time of assessment.

Conclusion: We describe very high rates of FAS/PFAS in an isolated rural part of the Western Cape that is not located in a viticultural region. Our study suggests that the prevalence of FAS may be very high in isolated communities, or in particular hot-spots. It adds to the growing evidence that FAS/PFAS is a significant, and underestimated, health problem in South Africa. Expanded screening and surveillance programmes, and preventive interventions, are urgently needed.

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170. OLFACTORY PREFERENCE FOR ETHANOL FOLLOWING SOCIAL INTERACTION WITH AN INTOXICATED PEER IN ADOLESCENT RATS EXPOSED TO ETHANOL IN-UTERO
Samanta M. March, Ricardo M. Pautassi, Michael Nizhnikov, Juan Fernández-Vidal, Norman E. Spear and Juan C. Molina

ABSTRACT
Background: Prenatal exposure to ethanol and later socially mediated exposure predicts ethanol intake in human adolescents. Animal rat models indicate that brief interactions with an ethanol-intoxicated peer result in heightened preference for ethanol odor and ethanol intake.

Methods: This study assessed preference for ethanol odor in adolescent male rats (observers) following social interaction with an ethanol intoxicated peer (demonstrators) as a function of prenatal ethanol exposure (gestational days 17-20, 1.0 g/kg, intragastric). Social behavior and locomotion during social interaction was also measured.
Results: Social investigation was greater in observers that interacted with an intoxicated demonstrator in comparison to those that interacted with a sober peer. Social contact increased when the demonstrator was under the effects of ethanol, but only if the observer had experienced ethanol prenatally. Ethanol inhibited locomotion in the demonstrators. Finally, social interaction with an intoxicated peer during adolescence as well as prenatal ethanol experience increased preference for ethanol odor.

Conclusions: Fetal exposure to ethanol mediated by maternal intoxication at late gestation or by interaction with an intoxicated peer at adolescence heightens preference for the chemosensory cues of the drug.

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172. BOUND BY THE CLOCK: THE EXPERIENCES OF YOUTH WITH FASD TRANSITIONING TO ADULTHOOD FROM CHILD WELFARE CARE
Burnside, Linda; Fuchs, Don

ABSTRACT
Fetal Alcohol Spectrum Disorder (FASD) is a condition that affects a significant proportion of children and youth in the care of child welfare agencies in Canada. Few studies have heard from the voices of youth with FASD themselves as they are leaving care. This article describes a qualitative study that focuses on the lived experiences of 20 youth with FASD in Manitoba as they were preparing for the transition from child welfare care to adulthood (or had recently emancipated from the system). The experiences and insights of these youth highlight the supports and services required by youth with FASD transitioning out of care, from both the child welfare system and from services for adults with FASD.


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173. MORPHOLOGICAL CHARACTERISTICS OF SPERMATOGENESIS IN THE OFFSPRING OF FEMALE RATS WITH CHRONIC ALCOHOL INTOXICATION
Sizonenko ML, Briukhin GV, La'skov DS.

ABSTRACT
Using general histological and morphometric methods, the peculiarities of spermatogenic epithelium were studied in the offspring of female rats with chronic alcohol intoxication, which was created before the onset of pregnancy by substitution of the drinking water by 15% solution of ethyl alcohol for the period of 3 months. Total number of animals was equal to 62 rat pups which were studied at postnatal days 15, 30 and 45, including 32 rats of the intact group (10 litters) and 30 pups of the experimental group (8 litters). It was found that in the offspring of female rats with chronic alcohol intoxication, the inhibition of the processes of spermatogenesis took place, as reflected by the reduction in the area of the convoluted seminiferous tubules (CST), decrease in the number of spermatogenic cells of the seminiferous layer, increase in the proportion of CST with desquamated epithelium and giant spermatogenic cells, as well as by the reduction of spermatogenic index, which reflects the average number of layers of spermatogenic cells in each CST.


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174. CHOICES: AN INTEGRATED BEHAVIORAL INTERVENTION TO PREVENT ALCOHOL-EXPOSED PREGNANCIES AMONG HIGH-RISK WOMEN IN COMMUNITY SETTINGS
Velasquez MM, von Sternberg K, Parrish DE.
ABSTRACT
CHOICES is an integrated behavioral intervention for prevention of prenatal alcohol exposure in women at high risk for alcohol-exposed pregnancies. The intervention uses motivational interviewing and cognitive-behavioral strategies, and targets adoption of effective contraception and reduction of alcohol use. The CHOICES intervention includes four manual-guided counseling sessions delivered by behavioral health counselors and one contraceptive session with a family planning clinician. CHOICES's efficacy has been established through a series of randomized controlled trials in settings including primary care, university hospital-based obstetrical/gynecology practices, urban jails, substance abuse treatment settings, and a media-recruited sample in three large cities. This article describes the CHOICES line of research including the epidemiology, feasibility, and efficacy studies. It also details the CHOICES intervention and the components of each session. In addition, the authors describe current studies testing modifications of the CHOICES intervention, the dissemination efforts to date, and implications for social work practice.


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176. GENETIC INSTRUMENTAL VARIABLE STUDIES OF EFFECTS OF PRENATAL RISK FACTORS
Wehby GL, Scholder Sv.
Department of Health Management and Policy, University of Iowa, Iowa City, IA, USA.
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ABSTRACT
Identifying the effects of maternal risk factors during pregnancy on infant and child health is an area of tremendous research interest. However, policymakers are primarily interested in unraveling the causal effects of prenatal risk factors, not their associations with child health, which may be confounded by several unobserved factors. In this article, we evaluate the utility of genetic variants in three genes that have unequivocal evidence of being related to three major risk factors—CHRNA3 for smoking, ADH1B for alcohol use, and FTO for obesity—as instrumental variables for identifying the causal effects of such factors during pregnancy. Using two independent datasets, we find that these variants are overall predictive of the risk factors and are not systematically related to observed confounders, suggesting that they may be useful instruments. We also find some suggestive evidence that genetic effects are stronger during than before pregnancy. We provide an empirical example illustrating the use of these genetic variants as instruments to evaluate the effects of risk factors on birth weight. Finally, we offer suggestions for researchers contemplating the use of these variants as instruments.

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forces influence the actions of professionals, and what barriers may exist in FASD-related practice. It aims to provide a nuanced analysis of how FASD is currently handled, and suggests potential strategies for achieving more effective service provision for FASD.

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178. ESTIMATION OF ALCOHOL CONTENT OF WINE, BEER AND SPIRITS TO EVALUATE EXPOSURE RISK: PILOT STUDY USING A QUESTIONNAIRE AND POURING TASK IN ENGLAND
Mukherjee RAS, Wray E, Curfs L, Hollins S

ABSTRACT
Aims: Research has shown different results regarding safe consumption levels of alcohol in pregnancy. We argued in 2005 that an individual’s inability to accurately predict their alcohol consumption may be one factor influencing risk. In order to re-evaluate within the England, this study sought to assess current knowledge of the public and healthcare practitioners.

Design: Both alcohol knowledge questionnaires and pouring tasks were conducted using standardised ethical committee-approved methods.

Settings: Different sites across the England including, Surrey, London, Oxford and Wigan where FASD support groups in England are based.

Participants: Health professionals and general public self selecting in response to advertisement.

Measurements: Frequency data and categorical data was collected and analysed using SPSS version 18

Findings: In total 1265 questionnaires were completed (688 public and 577 professionals). 140 people completed the pouring task. People’s ability to calculate accurately from strength and volume was within 20% of the accurate figure for units, although with a wide range.

Conclusions: These findings support the hypothesis that when asked to pour their own drinks, individuals are poor at estimating the alcohol content of that same drink. This has implications for public health strategies. Glass size and the strength of alcohol concentration have different implications in different countries. For those drinking in pregnancy however, the message that no exposure is no risk remains true.

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1. FROM TRAINING TO IMPLEMENTATION: ONTARIO YOUTH PROBATION OFFICERS’ USE OF THE ASANTE CENTRE FASD SCREENING AND REFERRAL TOOL
Burns SM¹, Bloom, HM², Stibbards A²
¹ Community Leadership in Justice Fellow, Law Foundation of Ontario & Georgian College of Applied Arts and Technology; ² Georgian College of Applied Arts and Technology

ABSTRACT
The research was designed to explore the impact and value of in-service training on FASD and the Asante Centre Youth Probation Officer FASD Screening and Referral Tool on youth probation officer practice in Ontario. It was hypothesized that training will improve FASD awareness, and enhance confidence, knowledge and response when developing case management, plans of care, probation orders and/or when recommending assessments. The research consisted of a pre/post-test self-administered questionnaire, a qualitative guided interview, and a follow-up survey four months after the 1-day/5-hour in-service training. Youth probation officers and managers from one regional office were invited to participate in the training. Of 23 participants, 19 consented to participate in the research including 17 youth probation officers and two managers; 13 completed all research components (n=13). Comparison data and statistical analysis provides evidence that training enhances probation officers’ confidence to describe and implement program modifications for youth who have or are suspected of having FASD. Training enhances knowledge of FASD, FASD profile identification, recognition of the assessment and diagnostic referral pathway, and confidence to make referrals. Staff efficacy in case management and plans of care improves but not probation orders. All participants indicated value in screening for FASD to improve client outcomes but identified internal and systemic barriers to implementation. They identified opportunities throughout the justice process to use or integrate a FASD screening and referral tool. Ten participants identified the combination of FASD training along with a screening tool as having the greatest impact on their practice.

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2. FATTY ACID ETHYL ESTERS IN MECONIUM OF FETAL SHEEP EXPOSED TO ETHANOL IN LATE GESTATION
Zelner I¹,², Kenna K³, Brien J⁴, Bocking A⁵, Harding R⁶, Walker D³, Koren G¹,²
¹ Div. of Clinical Pharmacology & Toxicology, The Hospital for Sick Children, Toronto, ON; ² Dept. of Pharmacology and Toxicology, University of Toronto, Toronto, ON; ³ Dept. of Physiology, Monash University, Clayton, VIC, Australia; ⁴ Dept. of Pharmacology and Toxicology, Queen’s University, Kingston, ON; ⁵ Dept. of Obstetrics and Gynaecology, University of Toronto, Toronto, ON; ⁶ Dept. of Anatomy and Developmental Biology, Monash University, Clayton, VIC, Australia
ABSTRACT

Introduction: Meconium fatty acid ethyl esters (FAEE) are established biomarkers of heavy fetal ethanol exposure. However, their utility in identifying fetal organ injury resulting from relatively moderate doses of in-utero ethanol exposure has not yet been investigated.

Objective: To measure FAEE in meconium of fetal sheep following daily ethanol exposure in late gestation, and assess their relationship with fetal organ injury.

Methods: Pregnant ewes received ethanol (0.75 g/kg; n=14) or saline (n=8) over a 1 hour daily IV infusion from 95-133 days gestational age (DGA; term ~147 days), while additional sheep served as untreated controls (n=6). Sheep were euthanized on 134 DGA, and meconium was collected and analyzed for FAEE (ethyl palmitate, stearate, linoleate, and oleate).

Results: The daily ethanol regimen produced similar maximal maternal and fetal plasma ethanol concentrations of 0.11-0.12 g/dL. Ethanol-exposed fetuses had significantly higher meconium total FAEE concentrations compared with controls, and the meconium FAEE concentration demonstrated high sensitivity and specificity for detecting fetal ethanol exposure. In the combined animal population (ethanol-exposed and controls), meconium total FAEE concentration in individual fetuses correlated with numerous pathological changes in fetal organs, including nephron endowment, relative heart weight and cardiomyocyte maturation, lung collagen deposition, and changes in gene expression in fetal lungs, cerebral vessels, and placenta. Furthermore, FAEE-positive and negative groups frequently differed with respect to these endpoints.

Conclusion: In fetal sheep, meconium FAEE concentration could serve as a biomarker of daily, moderate-dose ethanol exposure in late gestation, and could be used to identify fetuses exhibiting subtle ethanol-induced changes in various organs.

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3. PREVALENCE OF DRUG USE DURING PREGNANCY IN MIRAMICHI, NB – ANALYSIS OF A ROUTINE URINE DRUG SCREEN IN THE OBSTETRIC UNIT

Delano K1,2, Holland C3,4, McMackin L3,4, Pope E5,5, Dickinson M4, Koren G1,2
1 Department of Pharmacology, University of Toronto, Toronto, ON;
2 Division of Clinical Pharmacology and Toxicology, The Hospital for Sick Children, Toronto, ON;
3 Faculty of Medicine, Dalhousie University, Halifax, NS;
4 Miramichi Regional Hospital, Miramichi, NB;
5 Faculty of Health Sciences, McMaster University, Hamilton, ON

ABSTRACT

Background/Objectives: Since 2006, Miramichi Regional Hospital (MRH) in New Brunswick, conducts routine urine drug screens on all women admitted for labour and delivery to identify women using recreational substances during pregnancy. The aims of this study were to characterize this group by calculating rates of positivity for all drugs tested over the past 7 years, and to compare positive cases to non-drug using pregnancies and their respective maternal and neonatal outcomes.

Methods: A retrospective chart review is currently ongoing at MRH. Controls are matched by native status. Maternal urine drug screen results, medical history, and outcomes are
collected, as well as neonatal outcomes, through chart review.

**Results:** Thus far, 292 positive cases have been identified, with 25% being native status. The most common drugs detected are marijuana, opioids, and benzodiazepines. Cases were found to be significantly more likely to be smokers (62% vs. 17.5%), have lower education level (45% did not complete high school vs. 19%), and psychiatric disorders (ex. depression, anxiety) (20.5% vs. 9.2%). Cases had increased rates of hemorrhage and placental abruption when compared to controls. Neonates of cases were found to have significantly lower birth weight, increased length of hospital stay, and more symptoms associated with withdrawal (ex. increased muscle tone, high pitched crying) than controls.

**Conclusions/Discussion:** In this ongoing study, women who use substances during pregnancy have higher rates of adverse outcomes, both maternal and neonatal, than women who did not. These rates have major public health implications.

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4. **THEORY OF MIND IN ADOLESCENTS WITH FETAL ALCOHOL SPECTRUM DISORDER**
Matijasevich M1,2, Rinne A1,2, Agnihotri S1,2,3, Keightley M1,3
1 University of Toronto,
2 The Hospital for Sick Children,
3 Holland-Bloorview Kids Rehabilitation Hospital

**ABSTRACT**

**Background:** Adolescents with Fetal Alcohol Spectrum Disorder (FASD) demonstrate difficulty with social cognitive skills required for social relationships. Studies of younger children with FASD have linked these impairments to theory of mind (ToM) deficits, but these findings have not yet been extended to adolescents.

**Objective:** To examine whether ToM is uniquely impaired in adolescents with FASD.

**Methods:** Fourteen adolescents with FASD (M = 15.26 +/- 1.62 years) and 13 typically developing control participants (M = 15.61 +/- 1.36 years) between the ages of 13 – 17 completed the ToM subtests of the Neuropsychological Assessment-II (NEPSY-II) at the Hospital for Sick Children, Toronto. Using linear regression, the relationship between FASD and ToM was explored, considering the influence of age, gender, and socioeconomic status.

**Results:** No significant correlations were found between FASD diagnosis, age or socioeconomic status and ToM task performance on any subtest (p > 0.05). Gender significantly predicted performance on Verbal (r = -0.50, p < 0.01) and Total ToM subtests (r = -0.56, p <0.01). Gender explained 25% of the variance in ToM Verbal scores (F(1, 25) = 8.52, p < .01) and 31% of the variance in ToM Total scores (F(1, 25) = 11.12, p < .01). Females outperformed males on both subtests.

**Conclusions:** Near ceiling performance on NEPSY-II ToM subtests in both groups suggest that these tasks may not be sensitive enough to detect differences in adolescents’ ToM. Findings support previous research demonstrating that gender predicts ToM performance. Future research should investigate the use of dynamic ToM tasks to explore ToM in adolescents with FASD.

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5. DEVELOPING AN EARLY SCREENING AND DIAGNOSIS MODEL FOR FASD INTERVENTION
Gareri J\textsuperscript{1,2}, Nulman I\textsuperscript{1}, Mamak E\textsuperscript{1}, Levin L\textsuperscript{3}, Koren G\textsuperscript{1,2}
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\textsuperscript{3}Toronto Children’s Aid Society, Toronto, Canada

ABSTRACT
Background/Objectives: An objective biomarker, fatty acid ethyl esters (FAEE) in meconium, has been developed to provide confirmation of prenatal alcohol exposure; however no correlation between a positive FAEE in meconium result and deficits associated with FASD has been made within Canadian children. This study aims to determine the percentage of children with FAEE-positive meconium that will receive a diagnosis of FASD and create a model framework for early follow-up and diagnosis of children at risk.

Methods: The target enrolment is 32 FAEE-positive subjects and 32 FAEE-negative controls matched for concurrent prenatal drug exposures, gender, and number of foster home placements. The test-ordering social worker or physician on file was contacted for permission to contact the family by letter and/or telephone; once permission was granted the current guardian(s) of the child were contacted to request enrollment in the study. Once permission is granted, children are enrolled into the a neurodevelopmental monitoring programs as early as 6 months of age until 5 years at which time they undergo a full FASD assessment in accordance with the Canadian guidelines.

Results: In the first round of recruitment (2009-2011), of N = 61 children where contact was attempted; n = 4 children were enrolled and assessed. In the second round of recruitment (2012-2013), of N = 157 children where contact was attempted, n = 2 children were enrolled and assessed. To date, two of the four children that have completed FASD neuropsychological and physical assessment display probable or possible diagnoses of ARND.

Read Full Article, http://www.jptcp.com/pubmed.php?articleId=431

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6. ETHNOCULTURAL FACTORS INFLUENCE LOW PREVALENCE OF FASD
Kapalanga J\textsuperscript{1,2}, Ovuga E\textsuperscript{2}, Onen CL\textsuperscript{3}, Huizink L\textsuperscript{4}
\textsuperscript{1}Pediatrics, Schulich School of Medicine/Western University/GBHS, Canada,
\textsuperscript{2}Gulu University, Uganda,
\textsuperscript{3}Princess Marina Hospital, Gaborone, Botswana,
\textsuperscript{4}CNorth Benin Hospital, Benin

ABSTRACT
Background: Current evidence suggests that in-utero exposure to alcohol has the potential for causing FASD in all racial and cultural groups. However the prevalence of FASD is not uniform in different population groups. Reasons given for these disparities in FASD prevalence have included socio-economic, political, genetic and epigenetic factors.
Objectives: In this study we explore the influences of cultural, faith, and epigenetic factors on the prevalence of FASD in three racially similar populations. We hypothesize that singly or severally ethnocultural, faith and epigenetic factors influence the prevalence of FASD in a given population. Methods: Three geographically localized populations groups, with high and widespread alcohol consumption rates were identified. Physicians who are familiar with features of FASD and have been in practice in these populations for at least 10 years were sent standard FASD diagnostic questionnaires to identify patients aged 5 to 8 year, who show features of FASD.

Results: The populations size of the three groups studied ranged from 50,000 to 70,000. All three populations are Negroid; two of the Sudanic linguistic group and one of the Bantu linguistic group. All lived in rural areas of three different countries; Benin, Botswana and Uganda, respectively. One group is predominantly catholic, the second group predominantly Moslem and the third group is of mixed faith denominations. The prevalence of FASD in the three population groups ranged from 0.1 to 0.25 per 100. The differences in prevalence rates were not statistically significant.

Conclusion/Discussion: Ethnicity, faith, linguistic group and geographic factors do not influence the prevalence of FASD in the three population groups studied. Shared epigenetic factors and other shared cultural factor could be the explanation for the low prevalence of FASD in the three African populations. In certain African cultures drinking of alcohol by women is severely proscribed. This could be the most important single factor for explaining the low prevalence of FASD in these populations.

Read Full Article,
http://www.jptcp.com/pubmed.php?articleId=431

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2013 FACE (Fetal Alcohol Canadian Expertise) poster competition abstracts
7th September 2013

7. TRANSLATING EPIGENETIC ALTERATIONS IN A MOUSE MODEL TO HUMANS
Laufer BI1, Kapalanga J2, Diehl EJ1, Mantha K1, Kleiber ML1, Chokroborty-Hoque A1, Alberry BLJ1, Koren G3, Singh SM1
1 Molecular Genetics Unit, University of Western Ontario, London, Ontario,
2 Department of Pediatrics, Schulich School of Medicine & Dentistry, University of Western Ontario, London, Ontario, and
3 Division of Clinical Pharmacology and Toxicology, Department of Pediatrics, The Hospital for Sick Children, Toronto

ABSTRACT
Background: While much FASD research has focused on sociological, behavioural, and neuro-structural changes, prenatal alcohol exposure also results in long-term alterations in gene expression. A small but growing number of independent international research groups have begun to speculate that the mechanisms underlying the persistence of these changes are Epigenetic marks; Stable but potentially reversible alterations in gene expression that occur without changes to the underlying DNA sequence. Some epigenetic processes are linked to the chromatin (i.e., DNA, histone proteins, and other associated proteins), commonly involve chemical modifications (e.g. methylation) and operate at as on or off switches at the level of transcription, while others (e.g. miRNA) may fine-tune gene expression post-transcriptionally.

Results: In our latest research publication (Laufer et al. Disease Models & Mechanisms 2013), we used four ethanol treatment protocols to model developmental ethanol exposure in mice: injections at 3 specific neurodevelopmental time points that model a “binge” exposure,
and a voluntary maternal consumption model, which represents a moderate and chronic exposure throughout development. We then assessed small RNA brain gene expression in resulting adult offspring (PD 70) using miRNA expression arrays, gene expression arrays, and qPCR. The analysis revealed that a large number of microRNAs are altered, both up and down, depending on treatment paradigm. Some of these expression profiles are unique to a treatment protocol while others overlap. Strikingly, approximately 20% of the altered noncoding RNAs (ncRNAs) localized to three imprinted clusters. The first two, Snrpn-Ube3a (Human 15q11-q13) and Dlk1- Dio3 (Human 14q32.2), are associated with processes involved in neuronal plasticity and several neurodevelopmental disorders that include schizophrenia, autism, Prader–Willi syndrome, and Angelman Syndrome. Next, we assessed brain DNA methylation using methylated DNA immunoprecipitation followed by hybridization to DNA arrays (MeDIP-Chip), which revealed that even moderate fetal alcohol exposure has a genome-wide effect on DNA methylation, with PTEN/AKT/mTOR/PI3K signaling and imprinted regions of the genome appearing to be particularly sensitive. More recent results from our group have examined the hippocampal formation and confirmed brain region specificity of these results via analysis of gene expression, miRNA and snoRNA regulation, and DNA cytosine methylation. Histone modifications and trans-generational studies are a current avenue of investigation. Conclusion: Our results suggest that imprinted ncRNAs, which play critical roles in neurodevelopment and brain function, may have a role in the long-term maintenance of altered gene expression and cognitive endophenotypes associated with FASD.

**Discussion:** Current research from our group is examining how environmental conditions; both enriched and deprived, affect disease development. This presentation will conclude with our current efforts to translate these results to create a diagnostic molecular profile in humans, which may then be used by clinical researchers to establish therapeutic attenuations, given the highly dynamic nature of epigenetic marks.

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MOFAS

A. FASD AND THE IMPACT ON LAW AND POLICING- WEBINAR

David Boulding is a practicing attorney and has written extensively about Fetal Alcohol and the Law, and spoken to police forces and other audiences in the United States, Canada, Australia and the Philippines. David’s presentation will focus on a clinical and working analysis of defining Fetal Alcohol Spectrum Disorders. He will also discuss how to identify an individual with an FASD and how lawyers, judges, police, probation, and corrections can better understand the consequences of the disorder and the ethical issues raised by this preventable disorder. He will also provide some practical solutions and pointers to a complex problem.

Certificates of Attendance will be provided. MOFAS has applied to the following Minnesota Boards for Continuing Education Credits: Board of Continuing Legal Education and the Board of Social Work.

When: Tuesday, December 10, 2013
Time: 12-1:30PM
Registration: Click here
Cost: $25

Questions Contact: Ruth Richardson, ruth@mofas.org or 651-917-2370

Link to the Article

Crickey
Melissa Sweet
Nov 21, 2013

B. TO IMPROVE CHILDREN’S LIVES, AUSTRALIA MUST ACT ON ALCOHOL MARKETING, PRICING AND AVAILABILITY

A clear message from this week’s Australasian Fetal Alcohol Spectrum Disorders (FASD) conference in Brisbane is the need for population-based strategies to tackle alcohol availability, marketing and pricing.

And perhaps the rest of Australia could learn from community-driven initiatives at Fitzroy Crossing, suggests journalist Mardi Chapman.

Mardi Chapman reports:

In the 20th century Australia’s economy was described as ‘riding on the sheep’s back’. Yesterday at the Austrasian FASD conference, Australian society was more grimly described as floating in a sea of alcohol.

According to Sue Miers AM, chair of the National Organisation for Fetal Alcohol Spectrum Disorders (NOFASD Australia), alcohol was the elephant in the room.
For all the important work presented on understanding FASD, its prevention, diagnosis and management – the steps to making FASD history had to include a serious challenge to Australia’s infamous drinking culture.

The conference was told Australians consume about twice the amount of alcohol as comparable countries, on a per capita basis. That already alarming rate doubles again in some communities.

Conference participants also heard that young people, and women in particular, were increasingly engaging in risky behaviours such as binge drinking.

Alcohol contributes to illness, injury, violence and premature deaths, yet we remain strangely accepting of these harms.

The University of Sydney’s Professor Elizabeth Elliott AM said it was not reasonable to expect young women alone to change their drinking behaviour.

“Efforts to encourage women not to drink during pregnancy in order to avoid fetal alcohol exposure should be underpinned by strong action from government to reduce all alcohol related harms.”

There is strong evidence that population-based strategies such as alcohol pricing and taxation policy or restrictions on the density of liquor outlets and licensed premises’ opening hours are effective in reducing alcohol related harms.

Marketing and promotion of alcohol, especially through advertising and sponsorship in sport – that other great Aussie tradition – also came in for a serve during the conference.

June Oscar AO, CEO of the Marninwarntikura Women’s Resource Centre in WA’s Fitzroy Crossing, gave a passionate plea for the issue of FASD to become ‘everybody’s business’.

The extent of the crisis in her community, where FASD is so often ‘seen in the faces and behaviours of the children we love’ prompted locally driven alcohol restrictions, which have now been in place for six years.

It didn’t come easily and they initially faced ‘fierce resistance from those addicted to a destructive lifestyle’.

Without change, she said, the possibilities for the future of the children and the community would have been severally and cruelly limited.

About 80 km down the road from the FASD conference, thousands of school leavers are currently indulging in the modern tradition, and excesses, of Schoolies Week on the Gold Coast.

Most are under age, some of the images are unpleasant, and there will be alcohol related harm.

It seems that the rest of Australia could learn something from an Indigenous community prepared to ‘transform the environment we were trapped in’.

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EDMONTON – It’s a popular toy almost every person played with as a child, but a local program has proven Lego can also be a very beneficial therapy tool for children who live with mental health issues. 

“I just say ‘we go to Lego so we can have the building blocks to give you the building blocks,’” said Chris Parlee, whose twin boys are enrolled in the Lego-Social Skills Group in St. Albert.

Tyler and Tyson Burns have Fetal Alcohol Spectrum Disorder (FASD). Parlee says her sons don’t follow rules well, and she was looking for a therapy program for them that wasn’t too structured, but just structured enough that they could learn basic social skills like sharing.

“They need to have slower-paced, simple instruction,” Parlee explained. “The Lego gives them that.”

So she signed her boys up for Lego Therapy, led by Registered Nurse Dyan Eybergen. The therapy focuses on children with mental health issues, and helps them improve their social skills.

“A lot of them come in and they’re so lacking in social skills that we see a lot of parallel play; they’ll play around each other, they’ll play beside each other, but they won’t play with each other,” Eygerben explained.

Lego Therapy was developed more than 15 years ago by a doctor in New Jersey, after
noticing that children with autism and other neurobehavioural disorders were naturally attracted to the colourful building blocks.

“"It naturally re-enforces social behaviour. Because they have this affinity for Lego, because they love playing with it, they have to cooperate, they have to work with one another in order to build something and accomplish something," Eygerben explained.

Each therapy session begins with the children breaking off into groups of three, and with the help of a teenage leader, the small groups focus on peer interaction.

“They take turns being Lego leaders, Lego engineers, Lego sorters, Lego builders and we rotate every 15 minutes. So in that dynamic, they learn turn-taking and collaboration skills, problem solving skills when they come up with an issue, leadership skills as they’re following instructions, transitioning from one role to another role,” Eygerben said. “And it’s all through practice that they learn how to interact with one another in socially appropriate ways.”

After that, the children come together for free Lego-building time, which is a bit less structured.

“We really encourage self-initiated peer contact, we look for communication, we do a lot of conflict resolution during that time," Eygerben explained.

She says the group work gives the children time to learn how to invite others to play, and be open to other children’s ideas of play.

“They start to self-soothe and calm themselves much quicker than when they first come to us.”

Parlee says she’s noticed a huge difference in her boys’ behaviour since they started the program over the summer.

“They had learned how to share. And it wasn’t like they’re perfect, but you could hear them at home saying ‘you’re not supposed to take that from me. You’re supposed to ask nicely. How do you think I feel?’”

Parlee says Lego Therapy has not only helped the boys with their social skills, it’s also helped them build confidence.

“When they first met Dyan, I think we sat for an hour in a coffee shop and I think if she got three words out of them she was lucky. Now, they’ll approach people, they’ll say ‘hello, this is who I am.’ They’re polite,” she explained with a smile. “Just because they have a disability, just because they’re not like everybody else, doesn’t mean they can’t fit in. You want them to learn that.

“Just even knowing that they’re coming to a place and they can identify to a peer group boosts their self esteem. It’s incredible to watch.”

Lego Therapy is also offered at Terwillegar Recreation Centre. When Eygerben started the program in 2012, there were just four children enrolled. Now, she’s got a waiting list.

In order to keep up with the demand, Eygerben is running a crowdfunding campaign through Alberta BoostR. She’s trying to raise $5,000 to purchase more Lego and create a subsidy program to reach families who may not be able to afford program fees.

Link to the Article,  

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Thousands of people may be ending up on the wrong side of the law due to an alcohol-related brain disability caused before birth, a New Zealand judge says.

Auckland District and Youth Court Judge Tony FitzGerald gave the keynote address at the Australasian Fetal Alcohol Spectrum Disorder conference in Brisbane yesterday.

Justice FitzGerald, who has a special interest in the role of Fetal Alcohol Spectrum Disorder (FASD) in youth offending, said failing to address FASD and other neural disabilities was resulting in higher numbers of people becoming entrenched in the criminal justice system.

"We are not identifying these issues and one of the problems for us as a community is that, until we properly identify these issues and properly respond to them, we are not being effective in reducing the risk of reoffending," he said.

"The brain damage is often behind the behaviours that bring them into the criminal justice system. Once they're there, their disability is such that they are at much greater risk of worse outcomes than someone without the disability."

While New Zealand was yet to begin widespread screening for neural disabilities like FASD, caused by women drinking while pregnant, Justice FitzGerald said the country's prevalence rates were likely to match those found by research overseas.

Research figures suggest around 25,000 people who came into contact with the criminal justice system in New Zealand suffered from FASD. Those who suffered from FASD needed to be treated differently in the court system, he said.

The brain damage, caused when a fetus was exposed to alcohol in the womb, often resulted in people being unable to manage their emotions.

FASD sufferers also had difficulty in properly understanding social cues and observing social boundaries.

"For example, with young men entering adolescence, it can often play out with inappropriate sexual behaviour and offending because they don't have the ability to properly understand those social boundaries and social cues."

Resources need to be made available within the court system to ensure sufferers were picked up, he said.
"Then with the benefit of diagnosis, to be able to ensure that the right sort of responses be made to that case to ensure that the risk of reoffending is properly addressed."

Those who were deemed unfit to stand trial because of their disability would be directed to an "appropriate health related outcome" outside the justice system, he said.

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**E. EMORY ANNOUNCES NEW FREE SERVICE FOR EXPECTANT MOMS**

A new free service, providing expert answers about medications and other exposures during pregnancy and breastfeeding, is giving expectant moms in Georgia another reason to be thankful this Thanksgiving.

Emory University School of Medicine announces MotherToBaby Georgia, a free statewide counseling service that connects experts in the field of birth defects research with expectant moms, health care providers, and the general public. All it takes is a simple phone call to a toll-free number, 866-626-6847. MotherToBaby GA is funded by the Georgia Department of Behavioral Health and Developmental Disabilities.

MotherToBaby GA is an affiliate of the international non-profit Organization of Teratology Information Specialists (OTIS), a prestigious professional society that supports and contributes to worldwide initiatives for education and birth defects research. MotherToBaby affiliates and OTIS are suggested resources by many agencies, including the Centers for Disease Control and Prevention (CDC), dedicated to providing evidence-based information.

"Reliable information about the risks of medications, vaccines, alcohol, drugs of abuse, chemicals, and other exposures during pregnancy or while breastfeeding, is often difficult to find, especially online. We wanted to be sure that pregnant women and health care providers knew that experts on the most cutting edge research were readily available to them," explained Claire Coles, PhD, director of MotherToBaby GA, which is housed at the Center for Maternal Substance Abuse and Child Development Center in the Department of Psychiatry and Behavioral Sciences at the Emory School of Medicine.

Dr. Coles further explains the need for this sort of counseling since approximately 50% of women report taking at least one medication during pregnancy. "The average woman doesn’t find out she’s pregnant until she’s five or six weeks along. That means a woman could have been consuming alcohol or taking medications during that time without knowing she’s pregnant. She then finds herself deeply concerned about what it might mean for her developing baby."

Surveys indicate that while the majority of callers are pregnant women, most have been referred by physicians, nurses, midwives and pharmacists.
“What is passed from mother to baby is exactly what we educate the public about, which is why we strongly believe MotherToBaby GA will provide a beneficial service in our state,” said Patricia Olney, MS, board certified genetic counselor and pregnancy risk information specialist. Olney answers calls from around the state and provides counseling over the phone. “We offer an added layer of support by providing her with an individualized risk assessment so she may make informed health decisions along with her primary health care provider,” she added.

For more information about MotherToBaby GA please visit: http://www.MotherToBaby.org or http://www.emory.edu/msacd.

For counseling, call toll-FREE 866-626-6847 from throughout North America. In Georgia, you can also call 855-789-6222, or email: mothertobaby@emory.edu.

Link to the Article, http://atlantadailyworld.com/2013/11/18/emory-announces-new-free-service-for-expectant-moms/

ABC Kimberley
By Vanessa Mills and Ben Collins
18th November 2013

F. PUPPETS AND HIP HOP LEAD THE FIGHT AGAINST FOETAL ALCOHOL SPECTRUM DISORDER

A collection of puppets depicting a Tennant Creek family affected by foetal alcohol spectrum disorder (FASD), is opening a difficult conversation. The choices parents make could prevent babies being born into lives of health and behavioural problems.

FASD is an umbrella term describing a range of permanent birth defects caused by alcohol consumption during pregnancy. The challenge for Adele Gibson, the FASD Coordinator for the Anyinginyi Aboriginal Health Corporation in Tennant Creek, was overcoming cultural, educational and language barriers to the prevention message.

In the face of a serious and tragic health problem, Ms Gibson turned to puppets and hip hop. "Our three custom-made puppets have these fantastic latex faces, and they’re our FASD family. There’s mum - Lila, dad - Clem, and seven-year-old son Mathias who has FASD. And mum is pregnant for the second time, but this time she knows not to drink," says Ms Gibson.

"Basically what I’ve had to do is get across a negative message. So you’re telling people not to do something. Whenever you have a ‘not’ or a ‘no’, you’ve got to balance it with something positive," she says.

Over two years the community created the puppets which were used to tell stories of FASD in short films as well as original music which broached the issue. The film, Barkly Fights FASD, was launched to the community in October and had an immediate impact.

"A hundred people came through the doors and they loved the film. And we used the film as a catalyst for a community forum," Ms Gibson says.
Personal experience

Adele Gibson’s passion for preventing FASD comes from a very personal experience. She was a foster parent for the first eight years of a boy’s life who was severely affected.

"We watched what he went through. He had a lot of surgery. He had a tracheostomy. He is, and probably always will be fed through a tube in his stomach. He had a really hard time, and probably always will. So that gave me the passion for prevention I suppose, and was also my steep learning curve about foetal alcohol spectrum disorder," she says.

The Kimberley has played a role in helping Tennant Creek's attempt to prevent FASD. Fitzroy Crossing Aboriginal leaders and health campaigners June Oscar and Emily Carter, visited Tennant Creek to share ideas and inspiration.

"Strong Aboriginal women coming here and talking to other strong Aboriginal women was always going to be a winner," says Ms Gibson

"We've watched in amazement what they've done with their communities there, and it's given us hope for the future for us."

Listen to Adele Gibson on Kimberley Mornings with Vanessa Mills.


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Royal College of Obstetricians and Gynaecologists

G. BJOG RELEASE: PRE-PREGNANCY ALCOHOL CONSUMPTION AFFECTS ALCOHOL USE AMONG PREGNANT WOMEN, SUGGESTS NEW STUDY

Most women who consume alcohol pre-pregnancy will continue to consume alcohol throughout their pregnancy even when healthcare guidelines promote abstinence, suggests a new study published today in BJOG: An International Journal of Obstetrics and Gynaecology.

The study, which included 1,969 women from the Australian Longitudinal Study on Women’s Health (ALSWH), aimed to gauge the predictors of antenatal alcohol use, among women who usually consume alcohol, to identify those most at risk of an alcohol-exposed pregnancy.
In addition to pre-pregnancy alcohol consumption, other predictors investigated included socio-economic status, reproductive health, mental health, physical health, alcohol guidelines and healthcare variables.

The women completed five surveys in 1996, 2000, 2003, 2006 and 2009 (referred to as survey one to five respectively), but for this study researchers focused on women who reported being pregnant in surveys two to five and then used surveys one to four, respectively, to gauge pre-pregnancy behaviours.

Of the 1,969 women, 82% reported consuming some alcohol during pregnancy, although most reported low alcohol usage with 77% of these women consuming one or two drinks per drinking day and 90% drinking no more than once or twice a week.

The findings showed that women who drank weekly prior to pregnancy were 50% more likely to continue to drink during pregnancy, compared to those who drank less than weekly. Furthermore, women who reported prior binge drinking behaviour were more than twice as likely to continue to consume alcohol during pregnancy.

Researchers, from the University of Newcastle in Australia, also identified the presence of alcohol guidelines as an important factor in antenatal alcohol use, especially during the periods where the Australian healthcare guidelines changed throughout the study.

Women who reported a pregnancy at survey two (2000) or five (2009) were classified under the ‘no alcohol’ guidelines (promoting abstinence), whereas women who reported a pregnancy in surveys three (2003) and four (2006) were categorised under the ‘low alcohol’ guidelines (which condoned light drinking). Of the women who were pregnant during the times that alcohol guidelines promoted abstinence, 22% did not drink compared to 14.7%, who refrained under the low alcohol guidelines.

Other factors that influenced antenatal alcohol consumption included women with fertility problems, who were 36% less likely to consume alcohol than those who had no trouble conceiving, and women with a Health Care Card (indicating lower socio-economic status), who were 37% less likely to drink during pregnancy.

Amy Anderson, Priority Research Centre for Gender, Health and Ageing, University of Newcastle and lead author of the study, said:

“Most women who drank alcohol prior to pregnancy, especially those who drank weekly or reported binge drinking behaviour, continued to drink during their pregnancy indicating that pre-pregnancy alcohol consumption was a significant risk factor for antenatal alcohol use.

“Our results also suggest that more conservative drinking guidelines may influence the behaviour of pregnant women. However, even under guidelines promoting abstinence, the majority of women continued to consume alcohol while pregnant.

“These findings suggest that to avoid alcohol-exposed pregnancies, risky episodic and regular alcohol use by women of childbearing age should be addressed prior to conception. More effective dissemination of guideline recommendations may also be useful in reducing the high prevalence of antenatal alcohol use among Australian women.”

John Thorp, BJOG Deputy-Editor-in-Chief added:

“Heavy antenatal alcohol use has been shown to cause a number of adverse health outcomes but the effects of low to moderate alcohol use are less clear.

“While it is important to gauge pre-pregnancy behaviour and make clear the intent of alcohol guidelines relating to pregnant women, this study further verifies that socio-economic factors can also influence antenatal alcohol use.
“The fact that women identified as lower-income earners (Health Care Card holders) were found to drink less needs to be considered by antenatal healthcare professionals when informing pregnant women of alcohol use. Clinicians should not assume patient knowledge based on socio-economic status.

“Advice for women managing or planning a pregnancy should focus on the fact that the first trimester is a particularly sensitive time and alcohol should be avoided to prevent risks to crucial development at this stage. Women concerned about alcohol consumption during pregnancy should consult their midwife or GP.”

Ends

For press enquiries please contact Caitlin Walsh, Media Officer, Royal College of Obstetricians and Gynaecologists: 020 7772 6300 or cwalsh@rcog.org.uk

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MOFAS
2013
Victoria

H. MY TEENAGE LIFE WITH AN FASD

I love to share my opinion on how I feel about certain things, how I feel conflicted or how I feel personally about FASD and how it affects me.

I think that people should know more about it in general. There are so many people that know nothing about it, never have heard about it or may know a little about it or even don’t care. They need to be educated that there is a huge risk of drinking during pregnancy that will affect their kids’ future whether it be between relationships with friends and family, their education and how they handle different environments and fitting in.

Because fitting in is one of the biggest problems that all kids and teenagers face and kids with FASD it’s harder because of social difficulties. It’s harder for us to make friends and especially keep friends.

Another huge thing is that kids with FASD can’t process and they don’t pick up on social cues whether it be body movements or how other kids act towards them so often that they will have problems with their friends. If our friend gets mad at us, or is teasing us, we may take it very harshly and not know if they were teasing. I don’t pick up on when kids are teasing or joking or sarcastic but they don’t understand how difficult it is to process that sometimes. When we say they didn’t know, the other kid will laugh at us and it may be in a joking manner. It is hard to interact with our peers and teachers at school and often cannot focus in class and often don’t understand what is being taught.
We usually don't ask questions because we don't want to be looked at as unintelligent or want to fit in and that usually ends up in bad grades and misunderstandings between family members and educators. Often when we find someone we can connect with on a very high level, someone we get along with really well, we often become clingy and possessive and that behavior—the majority of the time will be taken as obnoxious or annoying.

Kids with FASD often do not learn from mistakes and repeatedly break the rules. We make decisions with poor cause and effect reasoning and not very exceptional judgment skills. If we have a bad relationship, like a parasitism relationship, with a friend, we might refuse the fact that that person is bad for us and stick with that person whether they are causing us emotional and/or physical harm/pain. Kids with FASD react on a larger scale to different things and we don’t understand why people take it as we are overreacting when we simply do not understand.

A positive is kids with FASD are very friendly, kind and helpful, very visual and creative. With everything that happened in my life, including FASD, positive and negative things that have happened to me throughout my life, I have to be grateful for everything that has happened and is going to happen for if it had not had happened, I wouldn’t be here today saying what I am, doing what I do, loving what I love and knowing the people I know and I am grateful for that.

Link to the Article,
https://www.mofas.org/2013/11/teenage-life-fasd/

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From Luxembourg (Translation follows)
21st July 2013

I. GEFAHR AUCH IN DEN ERSTEN MONATEN

(pm) - Ständig warnen Mediziner und Wissenschaftler vor den Gefahren von Alkoholkonsum während der Schwangerschaft oder nach der Geburt. Der Genuss kann zu fetalen Alkoholspektrumstörungen (FASD) mit körperlichen Missbildungen, Gehirnschäden oder geistigen Behinderungen führen.

Über die Nabelschnur oder die Muttermilch trinkt das Kind mit. So baut ein Kind im Mutterleib den Alkohol bis zu zehnmal langsamer ab als die Mutter.

Um werdende oder stillende Mütter über diese Gefahren zu informieren, stellte Gesundheitsminister Mars di Bartolomeo am Donnerstag die Kampagne „Null Prozent während der Schwangerschaft oder der Stillzeit“ vor.

Alkohol ist ein Zeitgift, das die heranwachsenden Organe, das Gehirn und/oder die Nerven schädigen kann. Der Konsum kann während der ganzen Schwangerschaft zu Schäden führen - auch in den ersten Monaten, wenn sich viele Frauen ihrer Schwangerschaft noch gar nicht bewusst sind.

In Luxemburg werden jährlich etwa dreißig Kinder mit FASD geboren. Weniger als zehn Prozent dieser Menschen können später selbständig leben.


Beide Syndrome - FASD und FAE - könnten demnach zu schwerwiegenden sozialen und/oder intellektuellen Auswirkungen im Umgang mit der Gesellschaft und zu einer radikalen Ausgrenzung führen.
Hunderprozentiger Verzicht

Die Betreuung z.B. von FASD-Betroffenen wirkt sich erheblich auf die Sozialkosten aus. Demnach sei FASD nur durch einen hundertprozentigen Verzicht auf Alkohol während der Schwangerschaft und der Stillzeit vermeidbar, so Simone Steil von der "Médicine préventive".

Karoline Noworyto vom Gesundheitsministerium betonte, dass 46 Prozent schwangerer Frauen im Jahr 2012 angegeben hatten, sich mit ihrem Arzt über „Alkohol in der Schwangerschaft“ beraten zu haben. 61 Prozent gaben an, vor der Schwangerschaft gelegentlich oder regelmäßig Alkohol getrunken zu haben, sechzehn Prozent auch während der Schwangerschaft.

Demnach werde Alkoholkonsum immer noch verharmlost oder durch unklare und falsche Vorstellungen banalisiert.

**English Translation:**

**RISK IN THE FIRST FEW MONTHS**

(Pm) - Employed physicians and scientists warn of the dangers of alcohol consumption during pregnancy or after birth. The enjoyment can lead to fetal alcohol spectrum disorders (FASD) with physical deformities, brain damage or mental disabilities.

Through the umbilical cord or breast milk drinking with the child. As a child in the womb builds the alcohol up to ten times slower than the mother.

To inform expectant or nursing mothers about these dangers, Health Minister Mars di Bartolomeo presented on Thursday the campaign "Zero percent during pregnancy or lactation" before.

Alcohol is a time poison that can damage the growing organs, the brain and / or nerves. The consumption can lead to damage during the whole pregnancy - even in the first few months of pregnancy when many women are not even aware of.

About thirty children are born with FASD each year in Luxembourg. Less than ten percent of these people live on their own.

Children who are born with fetal alcohol effect (FAE) are not immediately recognizable. In this country there are about 60 births this year. Brain damage and mental deficits make themselves apparent until later.

Both syndromes - FASD and FAE - could therefore lead to serious social and / or intellectual impact in dealing with society and a radical exclusion.

Hunderprozentiger waiver

The care e.g. affected by FASD has a significant impact on the social costs. Thus, FASD is preventable only through a wholly owned abstaining from alcohol during pregnancy and lactation, as "preventive medicine" Simone of the ball.

Caroline Noworyto stressed by the Health Ministry that 46 percent of pregnant women were given in 2012, to have to consult with their physician about "alcohol during pregnancy." 61 percent said they have occasionally or regularly drank alcohol before pregnancy, sixteen percent during pregnancy.

Thus, alcohol consumption will still downplayed or trivialized by unclear and misconceptions.
J. ALCOHOL, BABIES AND THE DEATH PENALTY: SAVING LIVES BY ANALYSING THE SHAPE OF THE BRAIN

Alcohol can damage the brains of unborn babies. Shape analysis can assess the damage in fetal alcohol spectrum disorders. Kanti Mardia, Fred Bookstein and John Kent explain how it works, and how it can help babies and even murderers.

Read Full Article,  

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FETAL ALCOHOL SPECTRUM DISORDERS INTERDISCIPLINARY PERSPECTIVES
Edited by Barry Carpenter, Carolyn Blackburn and Jo Egerton

Fetal alcohol spectrum disorders (FASDs) have emerged as a major phenomenon within the education, health, criminal justice and social care systems of many countries. Current prevalence figures suggest that one in 100 children and young people have FASDs – similar to those for autistic spectrum disorders. With contributions from leading academics, families and professionals from a range of disciplines around the world, this book offers an invaluable and cutting-edge contribution to how we understand and address the complex social, educational and health needs associated with this growing group of children and young people. The multidisciplinary and family perspectives and insights on FASDs create a rich knowledge base grounded in lived experience. Any education, social care, criminal justice or health professional working with children and young people with FASDs and their families will find this book a seminal and authoritative resource.

Barry Carpenter is Honorary Professor at universities in the UK, Ireland, Germany and Australia. He conducted research on teaching children with FASD at the University of Oxford. He regularly lectures on FASD throughout the world. Previously Barry was National Director for the DfE Project on Children with Complex Learning Difficulties and Disabilities. Carolyn Blackburn is a member of the Early Childhood Research Group at Birmingham City University, UK, where she is also a visiting lecturer on the Early Childhood Education Studies degree course. Carolyn was the lead researcher for two projects investigating the educational implications and early childhood practitioner knowledge of FASD in the UK. She is also the lead author of the first UK text on the educational needs of children and young people with FASD and has been an invited speaker at numerous conferences in relation to FASD. Jo Egerton is Research Project Coordinator for SSAT (The Schools Network) Ltd, and lead Research Coach for their Research Charter Mark Award. She has published in the areas of research methods, FASD, complex needs and ASD.


FASD RELATIONSHIPS
(2011) Rod Densmore, M.D.

Rod Densmore offers a unique perspective on FASD-related issues as both a medical practitioner and a parent of a young adult with FASD. With the intention of adding a practical, well-referenced, accessible, engaging and inexpensive teaching tool to existing resources, Densmore has successfully compiled research findings on a large variety of topics relevant to FASD and translated them into user-friendly language. He addresses the complexity of these issues through the expertise of a medical professional, yet with the sensitivity and passion that comes from parenting a person with FASD. FASD Relationships encompasses a book and five DVD set, with each chapter of the book forming a series of modules about FASD.

FASCETS Inc at 503-621-1271; $20.00 (US) or www.fascets.org Also now available at Odin Books 1-800-223-6346; $22.95 (Can.) or www.odinbooks.ca

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ORIGINAL ARTICLE

What Research Is Being Done on Prenatal Alcohol Exposure and Fetal Alcohol Spectrum Disorders in the Russian Research Community?

Svetlana Popova1,2,3,4,*, Aleksandra Yalontskaya5,6, Vladimir Yalontsky5,7, Yaroslav Kolpakov5,7, Ilya Abrosimov5,7, Kristina Pervakov1, Valeria Tanner1 and Jürgen Rehm1,2,4,8

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Abstract — Aims: Although Russia has one of the highest rates of alcohol consumption and alcohol-attributable burden of disease, little is known about the existing research on prenatal alcohol exposure (PAE) and Fetal Alcohol Spectrum Disorders (FASDs) in this country. The objective of this study was to locate and review published and unpublished studies related to any aspect of PAE and FASD conducted in or using study populations from Russia. Method: A systematic literature search was conducted in multiple English and Russian electronic bibliographic databases. In addition, a manual search was conducted in several major libraries in Moscow. Results: The search revealed a small pool of existing research studies related to PAE and/or FASD in Russia (126: 22 in English and 104 in Russian). Existing epidemiological data indicate a high prevalence of PAE and FASD, which underlines the strong negative impact that alcohol has on mortality, morbidity and disability in Russia. High levels of alcohol consumption by women of childbearing age, low levels of contraception use, and low levels of knowledge by health and other professionals regarding the harmful effects of PAE put this country at great risk of further alcohol-affected pregnancies. Conclusions: Alcohol preventive measures in Russia warrant immediate attention. More research focused on alcohol prevention and policy is needed in order to reduce alcohol-related harm, especially in the field of FASD.

INTRODUCTION

Russia has one of the highest rates of alcohol consumption in the world. According to the Global Burden of Disease Study (2010), alcohol use is the second leading risk factor contributing to the disease burden in Russia (Institute for Health Metrics and Evaluation, 2012). The World Health Organization (WHO) European and Global Status Report on Alcohol and Health reported that on average, for 2003–2005, the level of per capita consumption of pure alcohol in liters among adults older than 15 years of age reached 26.7 l (16.3 l for women and 35.4 l for men; WHO, 2010, 2011). In Russia, 5.8% of women and 22.1% of men are heavy episodic drinkers (i.e. consume at least 60 g or more of pure alcohol on at least one occasion weekly; WHO, 2010, 2011). The prevalence of alcohol use disorders is also high in Russia—2.6% for women and 16.3% for men (WHO, 2011). As a result, Russia is characterized as having the most risky pattern of drinking and the greatest alcohol-attributable burden of disease in the world.

A recent study conducted in Saint Petersburg and the Nizhny Novgorod region revealed that 89% of non-pregnant women reported consuming alcohol and 65% reported binge drinking in the past 3 months (Balachova et al., 2012a). Forty-seven percent of women in the Nizhny Novgorod region and 28% in Saint Petersburg reported at least one binge-drinking episode at least monthly. Women who might become pregnant consumed alcohol similar to women who were not likely to become pregnant, and 54% of women in the Nizhny Novgorod region and 32% of women in Saint Petersburg were considered to be at risk of having an alcohol-exposed pregnancy (Balachova et al., 2012a).

Popovitch et al. (2004) conducted a telephone-based behavioral risk factor surveillance survey in three Russian cities in 2000–2001. A random sample of 3032 residential telephone numbers was selected and 1693 interviews were conducted among adults 25–64 years of age in each selected household: 21% of women from Moscow, 8.2% of the women in Arkhangelsk and 10.8% of women in Murmansk abstain from alcohol. From 3 to 5% of women (and about 30% of men) in the above Russian cities consume >20 g of pure alcohol per day. Among women, about 30% of consumed beverages were spirits and 40% was beer. The highest level of hazardous alcohol consumption (>20 g of pure alcohol per day) among women was reported for those between 25 and 34 years of age, the most reproductive age group (Popovitch et al., 2004).

An article from the mid-1990s reported that 80–94% of high-school girls consumed alcohol and in major Russian cities, girls drank at the same level or higher than boys of the same age (Koshkina and Paronyan, 1995).

According to the official report of the Russian National Research Center on Addiction for 2011, the prevalence of alcoholism in the general population of Russia is estimated to be 1.4% (1402 per 100,000 persons), and ~0.5% of women (505 per 100,000) have been diagnosed with alcoholism (Koshkina et al., 2012). A ratio of 1:5 (women:men) has been reported to exist between the genders (Altshuler, 2010). Given that the official figures are based on reported cases only, the actual number of cases of alcoholism among women in Russia may be higher (if unreported cases are considered; Kirganova,
It has also been suggested that in Russia, from 1999 to 2003, the number of registered cases of alcoholism in women increased from 2.2 to 3.7% (Alshuler et al., 2006).

In addition to the alarming data on alcohol consumption by women in the general population, research indicates that there is a low level of contraception use. Only 49% of all women in Russia who have ever been sexually active use contraception, and that number increases to only 51% when women in the age group of 50–54 were excluded (Barden-O’Fallon et al., 2010). In another sample of 347 non-pregnant women 18–44 years of age recruited at women’s clinics, 44% of women in Saint Petersburg and 70% in the Nizhny Novgorod region were sexually active and not using contraception consistently (Balachova et al., 2012a).

Due to high levels of alcohol consumption within the population, paired with low rate of contraceptive use, many Russian women of childbearing age are at risk of having an alcohol-exposed pregnancy, which may result in having a child with Fetal Alcohol Spectrum Disorder (FASD). FASD is a non-diagnostic umbrella term that is used to represent the full spectrum of birth defects that are caused by prenatal alcohol exposure (PAE), which encompasses four categorical diagnostic entities: fetal alcohol syndrome (FAS), partial FAS (pFAS), alcohol-related neurodevelopmental disorder (ARND) and alcohol-related birth defects (ARBD; Chudley et al., 2005). PAE is a leading preventable cause of the birth defects, which can include physical, mental, behavioral and/or learning disabilities with possible lifelong complications. In North America, the lifetime cost for some cases of FASD has been estimated to be more than one million dollars (Popova et al., 2011).

Despite the high risk of, and potentially high cost associated with, FASD in Russia, there are no official surveillance efforts targeting FASD and no social facilities for children and/or adults with FASD in the country. Furthermore, very little is known about the existing pool of research on PAE or FASD in Russia. Therefore, the purpose of this study was to perform a systematic literature review in order to locate published and unpublished studies related to any aspect of PAE and FASD that were conducted in Russia or used study populations from Russia.

METHODS

Systematic literature search

Participating researchers from Canada and from Russia simultaneously conducted a systematic literature search. The procedure varied due to the differences in the availability of sources of information. For detailed search strategy in both countries and results, see Supplementary material, File S1.

The search was not limited by language of publication and was conducted up to and including December 2012.

RESULTS

In total, 22 studies in English and 104 studies in Russian were included in the present review. The results of the search strategy are shown in Fig. 1.

The 126 examined studies were categorized into the following six major groups, those that:

1. assessed the prevalence of alcohol consumption during pregnancy and the clinical complications of alcohol-exposed pregnancies;
2. examined the prevalence of FASD in the general population, or other specific populations (e.g. orphanages);
3. assessed the efficacy of preventive measures, methods of diagnostics and interventions for individuals with FASD;
4. conducted literature reviews on FASD and developed educational materials for health professionals;
5. clinically examined children and adults and demonstrated the lifelong polysystemic teratogenic effects of alcohol on the fetus and
6. experimentally studied the teratogenic effects of alcohol on laboratory animals or human biological material.

A summary of all examined studies in chronological order is available in Supplementary material, Table S1.

Studies on the prevalence of alcohol consumption during pregnancy and the clinical complications of alcohol-exposed pregnancies

Some studies reported both the prevalence of alcohol consumption and clinical complications. Therefore, these studies have been categorized by the primary purpose of the study.

(a) The prevalence of alcohol consumption during pregnancy

The search revealed 11 studies reporting the prevalence of alcohol consumption during pregnancy (see Supplementary material, Table S1). It was found that alcohol consumption during pregnancy in Russia varies from 3.1% (Talykova et al., 2007) to 83% (Kurianova et al., 2006) and heavy drinking during pregnancy varies from 0.2–1% (Radzinsky, 2002) to 9.2% (Shilko et al., 2011a). In a study by Koshy et al. (2010) it was reported that 50% of females consumed seven or more alcoholic drinks during the 3 months prior to pregnancy. Chambers et al. (2006) conducted a longitudinal pregnancy outcome study in the Moscow Region of Russia and revealed that 52% used alcohol in their most recent month of pregnancy. Further, these authors reported that, of those ‘ever drinkers’, 4.8% had at least one episode of five or more standard drinks, and 10.5% had at least one episode of three or four standard drinks in the most recent month of pregnancy. The survey conducted by Kristjanson et al. (2007) in Saint Petersburg among 899 pregnant women revealed that 96% of them drank alcohol in the year before they became pregnant; of these women, 60% reported drinking when they knew they were pregnant, and 35% had reported drinking within the past 30 days. Among pregnant women who drank within the past 30 days, 7.4% reported having 5 or more drinks on at least 1 occasion. One recent study reported that 16% of pregnant women with disorders of the pancreas and liver practiced heavy drinking (Egorova et al., 2012). The drink of choice among pregnant Russian women was reported most commonly as wine, sparkling wine and beer (Kurianova et al., 2009).
et al., 2006; Kurianova, 2006; Sashchenko, 2007; Kalinina et al., 2012).

Several studies revealed that Russian women substantially reduce their alcohol consumption after pregnancy recognition, when compared with pre-pregnancy levels (Balachova et al., 2012a; Gaidukov et al., 2008); however, about 20% continued to consume alcohol after pregnancy recognition (Balachova et al., 2012a). Binge drinking after pregnancy recognition declined by a factor of 10, but did not disappear completely; ~6% of women had at least one binge episode.

It is important to note that the levels of alcohol consumption during pregnancy within all of the reviewed studies were based on maternal self-reports, which are likely to be imprecise and most likely underreported due to bias (e.g. recall and social desirability biases). Furthermore, the term ‘standard drink’ is not a term commonly used in Russia; therefore, the respondents may have had difficulty understanding this concept. In addition, there were inconsistencies between studies in the definition of a ‘binge drinking’. For example, Balachova et al. (2012a) defined binging as ‘four or more standard drinks on a single occasion’, while Chambers et al. (2006) defined binge drinking as an episode of ‘five or more drinks’.

It should also be noted that Russia is geographically large and culturally diverse; therefore, regional variations are important. Thus, findings on the prevalence and patterns of alcohol consumption during pregnancy from individual studies may not be representative of all Russian women.

(a) The clinical complications of alcohol-exposed pregnancies

There were seven studies describing the clinical complications of alcohol-exposed pregnancies in Russia (Supplementary material, Table S1). The research studies included in this analysis have reported that alcohol consumption during pregnancy increases the risk of miscarriage (Sashchenko, 2007; Komarova, 2008), fetoplacental insufficiency (Orazmuradov et al., 2007), premature or accelerated delivery (Komarova, 2008; Alekseeva, 2011a,b), pathology of amniotic fluid (Komarova, 2008; Alekseeva, 2011a,b) and disturbances in the mother’s immune system (Kurianova et al., 2006; Sashchenko, 2007; Komarova, 2008). Several other research studies reported that alcohol consumption during pregnancy decreases the levels of zinc and copper in the blood stream of pregnant women, as well as the levels of cobalt, iodine, magnesium and phosphorus in pregnant women’s hair (Ogotoeva and Borisova, 2008; Ogotoeva et al., 2009a,b; Alekseeva, 2011a,b). Furthermore, these studies reported that newborns of these mothers are deficient in certain essential minerals, such as cobalt, iron, iodine and zinc, and have increased levels of lithium and nickel. Shilko et al. (2010c, 2011c) stated that increased levels of transforming growth factor (TGF-β1), found in the blood of alcohol misusing pregnant women, might explain the growth retardation in newborns.
Due to the small sample sizes of the studies presented above (Orazmuradov et al., 2007; Ogotoeva and Borissova, 2008; Ogotoeva et al., 2009a,b; Ulyanovskaya and Solovev, 2010; Shilko et al., 2010c, 2011c; Kalinina et al., 2012), and the fact that the level of alcohol consumption during pregnancy was not defined, was based on self-reported data obtained via unstructured interviews, and the timing of alcohol use was not specified (i.e., first, second or third trimester; Shilko et al., 2010c, 2011c; Alekseeva, 2011a,b; Alekseeva and Ivanova, 2011; Kalinina et al., 2012), these results should be interpreted with caution.

Studies reporting on prevalence of FASD
In total, 17 studies reported the prevalence of FASD in different populations of Russia (see, for example, Warren et al., 2001; Riley et al., 2003; Grigovich et al., 2006; Miller et al., 2007; Malakhova et al., 2008; Konovalova et al., 2009) (Supplementary material, Table S1). The prevalence of FAS in the general population was estimated in one study to be 18–19 per 1000 live births (Malakhova, 2012). However, a study of 326 kindergarten-aged children of the same area did not report any cases of FAS (Bubnov, 2009, 2010). The prevalence of FAS in maternity hospitals in Saint Petersburg was ranging from 3 to 35 per 1000 during 2000 (Palchik et al., 2009) to 340 (Sofronova and Palchik, 2012) per 1000 live births by Russian researchers and from 55 (Stromland et al., 2005) to 330 (The St. Petersburg-USA Orphanage Research Team, 2005) per 1000 live births by international researchers. For comparison purposes, it is believed that the prevalence rate of FAS in the North American general population is ≈1 per 1000 live births (Roberts and Nanson, 2000; May and Gossage, 2001).

Studies by Marincheva et al. (2003) reported the prevalence of FAS to be 129 per 1000 in a boarding orphanage, 58 per 1000 in boarding schools, 49 per 1000 in regular orphanages and 164 per 1000 in a school of the social welfare system. The highest prevalence of FAS was found in ‘psycho-correctional’ orphanages for children with special needs, ranging from about 427–680 per 1000 (Legonkova, 2011; Palchik and Legonkova, 2011).

The prevalence of FAS among adopted children from Russia (or in some cases, from Eastern Europe, including Russia) currently living in the USA was estimated to range from 15 to 70 per 1000 live births (Aronson, 2003; Robert et al., 2009). Farina et al. (2004) reported that 34% of children adopted from Russia were diagnosed with ARND. However, the records of alcohol-exposed pregnancy were significantly higher and constituted 19% (Albers et al., 1997) to 41% (McGuinness et al., 2000) of the cases. Albers et al. (1997) stressed that the majority of prospective adopting parents are concerned about the high risk of FASD among children adopted from Russia, due to the widespread availability of alcohol and the limited public awareness of alcohol’s detrimental effects on the fetus.

The existing FASD prevalence estimates in Russia vary greatly from population to population and study to study. These variations may reflect not only differences in maternal drinking behavior, but also different diagnostic criteria, methods of case ascertainment (i.e. surveillance method) and populations surveyed in the different studies.

The existing studies have a number of limitations due to (a) inadequate and/or unavailable diagnostic capacity across the country and (b) lack of nation-wide diagnostic guidelines and definitions. Further, the studies presented above have weak methodologies, small sample sizes that are restricted to sub-populations, used convenience sampling and had a low response rate. There are also inconsistencies in the use of the terms ‘incidence’ and ‘prevalence’ across the studies.

Studies on FASD prevention, diagnostics and interventions
There were five studies found on selective prevention measurement, two on interventions, and three on diagnostic procedures (Jones et al., 2006; Shilko et al., 2008a, 2009c, 2010b, 2011a; Kuznetsova et al., 2011) (Supplementary material, Table S1).

The studies by Balachova et al. (2007) and Isurina et al. (2009) demonstrated that, in Russia, with the exception of pediatricians, professionals such as physicians, psychologists, nurses and social workers have insufficient knowledge about FASD and usually do not discuss the negative effects of alcohol consumption during pregnancy with their female patients. These studies also demonstrated that gynecologists and pediatricians often had misconceptions or inconsistent attitudes toward alcohol exposure during pregnancy such as ‘it is tolerable to consume low doses of alcohol in cases involving a healthy pregnancy’ (a misconceived attitude). Pediatricians, who were characterized as having inconsistent attitudes, assumed that it is important to convince a woman to abstain from alcohol consumption during pregnancy; however, good quality alcoholic beverages can be consumed during the late stage of pregnancy. Only medical doctors specializing in addiction treatment stated that complete abstinence from alcohol during pregnancy is necessary. Despite the small number of participants, these results (Balachova et al., 2007; Isurina et al., 2009) indicate a lack of knowledge regarding the detrimental effects of alcohol use during pregnancy, even among highly educated individuals in Russia.

In a brief PAE prevention intervention based on evidence-based interventions using techniques of motivational interviewing (Balachova et al., 2010a,b), women were counseled to choose safe contraception or complete abstinence from alcohol consumption. It was recommended that gynecologists of the maternity welfare centers conduct this intervention because Russian women considered them as a reliable source of information (Moskalenko 2002, 2008a,b; Balachova et al., 2010a,b).

Balachova et al. (2012b) also evaluated different types of informational leaflets with positive (positive visual images, list of positive outcomes for baby’s health in case of abstinence from alcohol), negative (negative visual images, list of negative consequences of alcohol consumption during the pregnancy) and neutral information (general information about healthy lifestyle during pregnancy). The study found that information about FASD increases the general awareness of woman of childbearing age regarding FASD, stimulates them to consider changing their behavior toward abstinence from alcohol in case of pregnancy planning or pregnancy and decreases the level of alcohol consumption among women, in
Research on prenatal alcohol exposure and FASD in Russia

5

general. It was also found that women who read the leaflets with positive information had better memories and were able to reproduce factual information about FASD. However, women who read negative information were more likely to develop a strong decision to abstain from alcohol during pregnancy (Balachova et al., 2012b; Regentova, 2012).

As can be seen from the above, there are few published studies on FASD prevention, diagnosis and interventions in Russia. Studies of prevention efforts targeting high-risk women are completely absent.

Only two studies concerning the treatment of individuals with FAS were found in the Russian literature. Transcranial direct current stimulation was reported to be beneficial in normalizing the altered sleep-wake cycle in 13 children with FAS (Malakhova and Bubnov, 2011; Malakhova et al., 2011). Another study reported that after implementing peptide-antiminotoxic therapy on 56 children with FAS, an improvement in psychomotor development was observed in 56–77% of cases, while a positive effect of standard therapy on psychomotor development was observed in 45% cases (Khasanova, 2010). These studies should be considered with caution due to (a) the absence of an appropriate control group, (b) the lack of a clear definition of the outcomes, (c) small sample sizes and (d) lack of appropriate statistical analyses.

Literature reviews and educational materials for health professionals

The search revealed 28 literature reviews that included information on FASD for health professionals (see, for example, Petrov-Maslakov, 1961; Lezhepekova, 1981; Bakanov, 1986, 1999; Mastiukova, 1986, 1989; Skalny and Skosyreva, 1987; Radzinsky and Kostin, 2009; Popova, 2010). (Supplementary material, Table S1). The reviews provided information on a variety of topics including: the evaluation of medical views on the FASD problem through the years, descriptions of the mechanisms of the damaging effects of alcohol on a fetus, descriptions of the clinical characteristics of children with FAS (e.g. growth retardation, birth defects/abnormalities, changes in phenotype, and neurodevelopmental and intellectual disorders) and strategies for FASD prevention (see, for example, Badalyan, 1986; Tabolin and Uryvchikov, 1986; Alipov and Korkhov, 1988; Lisitcyn and Sidorov, 1990; Akhmadeeva, 1997; Ramazanova and Semiatov, 2002; Palchik and Shabalov, 2009; Balachova et al., 2012c).

An educational trial was conducted to evaluate FASD training developed for obstetricians/gynecologists and pediatricians (Balachova et al., 2010a,b). This study claimed that the inclusion of a 3-h FASD education module in continued medical education led to significant changes in physicians' knowledge, attitudes and skills necessary for diagnosing FASD and conducting brief prevention interventions. Furthermore, this study implemented new technologies to disseminate developed FASD education materials and designed the first internet-based FASD educational resource in the Russian language for physicians and other health professionals (www.NetFAS.net; Balachova et al., 2010a,b).

Several studies discussed the ways in which alcohol influences the development of FASD such as the direct damage of alcohol on sex cells, genitals (Koshkina et al., 1998; Shilko et al., 2008b), indirect autoimmune effects (Shilko et al., 2008b) and metabolic disturbance (Lebedev, 1974; Bakanov, 1986; Anokhina and Moskalenko, 1987; Garmasheva and Konstantinova, 1988). The negative effect of alcohol on newborns through breastfeeding has also been reported (Bisiarina and Lisitcyna, 1987; Koshkina et al., 1998). Additionally, some studies discuss the role of acetaldehyde in fetal central nervous system damage (Bakanov, 1986; Anokhina and Moskalenko, 1987; Garmasheva and Konstantinova, 1988). These studies report that an increase in acetaldehyde, ethanol’s metabolite, due to insufficient maternal alcohol dehydrogenase activity, is the leading damaging factor in FAS development.

Several studies underline the dose-dependent effect of alcohol on the fetus. Some studies suggest that consumption of 60–80 g of pure alcohol per day leads to FAS (Frolova and Nikolaeva, 1987), while others report that consumption of 150 g of pure alcohol per day increases the chance of FAS development by 50% (Koshkina et al., 1998).

Interestingly, in the older Russian sources from the 1980s, the detrimental effects of alcohol on the fetus were described and pregnancy termination for women with alcohol dependency was strongly recommended (Skosyreva, 1980; Kirushenkov, 1986).

As can be seen from the literature reviews listed above, the majority of them are outdated—70% were published before 2000. In addition, most of the existing reviews only provide general information on FAS. Information pertaining to the practical aspects of diagnostics, treatment, care and prevention are only touched on very briefly. Thus, they are extremely limited in their clinical applicability.

Clinical studies examining children and adults

There were 22 clinical studies conducted on children affected by FASD (Supplementary material, Table S1). These studies provide a description of neurodevelopmental, mental, cardiovascular, gastrointestinal, metabolic and other disorders presented in children with PAE and/or FASD. (Semenov et al., 1987; Donetc, 1992; Erkhoova and Bozhenov, 1997; Gribovski et al., 2002, 2004; Khatchek and Popov, 2005, 2009a,b, 2011; Miller et al., 2006; Ruchkin et al., 2008; Palchik et al., 2009, 2011; Kashirskaya, 2010; Khoroshkina and Krivtsova, 2010; Sheffer, 2012).

The majority of these studies have serious limitations, including: (a) small sample sizes (Shurygin, 1974; Usova et al., 1981; Grechany, 2002; Legonkova and Palchik, 2009), (b) lack of a control group (Usova et al., 1981; Gummel et al., 2007a,b; Khatchek and Popov, 2011) and (c) a failure to properly confirm PAE and thus, a failure to properly establish an FASD diagnosis (Shurygin, 1974; Kunikovskaja, 1980; Kornilov et al., 2005; Sokolovskaya et al., 2009). Most importantly, in all of the existing clinical studies examining children and adults a standardized diagnostic procedure was not used/indicated.

Experimental studies with laboratory animals or human embryos

The search revealed 25 experimental research studies conducted on rats (see, for example, Skosyreva, 1973; Skosyreva et al., 1973; Anokhina et al., 1989; Kolomeitceva et al., 1989; Maizelis et al., 1989; Nozdrecha et al., 1989; Zabludovsky et al., 1989; Zhulin and Bazyan, 1989; Chebotar and Konopistceva, 1993; Omelianchik et al., 1993; Kataeva et al., 2004; Kurch, 2004, 2011; Sverdlova, 2008; Vyatchanina and 1986; Anokhina and Moskalenko, 1987; Garmasheva and Konstantinova, 1988). The negative effect of alcohol on newborns through breastfeeding has also been reported (Bisiarina and Lisitcyna, 1987; Koshkina et al., 1998). Additionally, some studies discuss the role of acetaldehyde in fetal central nervous system damage (Bakanov, 1986; Anokhina and Moskalenko, 1987; Garmasheva and Konstantinova, 1988). These studies report that an increase in acetaldehyde, ethanol’s metabolite, due to insufficient maternal alcohol dehydrogenase activity, is the leading damaging factor in FAS development.

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As can be seen from the literature reviews listed above, the majority of them are outdated—70% were published before 2000. In addition, most of the existing reviews only provide general information on FAS. Information pertaining to the practical aspects of diagnostics, treatment, care and prevention are only touched on very briefly. Thus, they are extremely limited in their clinical applicability.

Clinical studies examining children and adults

There were 22 clinical studies conducted on children affected by FASD (Supplementary material, Table S1). These studies provide a description of neurodevelopmental, mental, cardiovascular, gastrointestinal, metabolic and other disorders presented in children with PAE and/or FASD. (Semenov et al., 1987; Donetc, 1992; Erkhoova and Bozhenov, 1997; Gribovski et al., 2002, 2004; Khatchek and Popov, 2005, 2009a,b, 2011; Miller et al., 2006; Ruchkin et al., 2008; Palchik et al., 2009, 2011; Kashirskaya, 2010; Khoroshkina and Krivtsova, 2010; Sheffer, 2012).

The majority of these studies have serious limitations, including: (a) small sample sizes (Shurygin, 1974; Usova et al., 1981; Grechany, 2002; Legonkova and Palchik, 2009), (b) lack of a control group (Usova et al., 1981; Gummel et al., 2007a,b; Khatchek and Popov, 2011) and (c) a failure to properly confirm PAE and thus, a failure to properly establish an FASD diagnosis (Shurygin, 1974; Kunikovskaja, 1980; Kornilov et al., 2005; Sokolovskaya et al., 2009). Most importantly, in all of the existing clinical studies examining children and adults a standardized diagnostic procedure was not used/indicated.
and other professionals is not only limited, but also outdated, as the majority of these studies have been published prior to 2000. Moreover, medical education rarely ever includes a course describing the negative consequences of alcohol consumption during pregnancy; most medical doctors are not trained to recognize the clinical features of FASD in Russia (Isurina et al., 2009).

Aside from medical doctors, other health and human services professionals (for example, social workers) are well positioned to help prevent FASD and to intervene with individuals and families affected by FASD. A major setback is that these professionals are also lacking appropriate knowledge and skills regarding the consequences of PAE in Russia (Balachova et al., 2007, 2010a,b). Given the exceptionally high prevalence of children with FASD in foster care, orphanages and other child welfare systems in Russia, there is a need for proper training for professionals in these systems in order to be able to recognize a child with behavioral and/or mental health problems that may be the result of PAE. It is important to make educational materials available to a variety of professionals—such as guidelines for diagnosing and working with individuals already affected, as well as their parents and caregivers.

As a result of the limited research and medical and other educational information, the awareness and knowledge of Russian medical professionals, psychologists, social workers and other professionals about harmful effects of alcohol consumption during pregnancy and FASD is extremely limited (Balachova et al., 2007, 2010a,b; Isurina et al., 2009). There is a large discrepancy between the number of individuals who need to be diagnosed with FASD and receive proper care and the number of available professionals able to provide a proper diagnosis, treatment and other support services.

There is an urgent need to reduce the harmful use of alcohol among women of childbearing age in Russia and initiate prevention strategies targeting pregnant women. Prevention efforts need to be widely spread and should target women of childbearing age and clearly state that there is no safe time to consume alcohol during pregnancy, nor is there a safe amount or type of alcohol. The present literature review presents some effective strategies that could be implemented in these types of FASD prevention programs. For instance, Moskalenko (2002, 2008a,b) advises that the main method of preventing alcohol intake during pregnancy should involve informing women through primary care physicians, and to use positive reinforcement rather than threats. Also, several studies point out that there are misconceptions about FASD, as well as lack of awareness of FASD symptomatology (Balachova et al., 2007). Improving awareness could be an effective way to minimize drinking in women of childbearing age. A randomized educational trial indicated that including FASD information in standard continuing medical education courses significantly improves physicians’ knowledge, attitudes and skills (Balachova et al., 2010a). Another study suggests that simple interventions such as distributing leaflets containing both positive and negative information about the consequences of drinking during pregnancy increase the general awareness of women of childbearing age and their attitudes about FASD (Balachova et al., 2012b; Regentova, 2012). Thus, such research studies are a valuable tool for structuring FASD prevention programs that target women who may become pregnant.

As the literature indicates, the population of alcohol-dependent women in Russia is large; however, the principles of depression-like symptoms or memory disorders in the newborns (Kolomeitceva and Levin, 1989). Furthermore, studies using human biological material demonstrated that when a mother consumes alcohol the embryo may develop many different deviations in brain development (Kovetch et al., 1991a; Solonsky, 2006, Solonsky and Logvinov, 2008). The details of these studies can be found in the Supplementary material, Table S1.

Many experimental research studies have serious limitations; they used small sample sizes (Skalny et al., 2001; Shilko et al., 2009a,b, 2010a,c, 2011b,c), did not describe their sample(s) (Skalny et al., 2009; Morozova and Popova, 2010; Kurch, 2011) and did not present statistical significance (for example, Babenko and Skalny, 1986; Artiukhina et al., 1989).

**DISCUSSION**

Overall, in the last five decades (the first identified article is dated as 1961). 126 studies related to alcohol consumption during pregnancy and FASD were found that were published in Russia (or on Russian samples). This seems low considering Russia’s large area and population [17.1 million km²; 143 million people as of 2012 according to the Federal State Statistics Service of Russian Federation (FSSS; FSSS, 2012)]. This is also surprising given that Russia has one of the highest rates of alcohol consumption in the world. This leaves us with the question ‘why is there such a low interest in this issue in the Russian research community?’

The reviewed studies suffer multiple methodological limitations and weaknesses outlined in the results section and thus, should be viewed with appropriate caution. Despite the limitations, these studies demonstrate a high level of alcohol consumption among Russian women of childbearing age, a low level of contraception usage and an especially high proportion of pregnant women with a pattern of heavy drinking. Based on these findings, it is conceivable that the prevalence of FASD is likely to be high in the general population of Russia.

The present systematic literature review revealed that only a few epidemiological studies reporting the prevalence of FASD exist. However, the reported figures are not generalizable to the general Russian population due to the methodological limitations of the studies (e.g. conducted in small communities with small sample sizes, or conducted among special populations). Valid population-based epidemiological studies are needed to examine the prevalence of FASD in both the general population and populations likely to be at high risk of PAE in Russia. Such information is crucial for understanding the magnitude of the problem and for initiating preventative measures at the country level.

The present review also revealed that the existing research on the negative effects of PAE and FASD available to health professionals is large area and population [17.1 million km²; 143 million people as of 2012 according to the Federal State Statistics Service of Russian Federation (FSSS; FSSS, 2012)]. This is also surprising given that Russia has one of the highest rates of alcohol consumption in the world. This leaves us with the question ‘why is there such a low interest in this issue in the Russian research community?’

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As a result of the limited research and medical and other educational information, the awareness and knowledge of Russian medical professionals, psychologists, social workers and other professionals about harmful effects of alcohol consumption during pregnancy and FASD is extremely limited (Balachova et al., 2007, 2010a,b; Isurina et al., 2009). There is a large discrepancy between the number of individuals who need to be diagnosed with FASD and receive proper care and the number of available professionals able to provide a proper diagnosis, treatment and other support services.

There is an urgent need to reduce the harmful use of alcohol among women of childbearing age in Russia and initiate prevention strategies targeting pregnant women. Prevention efforts need to be widely spread and should target women of childbearing age and clearly state that there is no safe time to consume alcohol during pregnancy, nor is there a safe amount or type of alcohol. The present literature review presents some effective strategies that could be implemented in these types of FASD prevention programs. For instance, Moskalenko (2002, 2008a,b) advises that the main method of preventing alcohol intake during pregnancy should involve informing women through primary care physicians, and to use positive reinforcement rather than threats. Also, several studies point out that there are misconceptions about FASD, as well as lack of awareness of FASD symptomatology (Balachova et al., 2007). Improving awareness could be an effective way to minimize drinking in women of childbearing age. A randomized educational trial indicated that including FASD information in standard continuing medical education courses significantly improves physicians’ knowledge, attitudes and skills (Balachova et al., 2010a). Another study suggests that simple interventions such as distributing leaflets containing both positive and negative information about the consequences of drinking during pregnancy increase the general awareness of women of childbearing age and their attitudes about FASD (Balachova et al., 2012b; Regentova, 2012). Thus, such research studies are a valuable tool for structuring FASD prevention programs that target women who may become pregnant.

As the literature indicates, the population of alcohol-dependent women in Russia is large; however, the principles...
of prevention measures and interventions for alcohol-dependent women are underrepresented in the Russian professional literature. This means that the at-risk women are unlikely to receive an intervention for FASD prophylaxis. System-wide screening strategies and targeted interventions for women of childbearing age with alcohol dependence need to be developed. These recommendations should be primary prevention priorities for substance abuse prevention programs.

To conclude, the existing PAE and FASD research underlines the strong negative impact that alcohol has on mortality, morbidity and disability in Russia (Institute for Health Metrics and Evaluation, 2012; Lim et al., 2012; Neufeld and Rehm, 2013). The regulations introduced within the last decade in Russia seem to show some positive effects on both drinking behavior and health outcomes (Neufeld and Rehm, 2013). However, there is an urgent need for further alcohol-control strategies to reduce alcohol-related harm, especially in the field of PAE and FASD.

SUPPLEMENTARY MATERIAL

Supplementary material is available at Alcohol and Alcoholism online.

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Canadian Children and Youth in Care: The Cost of Fetal Alcohol Spectrum Disorder

Svetlana Popova · Shannon Lange · Larry Burd · Jürgen Rehm

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Abstract

Background A high prevalence of prenatal alcohol exposure has been reported among children in care and thus, the risk of fetal alcohol spectrum disorder (FASD) in this population is high.

Objective The purpose of the current study was to estimate the number of children (0–18 years) in care with FASD and to determine the associated cost by age group, gender, and province/territory in Canada in 2011.

Methods The prevalence of children in care by province/territory was obtained from the Canadian Child Welfare Research Portal, and the number of children in care with FASD for each province/territory was estimated from available epidemiological studies. In order
to calculate the total cost per province/territory, the cost per individual per day, by age group, was applied to the respective number of children in care with FASD.

**Results**  The estimated number of children in care with FASD ranged from 2,225 to 7,620, with an annual cost of care ranging from $57.9 to $198.3 million Canadian dollars (CND). The highest overall cost ($29.5 to $101.1 million CND) was for 11–15 year-olds.

**Conclusion** The study findings can be used to demonstrate the substantial economic burden that FASD places on the child welfare system. Attention towards the needs of this population and prevention efforts to reduce FASD incidence in Canada, and other countries are urgently needed.

**Keywords**  Fetal alcohol syndrome · Fetal alcohol spectrum disorder · Children/youth in care · Child welfare · Cost · Canada

**Introduction**

Children and youth\(^1\) in care represent a unique population with disproportionately increased rates of developmental disabilities, congenital malformations, mental health diagnoses, and social maladjustment (Chernoff et al. 1994; Fuchs et al. 2008; Harman et al. 2000; Hostetter et al. 1991; Lindblad et al. 2003).

Children who are placed in care often are due to a number of unfavourable circumstances, such as, parental and/or drug problems, child abuse and/or neglect, child abandonment, and young maternal age. Such circumstances are likely to increase the likelihood that a child was exposed to alcohol in utero (Burd et al. 2011; Herrick et al. 2011). Thus, the risk of fetal alcohol spectrum disorder (FASD) in this population is likely to be high. FASD is not a diagnostic term, but is an umbrella term encompassing four categorical diagnostic entities: fetal alcohol syndrome (FAS), partial FAS, alcohol-related neurodevelopmental disorder, and alcohol-related birth defects (Chudley et al. 2005; Stratton et al. 1996). FAS is the most severe and visibly identifiable form of FASD. Prenatal alcohol exposure can affect any organ or system of the fetus, therefore, individuals with FASD may have a broad array of physical defects, cognitive, behavioural, emotional, and adaptive functioning deficits, as well as congenital anomalies, such as malformations and dysplasia of the cardiac, skeletal, renal, ocular, auditory, and other systems. These impairments are likely to have lifelong implications.

In a recently conducted study by the authors of this article, utilizing the current epidemiological and medical literature, over 300 disease conditions coded in the International Classification of Diseases, version 10 were identified to occur in individuals with FASD (Popova et al. in progress). The demonstrated complexity and chronicity of FASD draws attention to the fact that these affected individuals require a wide range of assistance from multiple service systems, including health care, community organizations, remedial education, and others. Without crucial support, people affected by FASD are at an increased risk of developing secondary disabilities, such as mental health problems, trouble with the law, school drop-outs, unemployment, homelessness, and/or alcohol and other drug problems (Streissguth et al. 2004). When combined with the child’s primary deficits, these secondary disabilities increase the complexity of care and result in significant social and

\(^1\) The broad term “children” will be used throughout to refer to children and youth.

In the general population of Canada, the crude prevalence of FAS and FASD have been reported as 1 per 1,000 (Roberts and Nanson 2000) and 9 per 1,000 (PHAC 2003), respectively. However, the prevalence of FASD among children in care\(^2\) has been reported to be much higher. For example, in the province of Manitoba, of the 1,869 children in care identified as having a disability, 640 (34\%) had FASD and an additional 280 (15\%) were suspected to have FASD (Fuchs et al. 2005). Based on a total of 5,664 children in care in Manitoba at this time, the prevalence of FASD within this population can be estimated to be 113 per 1,000, which is about 13 times higher than the estimated prevalence of FASD among the general population.

A recent analysis, conducted by the authors of this paper, revealed an alarming prevalence of FASD among children in care in different countries (Lange et al. 2012b, 2013). For example, in Chile, the prevalence of FAS and FASD among children (1 to 20+ years of age) in the care of child welfare (“protective”) services and homes for those with mental deficiencies was reported as 62 per 1,000 and 158 per 1,000, respectively (Mena et al. 1993). In the USA, the prevalence of FAS among children, up to the age of 13, living in foster care was found to be 10–15 per 1,000 (Astley et al. 2002). Further, the prevalence of FAS in foster homes and orphanages in Russia was reported to be extremely high—150 per 1,000 (Bubnov 2010)—and in orphanages for children with special needs, the prevalence was even higher—ranging from 427–680 per 1,000 (Legon’kova 2011). In Brazil, the prevalence of FASD among children residing in orphanages was reported as 277 per 1,000 (Strömland et al. 2011).

The severity of FASD and its associated disabilities are a significant and direct predictor of removing children from their birth homes and placing them in foster care. Thus, Kvigne et al. (2004) reported that among their sample of Northern Plains American Indian children, the children with full-blown FAS were 64 times more likely to be removed from their homes and children with partial FAS were 14 times more likely to be removed from their homes, when compared to children without FAS or partial FAS. Further, children with full-blown FAS were 28 times more likely to be placed in foster care and 14 times more likely to be living with their relatives and children with partial FAS were 5 times more likely to be placed in foster care and twice as likely to be living with relatives, when compared to children without FAS or partial FAS.

There are only a few studies in Canada and the USA that have attempted to estimate the cost associated with FASD (no studies exist in any other country; Popova et al. 2011b, c, 2012b). In the existing FASD-cost analyses (Abel and Sokol 1987, 1991a, b; Harwood 2000, 2003; Harwood et al. 1984, 1998; Harwood and Napolitano 1985; Rice et al. 1990, 1991; Rice 1993; Weeks 1989), the cost of children in care with FASD was not included in the overall estimates. In these studies, the total cost associated with FASD might be underestimated based on the fact that children with FASD are overrepresented in child care systems (Farriss-Manning and Zandstra 2003; Hutson 2006; Popova et al. 2012c, d).

There are only a couple of studies in Canada that have estimated the cost of children in care with FASD. Fuchs et al. (2008) estimated that in 2006 the total annual cost of 400 children in care with FASD in Manitoba, one Canadian province, was $9.5 million. The authors found that the average total daily cost of caring for a child with FASD in the child

\(^2\) The term ‘children in care’ will be used to refer to all children in out-of-home care (i.e., those in the custody of a child welfare agency, Crown Wards [a foster child who has been made the legal responsibility of the Canadian government], and those in foster care).
welfare system was $65 (or $23,760 per annum). However, a study by Stade et al. (2009) estimated that the average annual direct cost per child with FASD in foster care is about $2,000 in Canada. The noticeable disparity between the costs reported by two existing studies is likely due to the utilization of different methodologies and the inclusion and exclusion of different cost components.

The current study is part of a large economic project, with multiple components, working towards estimating the overall burden and cost associated with FASD in Canada (Lange et al. 2012a; Popova et al. 2011a, 2012a, c, d, 2013a, b). Until now, an estimation of the number of children in care with FASD and the associated cost, at the national level, has not been undertaken in Canada, or in any other country.

Hypotheses

It was hypothesized, based on the current Canadian and international literature, that the prevalence of children in care with FASD is likely higher than in the general population and thus, the economic cost associated with children in care with FASD in Canada is considerable.

Study Objectives

The current study was designed to: (1) estimate the number of children in care with FASD by age group, gender, and province/territory and (2) estimate the associated cost of children in care with FASD in Canada in 2011.

An estimation of the cost of FASD for the Canadian child welfare system is central to describing the extent of its impact on this population, the cost to society, and for evaluating the potential benefits of FASD prevention programs. Furthermore, the current economic estimate has the potential to provide additional policy insights in order to better address the needs of this unique population and to increase awareness of this problem not only in Canada, but also internationally.

Method

Design

The current study was a modeling study using secondary data.

Ethics Statement

Given that the current study utilized secondary data reported on the aggregate level, which is readily available in the literature, it was not necessary to obtain research ethics approval.

The Total Number and Prevalence of Children in Care in Canada

The latest estimates of the total number of children (0–18 years of age) in care and the prevalence estimates of children (0–18 years of age) in care per 1,000 among the general population in Canada, by province/territory in 2007 were obtained from the Canadian Child Welfare Research Portal (http://www.cecw-cepb.ca/statistics; CRCF 2011; Table 1).
The methodology of deriving a national estimate of children in care by province/territory is described in a report by Mulcahy and Trocmé (2010). This study used multiple ascertainment strategies to compile available statistics presented in federal, provincial, and territorial documents and websites, including: (a) placement statistics compiled by Human Resources and Social Development Canada for the Federal/Provincial/Territorial Directors of Child Welfare Committee for years 1992–2004 and Social Security statistics on the number of children in out-of-home care from 1971 to 2003; (b) information from Indian and Northern Affairs Canada tracking on reserve Aboriginal children in out-of-home care from 1969 to 2007; (c) statistics reported by provincial and territorial authorities in their annual reports and/or their websites; and (d) statistics reported by provincial/territorial associations (e.g., provincial/territorial Children’s Aid Societies), or through provincial/territorial reviews/reports. The national estimates of children in care by province/territory were calculated using Statistics Canada population estimates for the year 2007 for children 0–18 years of age (Mulcahy and Trocmé 2010).

The Cost of Caring for Children in Care with FASD in Canada

The cost of care per day, by age group and gender, for children in care was obtained from Fuchs et al. (2008). These cost figures were for 2006, but in the current study are reported as inflation adjusted costs for 2011, using the inflation calculator of the Bank of Canada (http://www.bankofcanada.ca/rates/related/inflation-calculator/; see Table 2).

The total cost of care per day, included basic maintenance, special rate/special needs, and exceptional circumstances. Basic maintenance refers to the funds that are required for the everyday costs of providing for children in care (e.g., food, utilities, child care,
replacement clothing); special rate/special needs funds are those that cover costs that exceed or were not intended to be covered by basic maintenance (e.g., fees for service, therapy, medical expenses); and exceptional circumstances are funds that cover expenses that are above and beyond those required for normal care (e.g., support services, criminal legal fees, renovations required to the foster home for a disabled child).

Data Analysis

Estimation of the Number of Children in Care with FASD, by Age Group, Gender, and by Province/Territory in Canada in 2011

To calculate the total number of children in care for each province/territory, the respective prevalence estimates of children in care, obtained from the Canadian Child Welfare Research Portal (CRCF 2011), were applied to the number of children in the general population for each province/territory in Canada in 2011 (obtained from Statistics Canada 2012).

There are only two estimates that exist on the prevalence of children in care with FASD for Canada. The first estimate is 33 per 1,000 (reported for the province of Ontario; Burge 2007) and the second estimate is 113 per 1,000 (reported for the province of Manitoba; Fuchs et al. 2005). In order to estimate the total number of children in care with FASD for each province/territory, these prevalence estimates were applied (as the lower and upper estimates, respectively) to the total number of children in care for each province/territory.

The age and gender distribution of children in care with FASD obtained from Fuchs et al. (2008; Table 2) and was applied to the total estimated number of children in care for each province/territory.

Estimation of the Cost Associated with Caring for Children in Care with FASD by Age Group, Gender, and by Province/Territory in Canada in 2011

The estimated cost figures for children in care with FASD for each age group, gender, and province/territory are reported in 2011 Canadian dollars. The cost per day (adjusted for
inflation for 2011) for each age group was multiplied by 365 (number of days in the year), and applied to the estimated number of children in care with FASD.

**Results**

The Number of Children in Care with FASD, by Age Group, Gender, and by Province/Territory in Canada in 2011

*Prevalence of Children in Care*

The highest prevalence of children in care, in 2011, was in the Northwest Territories (30.8 per 1,000—1 out of every 32 children in the territory), followed by Yukon Territory (24.7 per 1,000—1 out of every 41 children) and Manitoba (24.4 per 1,000—1 out of every 41 children), as reported by the Canadian Child Welfare Research Portal (http://www.cecw-cepb.ca/statistics; CRCF 2011; Table 1). The province/territory with the lowest prevalence of children in care was Prince Edward Island (5.2 per 1,000—1 out of every 192 children in the province), followed by Ontario (6.4 per 1,000—1 out of every 156 children) and Newfoundland (7.5 per 1,000—1 out of every 133 children). However, due to differences in base population sizes, Ontario had the largest number of children in care (18,546), followed by Quebec (12,674) and Alberta (9,377). In 2011, the total number of children in care in Canada was estimated to be 67,433.

*Estimated Number of Children in Care with FASD*

The three provinces with the highest number of children in care with FASD were as follows: Ontario [612 (lower estimate) to 2,096 (upper estimate)], Quebec (418–1,432) and Alberta (309–1,060). Overall, the total number of children in care with FASD in Canada, in 2011, ranged from 2,225 to 7,620.

The estimated number of children in care (0–18 years of age) with FASD by gender, and by province/territory and the associated cost in Canada in 2011 are presented in Table 3.

The Cost of Caring for Children in Care with FASD by Age Group, Gender, and by Province/Territory in Canada in 2011

The overall cost attributable to FASD among children in care ranged from $57.9 to $198.3 million (boys: $36.0 to $123.4 million; girls: $21.9 to $75.0 million) in Canada in 2011 (Table 3).

The age group associated with the highest overall cost was 11–15 years of age, followed by the age group of 6–10 years of age. The 0–5 years of age group was associated with the lowest overall cost. Please see Fig. 1 for the FASD-attributable cost of children in care with FASD by gender and age group in Canada in 2011.

**Discussion**

The results of this study suggest that, in Canada, the annual cost for children in care with FASD is likely to range from $57.9 to $198.3 million. This is clearly a cost component that
<table>
<thead>
<tr>
<th>Province/ territory; gender</th>
<th>Number of children in the general population(^a)</th>
<th>Prevalence of children in care in the general population(^b) (per 1,000)</th>
<th>Number of children in care(^c)</th>
<th>Number of children in care with FASD(^d)</th>
<th>Total cost of children in care with FASD(^e) ($)</th>
<th>Lower estimate</th>
<th>Upper estimate</th>
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<td>Alberta</td>
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<tr>
<td>Boys</td>
<td>193</td>
<td>660</td>
<td>5,010,132.92</td>
<td>17,115,909.68</td>
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<tr>
<td>Girls</td>
<td>117</td>
<td>400</td>
<td>3,042,792.80</td>
<td>10,422,684.42</td>
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<tr>
<td>Total</td>
<td>884,645</td>
<td>9,377</td>
<td>8,053,925.71</td>
<td>27,578,594.10</td>
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<td>163</td>
<td>140,233.54</td>
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</table>

\(^a\) Number of children in the general population
\(^b\) Prevalence of children in care in the general population
\(^c\) Number of children in care
\(^d\) Number of children with FASD
\(^e\) Total cost of children with FASD
\(^f\) Lower estimate $ \times 10^{-6}$
\(^g\) Upper estimate $ \times 10^{-6}$
should not be overlooked in FASD cost studies, when considering the overall burden of FASD on any society. However, the cost of children in care with FASD, estimated in the current study, is only one component of the overall direct cost associated with FASD in Canada.

Despite the fact that the cost for children in care with FASD is substantial, it is likely that it is still underestimated for the following reasons. Firstly, raising children with FASD is a challenging undertaking, one that many foster parents may not be fully prepared for. For this reason, caregivers may choose to put a child with FASD back into provincial/territorial custody, necessitating another foster family placement, which may be associated with additional costs. Secondly, fostering children with FASD requires special considerations, such as training for both staff and foster parents, which is also likely to result in additional costs.

There are several limitations in the current study. Firstly, the estimated number of children in care in Canada in 2011 was based on the prevalence estimates reported by the Canadian Child Welfare Research Portal for the year 2007 (the most recent available

| Province/territory; gender | Number of children in the general population | Prevalence of children in care in the general population (per 1,000) | Number of children in care | Number of children with FASD | Total cost of children in care with FASD ($)
<table>
<thead>
<tr>
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<tr>
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<td>Lower estimate</td>
<td>Upper estimate</td>
<td>Lower estimate</td>
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<td>Girls</td>
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<td>162,183.69</td>
<td>555,356.26</td>
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<td>CANADA (all provinces/territories)</td>
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<tr>
<td>Boys</td>
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<td>67,433</td>
<td>57,917,032.24</td>
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</table>

Due to rounding errors, columns may not add up to the totals reported

FASD fetal alcohol spectrum disorder

a Statistics Canada (2012)
b CRCF (2011)
c Estimated based on the respective provincial/territorial prevalence rates
d Estimated based on prevalence 33 per 1,000; Burge (2007)
e Estimated based on prevalence 113 per 1,000; Fuchs et al. (2005)
f Estimated based on the 2011 inflated cost per day; Fuchs et al. (2008)
and was assumed to remain constant during the year, which may not be accurate. Secondly, the prevalence of FASD among children in care is currently available for only two Canadian provinces—Ontario (Burge 2007) and Manitoba (Fuchs et al. 2005), which were used to estimate the prevalence of children in care with FASD in the other provinces/territories. Therefore, possible variations between the provinces/territories were not accounted for. Thirdly, due to a lack of respective provincial/territorial data, the age and gender distribution of children in care with FASD, as well as the costs of care per day, were obtained from a study based on the child welfare system in Manitoba (Fuchs et al. 2008) and applied to the other provinces/territories, which may not be generalizable to the other provinces/territories of Canada. Lastly, it should be noted that the provinces/territories of Canada vary in regard to the age at which youths are no longer permitted to be in care (i.e., some provinces/territories may only allow youths up to that age of 16 to be in care, while others may permit youths up to the age of 18 or even 21). These provincial/territorial differences were not considered in the current estimate.

Unfortunately, currently, there are no other national estimates on the cost of children in care with any other conditions/disorders. Such data would be useful to put the current estimate into context. However, it is known that the average per diem special rate/special needs cost for a child with FASD is approximately 20% higher than that of the general children in care population ($43 versus $35; Fuchs et al. 2008). It has also been documented that children with FASD enter care at an earlier age and are more likely to become permanent wards and therefore, spend a greater proportion of the lives in care than children in care without FASD (Fuchs et al. 2008). Thus, not only are daily special rate/special needs costs of children with FASD higher, but those costs are extended over a longer period of time.

Cost studies in general have important implications for public policy. Important public policy findings of this study include: (a) funding for FASD prevention (e.g., educating women of childbearing age about the detrimental consequences of consuming alcohol while pregnant) should be a priority—if 10% of the cost of care were devoted to prevention, Canada could allocate as much as $20 million each year to this objective; (b) improving data collection on prenatal alcohol exposure is necessary, especially for at-risk populations—this would facilitate early and accurate diagnostic evaluations; (c) enhancing access to substance abuse treatment programs for the mothers of children with FASD should be made possible—this could have substantial benefits not only for the
mother and her current and future children, but also for society as a whole; and (d) increasing the effectiveness of substance abuse treatment programs for women of childbearing age, should be a principle goal—this could provide an important opportunity to prevent the occurrence and/or recurrence of FASD within families. It is a well-known fact that FASD is highly recurrent within siblings (Abel 1988). Taking a long-term perspective on this issue would suggest that increasing access to and the effectiveness of substance abuse treatment could improve the lives of several thousand children and their families (Gelb and Rutman 2011; Popova et al. 2013b).

It is also very important to identify strategies to prevent affected children from needing to be placed in care. Multiple efforts will be required to address the needs of this unique population and, via increasing the awareness, to reduce the overall prevalence/incidence of FASD in both the child care population, as well as the general population in Canada and around the world. Further, the development and implementation of programs aimed at reducing or preventing secondary disabilities, if successful, could result in very large cost savings.

The implications of this study for child care agencies include several broad areas of emphasis. Substantial rates of FASD occurrence among children in care suggest that there is an increased need for specialized training of workers, caregivers, and service providers to improve FASD recognition, for understanding the specific needs of children with FASD, and an increased need for comprehensive service plans to support children with FASD and their adoptive families. Based on the high reported prevalence of FASD in child care systems around the world (Lange et al. 2012b, 2013), children in care must be routinely screened for FASD and be referred to diagnostic services where, if necessary, a formal diagnosis can be made. Such an approach has the potential to facilitate early diagnosis, which has several noteworthy benefits (Popova et al. 2013a).

In regard to future research, the limitations of the current study draw attention to the need for accurate prevalence estimates of children in care with FASD in the provinces/territories of Canada. Also, there is a need for cost analyses to be conducted within provincial/territorial child welfare systems. Such analyses will help to make informed decisions regarding the programs, policies, and funding support for the numerous activities required to improve the lives of children in care with FASD and to prevent further alcohol-exposed births in Canada. Further, research is also needed to identify the unique medical, educational, and social needs of children in care with FASD, and their current patterns of service use (versus service needs).

The burden of children in care with FASD is not measurable by cost of care alone, the lifelong hardships faced by these children, and their families should also be considered. A strategy that could change the lives of several thousand of Canada’s most vulnerable people surely deserves consideration at this time. Prevention of FASD may well be one of Canada’s priorities for the future. For many children and their families the cost of not acting to prevent FASD and to improve the treatment of individuals with FASD may be too great to bear. If we do not act, the children in care with FASD will likely not have their needs adequately addressed, and thus, will likely experience negative outcomes, such as secondary disabilities, that will not only impact the individual, themselves, well into adulthood, but also the society in which they live (Bueller et al. 2000; Quinton et al. 1984). The substantial number of children in care with FASD, the high associated cost, and their dependence on the child care system in general, emphasizes the urgency of strategically addressing their needs. We will act, won’t we?
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Conflict of interest  The authors declare that they have no conflicts of interest.

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Effects of Ethanol Exposure during Distinct Periods of Brain Development on Hippocampal Synaptic Plasticity

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4 Department of Cellular and Physiological Sciences, University of British Columbia, Vancouver, V6T 1Z4, British Columbia, Canada

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Abstract: Fetal alcohol spectrum disorders occur when a mother drinks during pregnancy and can greatly influence synaptic plasticity and cognition in the offspring. In this study we determined whether there are periods during brain development that are more susceptible to the effects of ethanol exposure on hippocampal synaptic plasticity. In particular, we evaluated how the ability to elicit long-term potentiation (LTP) in the hippocampal dentate gyrus (DG) was affected in young adult rats that were exposed to ethanol during either the 1st, 2nd, or 3rd trimester equivalent. As expected, the effects of ethanol on young adult DG LTP were less severe when exposure was limited to a particular trimester equivalent when compared to exposure throughout gestation. In males, ethanol exposure during the 1st, 2nd or 3rd trimester equivalent did not significantly reduce LTP in the DG. In females, ethanol exposure during either the 1st or 2nd trimester equivalents did not impact LTP in early adulthood, but following exposure during the 3rd trimester equivalent alone, LTP was significantly increased in the female DG. These results further exemplify the disparate effects between the ability to elicit LTP in the male and female brain following perinatal ethanol exposure (PNEE).
Keywords: dentate gyrus; ethanol; fetal alcohol spectrum disorders; fetal alcohol syndrome; hippocampus; long-term potentiation; synaptic plasticity; vulnerability period

1. Introduction

Fetal Alcohol Spectrum Disorders (FASD) occur when alcohol is consumed during pregnancy and result in abnormal brain development and long lasting deficits in cognitive function [1,2]. Recent reports have estimated the prevalence of FASD in young school children in the USA and some Western European countries to be as high as 2%–5% [3]. It is thought that FASD is the most common cause of mental retardation and birth defects in the United States [1]. The most severe disorder that results from ethanol exposure during pregnancy is Fetal Alcohol Syndrome (FAS). FAS is a disorder characterized by facial dysmorphologies such as midfacial hypoplasia, wide spaced eyes and a smooth philtrum, growth retardation and central nervous system (CNS) dysfunction resulting in cognitive, motor and behavioural problems [1]. Since FAS was first defined in the 1970’s [2,4] it has been realized that the extent of the damage caused by ethanol can vary due to the timing, frequency and volume of ethanol consumed, as well as the genetics and metabolism of the mother, leading to a wide variability in the severity and symptoms. The disorders that result from prenatal ethanol exposure are now grouped under the umbrella term FASD, which encompasses children who show various forms of central nervous system dysfunction.

The brain is not a static organ, but it can change both physically (structural plasticity) and functionally (synaptic plasticity) depending on environment and experience. In particular, it is believed that alterations in hippocampal synaptic plasticity (i.e., changes in the strength of neuronal connections in the hippocampus) may be involved in information storage and hippocampus-dependent learning and memory [5,6]. One form of synaptic plasticity is long-term potentiation (LTP), which corresponds to the strengthening of a synapse and is characterized by an increase in the size of an evoked postsynaptic potential or current in response to the same stimulus [6]. This results in long lasting neuronal changes involving protein expression and activation and an overall increase in synaptic transmission [7]. It is postulated that in the hippocampus LTP and its counterpart long-term depression (LTD), work together to refine and sculpt memories [8].

The hippocampus is one of the major brain regions affected in FASD [9], and both animals [10–18] and children [19–22] that were exposed to ethanol during the period of brain development show learning and memory impairments. Previous studies from our laboratory and others have also indicated that prenatal ethanol exposure can cause long lasting deficits in hippocampal synaptic plasticity [14,15,23–27]. In particular, our laboratory has shown that LTP is decreased in the dentate gyrus (DG) of both adolescent [23] and young adult [24,28] males that were exposed to ethanol throughout gestation.

It is well known that the sensitivity of the CNS to the effects of ethanol varies throughout the perinatal period, with specific cell types being more sensitive during certain stages of development [29–32]. For example, previous studies from our laboratory have shown that the effects of perinatal ethanol exposure (PNEE) on oxidative stress in the young adult rat hippocampus are more
pronounced when exposure occurred during all three-trimester equivalents [33] as compared to the 1st and 2nd trimester equivalents combined [34]. This indicates that the 3rd trimester equivalent (which corresponds to the brain growth spurt in humans; [31,35]) might be particularly sensitive to the effects of ethanol on oxidative stress. However, exposure during the 1st and 2nd trimester equivalents combined (i.e., throughout gestation in rats) is enough to induce a long-lasting impairment in hippocampal synaptic plasticity that can be detected in early adulthood [24,28]. Furthermore, a recent large-scale clinical study has shown that drinking during the 1st trimester (when many women are not yet aware of their pregnancy) has a strong association with signs of alcohol damage to the fetus [36]. A different study revealed that while pregnant women can eliminate ethanol from their blood faster during the 2nd trimester, this teratogen is cleared from the amniotic fluid at a much slower rate during this period, indicating that the fluid may act as an ethanol reservoir [37]. Therefore, it is of particular relevance to further characterize the effects that ethanol exposure might have during particular periods of brain development.

The aim of this study was to determine whether ethanol exposure during a specific trimester equivalent renders the DG of the hippocampus more vulnerable to deficits in LTP later on, when animals reach early adulthood. As previous research from our laboratory has demonstrated the existence of sexually dichotic effects of PNEE on LTP [23,24,28], the present study evaluated how ethanol-exposure during the 1st, the 2nd, or the 3rd trimester equivalent affected LTP both in the male and female young adult DG. Identifying intervals during the period of brain development of enhanced vulnerability to the effects of ethanol exposure on synaptic plasticity may further elucidate the mechanisms underlying the wide range of cognitive manifestations that are associated with FASD.

2. Results and Discussion

2.1. Effects of Ethanol Exposure on Body Weight and Litter Size

Weight data was taken from pregnant dams on gestational days (GDs) 1, 7, 14 and 21. The percentage weight gain over the course of pregnancy did not differ between perinatal conditions \(F(6, 21) = 1.7, p = 0.17\). However, there was a significant effect of perinatal condition on litter size \(F(6, 21) = 2.8, p = 0.04\), with 1st trimester pair-fed animals having larger litters as compared to 1st trimester ethanol-exposed animals \((p < 0.05)\). These results are summarized in Table 1.

Offspring weight was determined during the lactation period on postnatal days (PNDs) 2, 6, 10, 14, 18 and 22 to determine whether perinatal condition altered offspring weight gain. A repeated measures analysis of variance (ANOVA) revealed that there was no significant effect of sex \(F(6, 37) = 2.0, p = 0.99\), and therefore, data from both males and females were combined and a repeated measures ANOVA for perinatal condition (\textit{ad libitum}, pair-fed1, pair-fed2, pair-fed3, ethanol1, ethanol2, ethanol3) revealed a significant effect of perinatal condition \(F(36, 196) = 3.0, p < 0.0001\). Further post-hoc analysis revealed that at PND 2, 1st trimester pair-fed animals weighed significantly less than both \textit{ad libitum} \((p < 0.05)\) and 1st trimester ethanol-exposed animals \((p < 0.001)\). There were no significant differences in weight between PNDs 6 and 14 for all conditions. At PND 18, 3rd trimester pair-fed animals weighed significantly more than both 1st trimester pair-fed animals \((p < 0.05)\) and 2nd trimester pair-fed animals \((p < 0.01)\). This difference remained at PND 22, with 3rd trimester
pair-fed animals still weighing significantly more than 1st trimester pair-fed animals \((p < 0.001)\) and 2nd trimester pair-fed animals \((p < 0.01)\). These results are summarized in Table 2.

**Table 1.** Effect of perinatal ethanol exposure on dam weight gain during pregnancy and litter size. There were no significant differences in weight gain over pregnancy between perinatal conditions. 1st trimester pair-fed animals had significantly more pups than 1st trimester ethanol exposed animals, but all other groups had comparable litter sizes. Results are expressed as means ± standard error of the mean (SEM). Results are considered statistically significant if \(p < 0.05\). * \(p < 0.05\), as compared to 1st trimester ethanol exposed dams.

<table>
<thead>
<tr>
<th></th>
<th>% weight gain during pregnancy</th>
<th>Number of pups per litter</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ad libitum</td>
<td>61.6 ± 11.3</td>
<td>13.7 ± 2.3</td>
</tr>
<tr>
<td>Ethanol 1st</td>
<td>74.4 ± 3.8</td>
<td>12.8 ± 1.3</td>
</tr>
<tr>
<td>Pair-fed 1st</td>
<td>66.3 ± 9.3</td>
<td>18 ± 1.7 *</td>
</tr>
<tr>
<td>Ethanol 2nd</td>
<td>51.1 ± 3.2</td>
<td>15.3 ± 0.5</td>
</tr>
<tr>
<td>Pair-fed 2nd</td>
<td>63.1 ± 4.3</td>
<td>13.0 ± 0.6</td>
</tr>
<tr>
<td>Ethanol 3rd</td>
<td>65.4 ± 4.3</td>
<td>14.0 ± 0.8</td>
</tr>
<tr>
<td>Pair-fed 3rd</td>
<td>69.7 ± 5.2</td>
<td>15.6 ± 0.2</td>
</tr>
</tbody>
</table>

**Table 2.** Effect of perinatal ethanol exposure on offspring body weight. A repeated measures ANOVA revealed that there was no significant effect of sex and therefore data from both males and females were combined. A significant main effect of perinatal condition was observed (see text for statistical details). Results are expressed as means ± SEM. Results are considered statistically significant if \(p < 0.05\). * \(p < 0.05\) compared to *ad libitum* controls and 1st trimester ethanol exposed animals (postnatal day (PND) 2). # \(p < 0.05\) compared to 3rd trimester pair-fed animals (PND 18). $$$ \(p < 0.01\) compared to 3rd trimester pair-fed animals (PND 22).

<table>
<thead>
<tr>
<th></th>
<th>Ad libitum</th>
<th>Ethanol-exposed</th>
<th>Pair-fed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight (g)</td>
<td>1st</td>
<td>2nd</td>
<td>3rd</td>
</tr>
<tr>
<td>PND2</td>
<td>8.0 ± 0.3</td>
<td>8.3 ± 0.3</td>
<td>7.5 ± 0.2</td>
</tr>
<tr>
<td>PND5</td>
<td>15.0 ± 0.5</td>
<td>14.6 ± 0.4</td>
<td>14.5 ± 0.4</td>
</tr>
<tr>
<td>PND10</td>
<td>23.6 ± 0.8</td>
<td>23.3 ± 0.6</td>
<td>23.2 ± 0.7</td>
</tr>
<tr>
<td>PND14</td>
<td>33.3 ± 1.1</td>
<td>32.8 ± 0.9</td>
<td>33.4 ± 0.9</td>
</tr>
<tr>
<td>PND18</td>
<td>43.7 ± 1.4</td>
<td>41.8 ± 1.2</td>
<td>43.2 ± 1.2</td>
</tr>
<tr>
<td>PND22</td>
<td>60.4 ± 1.9</td>
<td>60.4 ± 1.7</td>
<td>59.8 ± 1.7</td>
</tr>
</tbody>
</table>

When animals reached experimental age (PNDs 55–70), a significant main effect of sex was observed \([F(1, 101) = 1481.6, p < 0.0001]\), with males being significantly heavier than females \((p < 0.001)\) and data from males and females was subsequently analyzed separately. Nevertheless, there was no significant main effect of perinatal condition on body weight at experimental age both in males \([F(6, 50) = 2.05, p = 0.08]\) and females \([F(6, 51) = 1.07, p = 0.39]\). These results are summarized in Table 3.
**Table 3.** Effect of perinatal ethanol exposure on offspring body weight at experimental age. When reaching early adulthood, males weighed significantly more than females and therefore their weights were analyzed separately. There were no significant effects of perinatal condition on body weight in either males or females (see text for statistical details). Results are expressed as means ± SEM. Results are considered statistically significant if $p < 0.05$.

<table>
<thead>
<tr>
<th></th>
<th>Weight (g)</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Ad libitum</strong></td>
<td>1st</td>
<td>417.5 ± 5.2</td>
<td>281.1 ± 11.4</td>
</tr>
<tr>
<td></td>
<td>2nd</td>
<td>400.0 ± 7.0</td>
<td>267.0 ± 6.1</td>
</tr>
<tr>
<td><strong>Ethanol-exposed</strong></td>
<td>3rd</td>
<td>408.4 ± 4.7</td>
<td>257.1 ± 5.4</td>
</tr>
<tr>
<td><strong>Pair-fed</strong></td>
<td>1st</td>
<td>413.0 ± 1.6</td>
<td>263.1 ± 3.0</td>
</tr>
<tr>
<td></td>
<td>2nd</td>
<td>413.2 ± 3.4</td>
<td>269.7 ± 4.0</td>
</tr>
<tr>
<td></td>
<td>3rd</td>
<td>421.6 ± 4.1</td>
<td>272.1 ± 5.7</td>
</tr>
</tbody>
</table>

2.2. Intoxication Levels

Peak blood alcohol concentrations (BACs) were measured from blood taken two hours after the dark cycle commenced on GD 10 or GD 20 for 1st or 2nd trimester equivalent exposure, respectively. Blood taken 2 h following the last feeding of the ethanol diet on PND 10 was used to measure peak BAC levels for 3rd trimester equivalent exposure. The BACs for 1st, 2nd and 3rd trimester equivalent exposed animals were 91.6 mg/dL, 94.3 mg/dL, and 255.1 mg/dL, respectively. These results are in agreement with the literature, with the gavage model (3rd trimester equivalent) producing considerably higher BACs than the prenatal liquid diet exposure model [33,38,39]; for review see [40].

2.3. Effects of Ethanol Exposure on Basal Synaptic Transmission and Post-Tetanic Potentiation

To determine whether ethanol exposure during a specific trimester equivalent affected basal synaptic transmission, input/output (I/O) analysis was performed. In all animals, the slope of the field excitatory post-synaptic potential (fEPSP) significantly increased with increasing stimulation [repeated measures ANOVA: $F(4, 288) = 287.2, p < 0.0001$]. Perinatal condition had no significant effect on I/O function repeated measures ANOVA: $[F(6, 72) = 0.51, p = 0.80]$, regardless of sex $[F(1, 72) = 0.005, p = 0.94]$ (data not shown).

To assess the effects of ethanol exposure during specific trimester equivalents on short-term plasticity, post-tetanic potentiation (PTP; defined as the first minute following stimulation) was assessed. In males, a one-way ANOVA revealed a significant main effect of perinatal condition $[F(6, 47) = 2.78, p = 0.02]$. Post-hoc analyses showed that PTP was significantly reduced in males exposed to ethanol during the 1st trimester equivalent when compared to males exposed to ethanol during the 3rd trimester equivalent ($p < 0.05$). In females, a one-way ANOVA revealed no significant effects of perinatal condition $[F(6, 45) = 1.73, p = 0.14]$. Additionally, the results for each trimester equivalent were analysed individually. In males, there was a significant main effect of perinatal condition on PTP in the 1st trimester equivalent exposure group $[F(2, 20) = 4.05, p = 0.03]$, but post-hoc analysis showed only a trend towards a decrease in the ethanol-exposed animals when
compared to the *ad libitum* controls (*p* = 0.06). There was no significant main effects of perinatal condition on PTP in the 2nd \( F(2, 20) = 1.28, p = 0.3 \) or 3rd \( F(2, 17) = 1.3, p = 0.3 \) trimester equivalent exposure groups. In females, there was no significant main effect of perinatal condition on PTP in the 1st \( F(2, 18) = 0.3, p = 0.7 \), 2nd \( F(2, 18) = 2.0, p = 0.17 \) or 3rd \( F(2, 19) = 2.4, p = 0.12 \) trimester equivalent exposure groups.

### 2.4. Effects of Ethanol Exposure on Young Adult DG LTP

We have previously established that PNEE young adult males (PNDs 55–70) show a significant reduction in LTP in the DG, whereas young adult female animals do not show such deficits \[24,28\]. The present study aimed to determine whether LTP is affected differently if exposure to ethanol occurs only during a specific trimester equivalent in both male and female offspring.

In male offspring, a one-way ANOVA for perinatal condition (control, pair-fed1, pair-fed2, pair-fed3, ethanol1, ethanol2, ethanol3) revealed that there was no significant effect of perinatal condition on LTP \( F(6, 53) = 0.601, p = 0.728 \); Figure 1). Additionally, when the results for each trimester equivalent were analysed individually, 3 out of 8 males that were exposed to ethanol during the 1st trimester equivalent alone showed less than 30% LTP, whereas 2 out of 8 males that were exposed to ethanol during the 2nd trimester equivalent alone showed less than 30% LTP (data not shown). This is in contrast to *ad libitum* control males, pair-fed males and 3rd trimester equivalent ethanol-exposed males, where no animals showed less than 30% LTP. However, additional statistical analysis of each individual trimester equivalent failed to reveal any significant effects of perinatal condition during either the 1st \( F(2, 20) = 1.63, p = 0.22 \), 2nd \( F(2, 20) = 0.96, p = 0.40 \) or 3rd \( F(2, 19) = 0.12, p = 0.89 \) trimester equivalent exposure groups. These results indicate that overall, ethanol exposure during a single trimester equivalent is not enough to cause reliable long-term deficits in LTP in the male DG.

In females, a one-way ANOVA (control, pair-fed1, pair-fed2, pair-fed3, ethanol1, ethanol2 or ethanol3) showed no significant effect of perinatal condition \( F(6, 45) = 1.45, p = 0.22 \) (Figure 2). Interestingly, if the results for each trimester equivalent were individually analyzed, no significant effects of perinatal condition were obtained for either the 1st \( F(2, 19) = 0.21, p = 0.81 \) or 2nd \( F(2, 18) = 0.09, p = 0.92 \) trimester equivalent exposure groups, but a significant effect was revealed for the 3rd trimester equivalent exposure group \( F(2, 18) = 3.73, p < 0.05 \). *Post-hoc* analyses further demonstrated that when ethanol exposure occurred during the third trimester equivalent, ethanol exposed females have increased LTP as compared to both *ad libitum* control \( p < 0.05 \) and pair-fed \( p < 0.05 \) females.
Figure 1. The effects of perinatal ethanol exposure (PNEE) during specific trimester equivalents on long-term potentiation (LTP) in the dentate gyrus (DG) of young adult male rats. PNEE during either the 1st (A), 2nd (B), or 3rd (C) trimester equivalents does not result in a significant reduction in DG LTP. (D) Summary of LTP results calculated by assessing the initial phase of the excitatory post-synaptic potentiation (EPSP) slope (10%–80%) at 55–60 min post-theta burst stimulation (TBS). Results are presented as means ± SEM and were considered statistically significant when \( p < 0.05 \).

Figure 2. The effects of PNEE during specific trimester equivalents on LTP in the DG of young adult female rats. PNEE during either the 1st (A) or 2nd (B) trimester equivalents does not result in a significant reduction in DG LTP. (C) PNEE during the 3rd trimester equivalent significantly increased LTP in females when compared to their respective ad libitum (\( p < 0.01 \)) and pair-fed (\( p < 0.01 \)) controls (see text for additional details on the statistical analysis). (D) Summary of LTP results calculated by assessing the initial phase of the EPSP slope (10%–80%) at 55–60 min post-TBS. Results are presented as means ± SEM and were considered statistically significant when \( p < 0.05 \).
2.5. Discussion

Previous research from our laboratory has indicated that ethanol exposure throughout gestation (i.e., 1st and 2nd trimester equivalents in rats) causes long-lasting deficits in DG LTP in young adult males (PNDs 55–70) [23,24,28]. Females on the other hand, show an enhanced capacity for DG LTP in adolescence (PNDs 30–35) [23], a phenomenon which disappears in early adulthood (PNDs 55–70), where LTP levels are comparable to those of control females [24,28].

In this study we examined which periods during brain development are more susceptible to ethanol exposure with regards to the ability to elicit LTP in the DG of the hippocampus. Not surprisingly, we found that in males the long-term effects of ethanol on DG synaptic plasticity were considerably less robust when exposure was limited to an individual trimester equivalent (present study) as compared to continuous exposure throughout gestation [23,24,28]. In fact, for all three exposure periods (i.e., either 1st, 2nd, or 3rd trimester equivalents), there were no statistically significant differences in the ability to elicit LTP among ethanol-exposed, pair-fed, and ad libitum control males. In a 1990 study conducted by Tan et al. [41], an in vitro examination of LTP in the CA1 region of the hippocampus showed that when alcohol exposure (through a liquid diet with either 35% or 17.5% ethanol) occurred between GDs 8 and 22 (i.e., late 1st trimester–2nd trimester exposure) no changes in LTP were observed [41]. These results, in combination with the results from the present study, indicate that alcohol exposure during the second trimester does not reduce LTP in either the DG or CA1 sub-regions of the hippocampus. These results are also in line with previous studies that have shown that ethanol exposure during the 3rd trimester equivalent does not affect synaptic plasticity in the CA1 [42,43] and that ethanol exposure only during the 1st, 2nd, or 3rd trimester equivalent does not result in deficits in spatial navigation [42,44]. Overall, these results indicate that ethanol exposure during a limited period of brain development is not enough to robustly alter hippocampal synaptic plasticity (and possibly hippocampal-sensitive behaviours; [42,44]) when animals reach early adulthood.

Of note, a degree of variability was observed in the animals that were exposed to ethanol during either the 1st or 2nd trimester equivalents, with 2–3 out of 8 males that were exposed to ethanol during either the 1st or 2nd trimester equivalent alone showing less than 30% LTP. In contrast, no such variability was noted among the males that were exposed to ethanol during the 3rd trimester equivalent alone. The reasons underlying the variability that was obtained following exposure during the 1st or 2nd trimester equivalents are likely related with the different modes of ethanol administration that were used for the 1st and 2nd trimester equivalent groups (i.e., voluntary consumption of a liquid diet containing ethanol by the pregnant dam) and the 3rd trimester equivalent group (i.e., intragastric intubation of the offspring with a pre-determined amount of ethanol).

Indeed, due to the fact that in rats the third trimester equivalent of brain development occurs postnatally (from PNDs 1 to 10), a liquid diet cannot be administered across all three trimester equivalents. While it is possible to administer the liquid diet to lactating mothers during the third trimester equivalent, it is uncertain how much ethanol crosses into the breast milk, and whether the pups will suckle if ethanol is present in the milk (for review see [40]). Previous studies have used either the vapour inhalation method or the gavage (i.e., gastric intubation) method to expose rat pups during the third trimester equivalent and while both methods are associated with high BACs, the gavage method better models the route of ethanol consumption in humans (i.e., oral administration),
and is therefore preferred. Additionally, the gavage model has the added advantage of controlling for the timing of exposure and dosing of ethanol, thus significantly reducing variability within groups (for review see [40]). On the other hand, while the liquid diet model usually produces BACs that more closely reflect those obtained with moderate drinking (i.e., more representative of the vast majority of the human population that consumes alcohol during pregnancy [45]), it is also associated with an increased variability when compared with the gavage model. For example, the time of exposure, the daily period of exposure, or the time during the day when the highest BAC is reached may differ dramatically among pregnant dams that have free access to an ethanol liquid diet and these factors may differentially affect their offspring (for review see [40]). Thus, it is not surprising that higher group variability was observed in the 1st and 2nd trimester equivalent groups. Future studies utilizing the gavage model for all periods of exposure may provide a better understanding of how LTP is affected during each trimester equivalent alone.

Interestingly, exposure to a high BAC (255.1 mg/dL) during the 3rd trimester equivalent also failed to induce significant changes in LTP in the DG of young adult male rats. This may be related to the fact that in this case, exposure to ethanol occurred after the population of DG precursor cells (i.e., the radial glia cells) had been generated. Indeed, this population of precursor cells is formed during the 2nd trimester equivalent [46,47], and therefore if animals are only exposed to ethanol during the 3rd trimester equivalent, this process is not affected. On the other hand, while increased cell proliferation is known to occur in the DG during the 3rd trimester equivalent (i.e., the period of brain growth spurt) [35] and exposure to ethanol during this period may disrupt this process by increasing apoptosis of the newly generated cells, previous studies from our laboratory indicated that ethanol exposure during the three trimester equivalents does not impact the rate of cell proliferation and in fact increases the rate of neuronal differentiation in the adult DG [38,39]. Thus, despite a potential ethanol-induced increase in apoptosis during the third trimester, it is possible that the size of the DG granule cell population can be restored due to a compensatory mechanism of ethanol-induced increased neuronal differentiation [38,39]. This might explain why, by the time LTP was examined (i.e., when animals reached adulthood), no significant differences in LTP were detected between control and ethanol-exposed animals. On the other hand, when exposure occurs during gestation (and concomitant with the generation of DG precursor cells), even though lower BACs are achieved, the DG population of precursor cells may be intrinsically affected by ethanol and not be able recover. In this case, even though the rate of adult hippocampal neurogenesis is not affected by ethanol exposure [38,39], the precursor cells responsible for producing the new granule cells may be intrinsically damaged and hence all daughter cells will also be potentially affected and unable to function properly. This may then explain why significant deficits in DG LTP are observed when ethanol exposure via an ethanol-containing liquid diet occurs throughout gestation (i.e., 1st and 2nd trimester equivalents combined) [24,28].

As well as LTP, PTP was also examined in these male offspring. PTP describes an enhancement of transmitter release lasting for minutes after HFS-induced LTP of synapses in the MPP of the DG. This enhancement is largely due to an increase in intracellular Ca\(^{2+}\) concentration in the pre-synaptic terminal during the stimulus trains. The increased residual Ca\(^{2+}\) combines with the Ca\(^{2+}\) influx that is triggered by the following action potential to enhance neurotransmitter release [48]. Our findings indicate that males exposed to ethanol during the 1st trimester equivalent have a decreased capacity for
PTP compared to *ad libitum* control males which may indicate differences in neurotransmitter release probabilities. However, this difference does not cause significant changes in the ability to elicit LTP (Figure 1). These presynaptic differences and their potential effects on neurotransmission and behaviour should be explored in future studies.

Previous studies have shown that PNEE females show increased LTP during adolescence (PNDs 30–35) [23] and no differences in LTP during early adulthood (PNDs 55–70) [24,28]. It is therefore not surprising that PNEE during either the 1st or the 2nd trimester equivalent alone did not impact LTP in the young adult female DG (Figure 2). However, when PNEE occurred during the 3rd trimester equivalent alone, a significant increase in DG LTP was detected when compared to *ad libitum* control and pair-fed females (Figure 2). This increase is similar to that observed in the DG of adolescent females that were exposed to ethanol throughout gestation (i.e., 1st and 2nd trimester equivalents combined) [23]. Reasons for this increase are currently unknown but may be related to a dysregulation of estrogen levels with PNEE. Indeed, PNEE females exhibit an increased hypothalamic-pituitary-adrenal (HPA) sensitivity to estrogen, and estrogen levels are higher during proestrous in PNEE females compared to controls [49]. Furthermore, a recent study suggests that PNEE may lead to increased basal estrogen levels [50] and high levels of this hormone are known to increase LTP [51,52], possibly explaining the increase in DG LTP observed in PNEE females. Why this enhancement is only observed after ethanol exposure during the 3rd trimester is currently unknown, but may be related to the fact that estrogen does not begin to be produced in the ovaries until PND5 (i.e., during the 3rd trimester equivalent) [53]. Perhaps the most striking result is that by examining ethanol exposure during distinct periods of brain development we have uncovered a defined time window during which exposure to ethanol results in enhanced LTP in the young adult female DG.

Of note, while enhanced LTP is often associated with increased learning and memory, this is not always the case. In fact, impairments in spatial performance have been accompanied by increased LTP in the CA1 [54], and Jeffery *et al.* (1995) found a negative correlation between magnitude of LTP in the DG and spatial memory performance [55]. Therefore, no extrapolations should be made with regards to the possible effects of ethanol exposure during the 3rd trimester equivalent and learning and memory abilities in females. In fact, learning and memory deficits are commonly observed in females following PNEE, particularly with 3rd trimester exposure [11,12,17].

### 3. Experimental Section

#### 3.1. Animals and Breeding

All animal procedures were performed in accordance with the University of Victoria and the Canadian Council for Animal Care policies.

Four Male (300–350 g) and 30 virgin female (250–275 g) Sprague-Dawley rats were obtained from Charles River Laboratories (Quebec, Canada). Females were housed in pairs and breeding males were housed individually in clear polycarbonate cages (46 × 24 × 20 cm) with Carefresh contact bedding (Absorption Corp., Bellingham, WA, USA). The room was maintained on a 12-h light:dark cycle (lights on at 7 a.m.) with constant humidity and temperature (22 ºC). Following an acclimation period in the unit for at least one week, females and males were housed together and a vaginal smear using
0.9% sodium chloride (NaCl) was performed at the beginning of each light cycle to determine pregnancy. An Olympus Microscope with a 10× objective (Olympus CX21, Center Valley, PA, USA) was used to detect the presence of sperm, which indicated GD 1. The female was immediately removed to a private container supplied with nesting material and placed in one of seven prenatal treatment groups (Figure 3).

**Figure 3.** Experimental Timeline. On GD1 pregnant dams were assigned to one of seven treatment groups: *Ad libitum* controls, 1st trimester equivalent ethanol exposure, 1st trimester equivalent pair-fed, 2nd trimester equivalent ethanol exposure, 2nd trimester equivalent pair-fed, 3rd trimester equivalent ethanol exposure, or 3rd trimester equivalent pair-fed (see text for a detailed explanation of the various groups). Blood samples to assess BAC were taken on GD 10 for the 1st trimester equivalent exposure condition, GD20 for the 2nd trimester equivalent exposure condition, and PND 10 for the 3rd trimester equivalent exposure condition. When animals reached experimental age (PNDs 55–70) they were used for *in vivo* electrophysiological experiments to examine LTP in the DG of the hippocampus. Basal recordings were first obtained by administering a pulse (0.12 ms in duration) at 0.067 Hz (pre-stimulation). Once a stable baseline was observed for at least 15 min, LTP was induced by applying TBS consisting of 10 bursts of 5 pulses at 400 Hz with an inter-burst interval of 200 ms, which was repeated 4 times at 30 s intervals. The pulse duration was changed to 0.25 ms during TBS. Following TBS, baseline stimulation resumed for 60 min (post-stimulation).

**Abbreviations:** Blood alcohol concentration (BAC); Dentate gyrus (DG); Gestational day (GD); Long-term potentiation (LTP); Postnatal day (PND); Theta burst stimulation (TBS).

### 3.2. Prenatal Diets

On GD1 pregnant dams were randomly assigned to one of seven groups (4 dams per treatment group).

**1st or 2nd trimester equivalent ethanol exposure—*Ad libitum* access to a liquid diet containing 35.5% ethanol derived calories during either the 1st trimester equivalent (GDs 1–10) or the 2nd trimester equivalent (GDs 11–21) of pregnancy. Ethanol dams were gradually introduced to the liquid**
diet over a three-day period (GDs 1–3 or GDs 11–13). On GD1 or 11, one third of the ethanol diet was combined with two thirds of the pair-fed diet (see below), on GD2 or 12, two thirds of the ethanol diet was combined with one third of the pair-fed diet and on GD 3 or 13, 100% of the ethanol diet was supplied to the dam. Dams exposed to ethanol liquid diet during the 1st trimester equivalent received regular chow (Lab Diets 5001, LabDietS, Richmond, IN, USA) from GD 11 onwards. Dams exposed to ethanol liquid diet during the 2nd trimester consumed regular chow from GDs 1 to 10 and then were switched back to a regular chow diet on the final day of pregnancy (GD 22).

1st or 2nd trimester equivalent pair-fed diet—The pair-fed groups received a liquid diet with maltose-dextrin isocalorically substituted for the ethanol-derived calories. This liquid diet was not provided ad libitum. To control for stress and malnutrition of the ethanol-exposed animals, pair-fed groups received the same amount of food in g/kg/day as their matched ethanol-exposed dams. As above, dams in the 1st trimester equivalent exposure group were given the pair-fed liquid diet between GDs 1 and 10. On GD 11, animals were placed back on a regular chow diet for the remainder of the pregnancy. Dams in the 2nd trimester equivalent exposure group received regular rat chow from GDs 1 to 10 of gestation and were switched to the pair-fed liquid diet between GDs 11 and 21. On GD 22 the dams were switched back to the chow diet.

All liquid diets were given to the animals two hours prior to the beginning of the dark phase each day of the pregnancy. This was done to ensure there were no shifts in the circadian rhythm [56]. When liquid diet bottles were replaced, the bottle from the previous day was weighed to determine the amount of liquid consumed each day. On average animals consume approximately 90–100 g of diet/day. Females were weighed on GDs 1, 7, 14 and 21.

Liquid diets were obtained from Dyets (Bethlehem, PA, USA) where they are sold as Weinberg/Keiver high protein liquid diet-control (no. 710109) for the pair-fed diet and Weinberg/Keiver high protein liquid diet-experimental (no. 710324) for the ethanol diet. These liquid diets have been nutritionally fortified to ensure that adequate nutrition is provided to the pregnant rats. The ethanol diet contains 1.0 kcal/mL, of which 16.4% are fat derived, 23% are derived from carbohydrate, 25.1% are derived from protein, and 35.5% are ethanol derived. The pair-fed diet is isocaloric, where the carbohydrate-derived calories were increased to 58.5% to substitute for those derived from ethanol [57].

3rd trimester equivalent ethanol exposure—Pregnant females were left undisturbed throughout gestation. The day pups were born was considered PND 1. Pups received a dose of 4 g/kg/day of 12% (v/v) ethanol in milk solution between PNDs 4 and 10 (3rd trimester equivalent). Ethanol was dissolved in a nutritional milk solution similar in composition to rat milk [58] and supplemented with a specially formulated vitamin mix (Bio-Serv; Frenchtown, NJ, USA). Solutions were administered by intragastric intubation as previously described by our laboratory [33,37–39]. The solution was administered in two separate intubations 2 h apart. An additional feeding of pure milk solution was supplied to ethanol-exposed pups in the evening to counteract the inadequate nutrition, low birth weight and high mortality rate that can be observed with this model of postnatal ethanol exposure [33,38,39].

3rd trimester equivalent pair-fed diet—As above, pregnant females were left undisturbed throughout gestation. Between PNDs 4 and 10 (3rd trimester equivalent), pups received a dose of an iso-caloric and iso-volumic maltose-dextrin milk solution. Maltose-dextrin was dissolved in the same nutritional milk solution used to prepare the 3rd trimester equivalent ethanol diet (see above). The
solution was administered in two separate intubations 2 h apart. The pair-fed pups were sham-intubated during the third feeding, as extra feeding could cause an excess weight gain in these animals (reviewed in [40]).

**Ad libitum control**—Pregnant dams had *ad libitum* access to a regular chow diet (Lab Diets 5001, LabDiets, Richmond, IN, USA) throughout pregnancy. Pups were left undisturbed during the 3rd-trimester equivalent.

### 3.3. Blood Alcohol Concentration Assay

For all ethanol-fed dams a single tail blood sample was obtained on GD10 for 1st trimester equivalent exposure or GD20 for 2nd trimester equivalent exposure, approximately two hours after the beginning of the dark phase. For animals that were exposed to ethanol in the 3rd trimester, a tail blood sample was obtained from the pups on PND10, approximately two hours after the last feeding of ethanol-containing diet of the day. Blood was collected in a microcentrifuge tube and allowed to clot overnight at 4 °C. Samples were centrifuged the following day at 3000 g for 10 min and the serum (supernatant) was then stored at −20 °C until analysed. Analysis of BACs was performed using the Analox Alcohol Analyzer (Model AMI; Analox Instruments, Lunenberg, MA) and expressed as mg/dL of serum.

### 3.4. Litters and Weaning

Dams and pups were not disturbed during the initial 24–36 h post-partum to facilitate bonding. Litters were culled to ten pups on PND 2 and all animals were weighed on PNDs 2, 5, 10, 14, 18 and 22. Pups were weaned on PND22 and housed in pairs (based on sex) in standard caging and left undisturbed until experimental age (PNDs 55–70) was reached (Figure 3).

### 3.5. In Vivo Electrophysiology

Male and female offspring were used for *in vivo* electrophysiology experiments of DG LTP between the ages of PNDs 55 and 70. To reduce any possible litter effects only two males and two females per litter were included in each experimental group. Eight animals per trimester equivalent, perinatal condition, and sex were used for these experiments. Female subjects were examined each day for at least five days before experimentation using the lavage technique where a vaginal smear using 0.9% NaCl was performed at the beginning of each light cycle to determine the estrous cycle. Females were not used for experimentation if they were in proestrous, where estrogen levels are the highest, as high levels of this hormone may enhance LTP and confound results [51,52].

Animals were anaesthetized with urethane (1.5 mg/kg, delivered intraperitoneally, i.p.) and placed on a Kopf stereotaxic apparatus. Body temperature was maintained at 37 ± 0.5 °C throughout the experiment with a grounded homeothermic temperature control unit (Harvard Instruments, MA, USA). Extracellular field potentials were recorded by inserting a 125 μm stainless-steel recording electrode into the hilus of the DG (3.5 mm anterior, 2.0 mm lateral to bregma; [59]) and a 125 μm monopolar stimulating electrode into the ipsilateral medial perforant path (7.4 mm anterior, 4.2 mm lateral to bregma; [59]). Stimulating and recording electrodes were lowered to elicit a maximal response and the
stimulation required to induce a 1–2 mV population spike was determined. Prior to basal recordings input/output (I/O) function was assessed by stimulating the tissue with increasing pulse widths (0.04, 0.08, 0.12, 0.16, 0.20 and 0.24 ms in duration) at 0.067 Hz, repeated 5 times at each pulse width. Basal recordings were then obtained by administering a pulse (0.12 ms in duration) at 0.067 Hz. Once a stable baseline was observed for at least 15 min, LTP was induced by applying TBS consisting of 10 bursts of 5 pulses at 400 Hz with an inter-burst interval of 200 ms that was repeated 4 times at 30-s intervals. The pulse duration was changed to 0.25 ms during TBS. Following TBS, baseline stimulation resumed for 60 min (Figure 3), as previously described by us [23,24,28]. Animals were then sacrificed by rapid decapitation.

Signals from the DG were collected on custom-made software (Lee Campbell; Getting Instruments). Signals were amplified (Getting Instruments), filtered (1–3 Hz) and digitized at 5 kHz. For analysis the slope of the rising phase of the fEPSP was used to determine alterations in the level of synaptic efficacy. All fEPSP slope data are presented as the mean percent change from the pre-conditioning baseline ± SEM.

3.6. Statistical Analyses

Statistical analysis was performed using the Statistica 7.1 analytical software (StatSoft Inc., Tulsa, OK, USA). All data are presented as means ± SEM. A one-way ANOVA was used to determine the effect of perinatal condition (Ethanol-1, Ethanol-2, Ethanol-3, Pair-fed-1, Pair-fed-2, Pair-fed-3 or Ad libitum control) on weight gain across pregnancy and litter size. A repeated measures ANOVA for perinatal condition (Ethanol-1, Ethanol-2, Ethanol-3, Pair-fed-1, Pair-fed-2, Pair-fed-3 or Ad libitum control) was used to analyze pup weights taken on PNDs 2, 6, 10, 14, 18 and 22. A two-way ANOVA for sex (male or female) and perinatal condition (Ethanol-1, Ethanol-2, Ethanol-3, Pair-fed-1, Pair-fed-2, Pair-fed-3 or Ad libitum control) was used to analyze weights at experimental age (PNDs 55–70). A significant main effect of sex was obtained at this age \([F(1, 101) = 1481.6, p < 0.0001]\), with males weighing significantly more than females \((p < 0.001)\). Therefore, male and female data were subsequently analysed separately using one-way ANOVAs.

A repeated measures ANOVA for perinatal condition (Ethanol-1, Ethanol-2, Ethanol-3, Pair-fed-1, Pair-fed-2, Pair-fed-3 or Ad libitum control) was used to analyze I/O data. LTP data were analyzed by assessing the initial phase of the EPSP slope (10%–80%) at 55–60 min post-TBS. Male and female LTP data were analysed separately as previous studies have revealed significant differences between PNEE males and females with regards to DG LTP [24,28]. One-way ANOVAs were used to determine the effect of perinatal condition (Ethanol-1, Ethanol-2, Ethanol-3, Pair-fed-1, Pair-fed-2, Pair-fed-3 or Ad libitum control) on PTP and LTP in both males and females. Additionally, PTP and LTP data from each trimester equivalent was individually analyzed with one-way ANOVAs for perinatal condition (Ethanol, Pair-fed, and Ad libitum control). Post-hoc analyses were conducted using Tukey’s test. A \(p\) value < 0.05 was considered to be statistically significant.

4. Conclusions

Results indicate that under the experimental conditions employed in this study, exposure to ethanol during restricted periods of brain development is not as detrimental for DG LTP later in adulthood as a
more prolonged ethanol exposure through multiple trimester equivalents of brain development. Indeed, and as one might expect, when ethanol exposure occurs only during one of the trimester equivalents, less robust effects on LTP are observed. Additionally, these results extend previous findings of sex differences with PNEE and indicate that the capacity to elicit DG LTP is differentially affected by ethanol in the male and female hippocampus.

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Conflict of Interest

The authors report no conflict of interest.

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The impact of raising a child with FASD upon carers: findings from a mixed methodology study in the UK
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The impact of raising a child with FASD upon carers: findings from a mixed methodology study in the UK

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Abstract
Research suggests that caring for a child with Fetal Alcohol Spectrum Disorders (FASD) creates unique challenges for carers. To investigate this, three focus groups and education sessions, attended by 66 people, were held in the UK. Knowledge about FASD and its impact on families was evaluated using the focus groups, the Parental Stress Index and knowledge questionnaire. Eight broad themes were identified from thematic analysis of the focus groups. The findings suggest more support is needed for carers of children with FASD, especially as carers grow older. The implication for current practice should be further evaluated in this group.

Keywords
FAS, FASD, carers, parenting, experiences

Introduction
Fetal Alcohol Syndrome (FAS) and Fetal Alcohol Spectrum Disorders (FASD) represent a range of preventable conditions, categorised by impairments to growth, facial features and neurological deficits that can lead to ongoing difficulties with behaviour, learning and...
day-to-day function. This damage is caused by maternal use of alcohol during pregnancy (BMA Board of Science, 2007). FAS and FASD are not always easily recognised (Chandresenna, Mukherjee and Turk, 2009; Mukherjee, Hollins and Turk, 2006) and evidence suggests that the rates of both of these conditions are increasing in the UK (May et al, 2006, 2007, 2009; Petkovic et al, 2010). Thus, the most recognisable part of the spectrum represents only a small proportion of the overall group (BMA Board of Science, 2007; Mukherjee, Hollins and Turk, 2006).

While the precise prevalence of FASD in the UK is currently unknown, studies which have looked at reported clinical cases in hospital episode statistics suggest that these rates are underestimated (Morleo et al, 2011), with a recent study of children in care suggesting that the rates in this population were possibly as high as a third of the whole group (Selwyn and Wijedesa, 2011). The impact of these factors on both children and their carers is currently unclear.

In order to understand and identify the needs of carers of children with FASD in the UK it was decided to conduct an exploratory study to help generate specific recommendations for practice and for further research.

There has been no apparent research published in the UK looking at the experiences or needs of carers of children with FASD. This contrasts with other parts of the world where several studies have been conducted. These have looked at the impact on carers, both biological and adopted, living with children affected by the condition. One such study interviewed adopted parents in the US state of Massachusetts, charting their experiences over time. It reported the journey of anticipation and expectation about adoption, described concerns over the amount of initial information provided about the child and documented frustration about not knowing how to manage a child with FASD and his or her associated behaviours, despite a sincere desire to support such children in realising their full potential (Granitas, 2004).

Concerns about parents’ ability to bring up children with FASD have been studied over several years, for example in a series of publications from Canada. The first of these investigated what it requires to be a good parent of such a child. In a series of interviews with 19 foster carers and adoptive parents, the study found that close collaboration and support from professionals and educators, alongside the development of functional management skills for the children (who were recognised as being unique in nature), correlated with better outcomes (Brown and Bednar, 2003).

In 2005 a study of 65 foster carers reported what made a successful placement of a child with FASD. It highlighted the need for support from professionals and extended family alongside structured environments with an understanding of the specific needs of the children (Brown, Sigvaldason and Bednar, 2005).

The final two studies from the series, published in 2007, involved 63 foster carers in Canada. The first investigated the motivation to provide foster care to a child with FASD and showed carers’ desire to achieve positive changes in the children, with helping the children to stay connected with their biological family as an important part of their decision to look after a child. The carers also reported that the children with FASD required different parenting skills than others previously fostered by the same families, even if those children also had special needs (Brown, Sigvaldason and Bednar, 2007). The second study looked at factors
that led to the breakdown of placements. The dominant reasons for placement disruption were: feeling taken for granted by professionals, the perceived possibility of burnout and the lack of information and support about how to manage difficult behaviours associated with FASD (Brown, Bednar and Sigvaldason, 2007).

These studies all point to the difficulties faced by carers and the unique parenting needs of children with FASD. Moreover, these difficulties appear to be often exacerbated by poor communication at the time of adoption. For example, one US study reported that the following factors increased stress in the adopting family: lack of knowledge on the part of professionals, blame directed at parents for the behaviour of their children and a failure by those same professionals to communicate sufficient information about potential difficulties to be faced by the parents (Caley, Winkelman and Mariano, 2009).

The impact on families who have adopted children with FASD has also been documented. Interviews conducted with eight US foster carers, who had fostered over 17 children with FASD, found that the difficulties with memory, lack of understanding of consequences, aggression and a lack of parental strategies to address these difficult behaviours caused the most problems for both the children and the host family (Gardner, 2000). The requirement for constant observation, the apparent absence of support from professionals and a lack of understanding in the wider community about the difficulties faced in child management were found to place excess stress on the carers’ relationships. In some cases this led to marital breakdown (Morissette, 2001). The impact on the physical and mental health of adults, as well as ongoing financial and emotional pressures on the family, was reported to be a large source of the stress found in US families needing professional support (Gelo and O’Malley, 2003).

Some researchers have used the Parenting Stress Index (PSI) alongside other measures to measure the impact of children with FASD, aged six to 12, on family stress dynamics in adoptive parents. Problems with executive function (the umbrella term for cognitive processes such as planning, working memory, attention, problem-solving and other such processes) in the children, and poorer parental resources, such as being a biological parent or a single parent, were associated with increased stress (Paley et al, 2006).

However, as is often the case with risk factors, these findings do not appear to be unique to FASD. Other neurodevelopmental disorders have raised similar issues and may also offer insights to help improve the situation. For instance, a study of 43 families of children with Joubert syndrome (a genetic disorder associated with neurodevelopmental problems) showed that the increased difficulties with behaviour and its management led to heightened stress in families (Farmer et al, 2006). Similar findings emerged from a longitudinal survey of four carer groups of children with broader neurodevelopmental disorders. The caregiver groups were more likely to have poor health and be depressed, and displayed greater problems with family functioning (Lach et al, 2009).

Despite these negative implications, other studies have shown possible avenues for improvement. A ten-year follow-up of both knowledge and stigma related to children with autism noted a shift in attitudes and decreased stress felt by families, a trend that the authors suggested could be related to changes in societal attitudes (Gray, 2002). When families retained balance in their lives and received social support to help them access adequate services for their children, outcomes improved (Trepagnier, 1999).
A scrutiny of the literature reveals that the impact of children with FASD upon carers in the UK has been little investigated. Research in other countries suggests that such children can create unique stressors for carers and provides some insights into factors that influence these. However, it is not possible to directly transfer evidence from one country to understand needs in another. Differences in social and health care systems mean it is important to investigate the situation in each country. Therefore, based on direct observations from clinical samples and suggestions in publications from other countries, we decided to test our hypothesis: namely, that despite universal access to health and social care services, carers within the UK struggle to bring up children with FASD. We also hoped to identify and explore factors that lessen or enhance the stress experienced by the carers of such children.

Methods

The research presented here was part of a wider study designed to ascertain what is known in the UK about FASD and its impact on individuals. The study was undertaken in a series of focus groups in different populations, supplemented by questionnaires developed and approved by research ethics to allow broad participation. The mixed-methods approach was used throughout in the hope that they would ascertain what people thought and inform a deeper understanding of associated issues. This article presents the findings of the carers’ evaluation.

Ethical considerations

In view of the sensitivity of the questions being asked about FASD, permission for the use of unsolicited questionnaires or direct mail contact was not granted by the research ethics committee. Specifically, direct contact with teenagers and pregnant women was also rejected, unless people from these groups themselves responded to advertisements. This introduced limitations to possible analysis, but as this population had not been studied in the UK, it was considered necessary to investigate the knowledge of a wider group before focusing on those associated with greater risk. Ethical protocol also required information to remain anonymous, keeping only broader, non-identifiable data. This requirement limited comparison and correlation of some parts of the data.

Sample

Advertisements for the project were sent to carers via three main FASD support charities in the UK. These are based in the north-west of England, London and Oxfordshire. Those carers wishing to participate were asked to contact the research team and were then invited to attend an educational question-and-answer (Q&A) session conducted by the lead researcher. In keeping with ethical committee requirements, it was made explicit that people could register to attend the Q&A session without having to participate in the overall study. Three focus group sessions for carers took place immediately before the education session and these provided the qualitative data. A maximum of 10 people per group was implemented so that if more wanted to participate, 10 were randomly selected and invited to a focus group. All interested parties were welcome to attend the education session from which quantitative data were collected.
Measures

The Parenting Stress Index questionnaire (PSI), a validated tool to look at the source of stress in a parent–child relationship (Abidin, 1995), was completed by all participants. As the PSI has been validated up to and including age 12, those parents whose children were older than this were asked to report the situation as they remembered it when their child was that age. While this was known at the outset to potentially introduce recall bias, we believe the value of this response outweighed the risks the method used.

Statistical analysis

PSI data were analysed using SPSS Version 18. The distribution of the data was confirmed using skewness and kurtosis as well as the Kolmogorov-Smirnov test (Field, 2009). Frequency data for multiple categories were assessed and comparisons between two continuous variables measured using t-tests. Suitability criteria for multivariate analysis were confirmed: namely, that the predictor variables were quantitative and outcome variables were continuous, and that multicollinearity had been avoided. ANOVA was used to address the variations seen by subtests. Post hoc Bonferroni corrections were made (Field, 2009).

Focus group process

Focus groups were conducted on all occasions by the lead researcher using a semi-structured interview with support from the project research assistant. The questions used had been previously approved by the research ethics committee (see Appendix 1). Each interview was allowed to flow freely based on participants’ responses to initial questions.

The project research assistant took field notes and each session was recorded on a digital tape recorder following written permission from the participants. The transcription was compared to written notes to allow for later data completion and verification. This was to ensure accuracy of the transcription as well as allow for annotation with supplementary information collected in the handwritten notes. There was no time limit on the sessions and participants were allowed to express their views openly.

Analysis of qualitative data

A thematic approach as described in Bazeley (2009) was used to analyse the qualitative data. Transcribed and verified data were entered into NVIVO Version 8. Initial coding was made separately by the two lead authors before coming together to complete more selective coding of the data and extract themes. The two researchers independently undertook the coding to establish its validity.

Results

Sixty-six people aged between 30 and 62 attended the three education sessions from which 10 people were selected randomly for each focus group. No one who was invited to attend a focus group refused. While the majority were adoptive or foster parents, two were biological parents. It is unclear why more biological parents did not attend. Of the 66 people who completed the PSI, only 54 completed it sufficiently to allow for analysis. The numbers
participating overall were considered low. As such, only the parental domain met the basic assumptions required for multivariate analysis and analysis of variance.

Table 1 summarises the ranked variance that accounted for parental stress for all cases in which the PSI was completed. As this was an exploratory study and the numbers participating affect the ability to draw conclusions from the data, it suggests possible trends that may be influencing the overall stress. The variance highlights the relative importance of various types of stress found in parents of children with FASD and offers suggestions into the areas that increase this stress by their rank. The higher the figure the more influence that factor has on the overall stress in the parent–child dynamic.

Table 2, supported by Graph 1, provides further analysis of the data considering the variance that is seen by age of the parents and the stress scores seen on the PSI. While there is overlap between the confidence intervals, these scores suggest a positive trend between overall stress and parental age. This is discussed further below, alongside the eight broad themes that were identified from the analysis of focus group data.

**Themes identified**

**Different from other children.** Families held the strong view that parenting a child with FASD was a very unique experience. In some families, other children with special needs and

<table>
<thead>
<tr>
<th>Subscale</th>
<th>Proportion of overall variance accounted for by each subscale (ranked)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Competence</td>
<td>.23</td>
</tr>
<tr>
<td>Spouse</td>
<td>.21</td>
</tr>
<tr>
<td>Role restriction</td>
<td>.21</td>
</tr>
<tr>
<td>Attachment</td>
<td>.18</td>
</tr>
<tr>
<td>Isolation</td>
<td>.17</td>
</tr>
<tr>
<td>Depression</td>
<td>.18</td>
</tr>
<tr>
<td>Health</td>
<td>.14</td>
</tr>
</tbody>
</table>

Note: Subscales are defined in PSI manual. In summary Competence: relates to the perceived ability to know how to deliver care; Spouse: relates to difficulties with a partner or spouse; Role restriction: relates to a perception that the role undertaken is isolated to only areas of care giving; Attachment: relates to the relationship between parent and child to bond appropriately; Isolation: relates to the perception the carers are restricted and away from wider society; Depression and Health: related to these factors in a carer’s life influencing the outcome.
emotional difficulties had previously been fostered, yet the children with FASD were still considered to be exceptional:

It wasn’t until I had my other two children and noticed that R’s behaviour was substantially different to the other two.

A competence deficit relating to the parenting of children with FASD was revealed in the PSI scores (Table 1). This suggests that the unique needs of children with FASD reported by carers have an effect on parents’ perceived ability to adequately rear the child. Even among experienced parents who reported having successfully parented other children, examples of unique difficulties were forthcoming:

Just a different behaviour totally, isn’t it? We had two boys of our own so we knew how to bring children up but with C she was just hot and cold all the time, wasn’t it?

Table 2. Parenting Stress Index scores by age: comparisons by individual subgroup ANOVA with post hoc Bonferroni correction.

<table>
<thead>
<tr>
<th>Scale</th>
<th>Age range studied (n)</th>
<th>Mean PSI score</th>
<th>SD</th>
<th>PSI percentile</th>
<th>95% CI</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total stress by parent age</td>
<td>30–39(10)</td>
<td>289.2</td>
<td>33.65</td>
<td>Approx 94</td>
<td>265.13 – 313.27</td>
<td>244–335</td>
</tr>
<tr>
<td></td>
<td>40–49(22)</td>
<td>305.18</td>
<td>39.19</td>
<td>Approx 97</td>
<td>287.8 – 322.56</td>
<td>212–370</td>
</tr>
<tr>
<td>Max score 373</td>
<td>50–59(11)</td>
<td>338.18</td>
<td>55.18</td>
<td>Max scale</td>
<td>301.11 – 375.25</td>
<td>304–373</td>
</tr>
</tbody>
</table>

F(3,46) = 3.28
p = 0.03
Significant Bonferroni corrected result between 30–39 and 50–59 showing increased stress by age

Graph 1. Mean and range of Parental Stress Index total score (max 373) by age group.
Parental stress was less influenced by the attachment between child and parent as seen in Table 1. This suggests that their own internal sense of competence and inability to rear their children were the main causes of stress. Table 2 highlights that this is exacerbated by age.

**Lack of information.** Adoptive parents felt they had not been given enough information by social workers to make informed decisions about the children:

- We were absolutely told no information at all even though we requested it many times.
- You should have enough information to be able to make an informed decision.

There was a sense that families’ difficulties with children with FASD were belittled or distorted. Parents reported (based on what they knew post adoption), that at the time of the pre-adoption interview, they had not been given enough information to allow for a truly informed decision. Whether this was related to the unavailability of information or information being withheld was not possible to establish.

**Lack of knowledge among professionals.** Many of the families reported difficulties in getting appropriate and sufficient support from professionals they met. This included doctors, social workers and educators. This led to greater perceived difficulties and the impression that they had nowhere to turn to obtain answers about how to best support their children:

- He was diagnosed with Fetal Alcohol Syndrome before he came to me and then I got the letter back saying he can’t have FAS, he’s too young to have it [group laugh], and that was from a proper consultant.
- You yourself have to educate the people that you are asking to help you about what is wrong with your daughter [laugh], so you end up feeling like you’re the professional!

These and similar experiences were reported to have increased rather than reduced the stress and difficulties for families caring for children with FASD.

**Having to ‘fight’ for things.** The lack of perceived support led some respondents to assert that they had to struggle to obtain the support to which they felt they were entitled. They felt that too much of their time was spent trying to obtain the help needed for their families, often with little benefit. These struggles were often experienced with multi-professional groups. The unrecognised uniqueness of the needs of children with FASD – and the different presentations of symptoms in that group when compared to other disabled groups – were seen as compounding these problems:

- I would want someone to say you’re not going to have to fight social services, you’re not going to have to fight education to get a placement, you’re not going to have to argue with health over resources or whatever, and then I would have felt more confident about carrying on.
- It’s this brick wall I keep hitting, what is FAS, that’s the brick wall I keep hitting all the time.

**Feeling misunderstood and blamed.** Often families reported that as a result of this lack of knowledge about FASD, they were blamed for poor parenting rather than supported by professionals. When comparing total scores for the child and parental scales, the PSI suggested that the child domain, which reflect the child’s influence on overall stress score, had a significantly greater effect than parental domains \( r(53) = 7.09, p < 0.0001 \). Those parents whose
children were aged 12 years or under showed a similar result \([t(44) = 6.28, p<0.0001]\). The lack of understanding regarding these child factors in the behavioural presentation by some professionals and the subsequent blame attributed to the parent compounded the effects on the stress experienced by families:

I wonder how many of us round here have been called dysfunctional parents? [Group put up hands] All of us.

Teachers would come out and blame us for bad parenting.

**Family stress and benefits of one to one.** Families reported that the impact of feeling blamed and lack of support increased the pressure they felt. In at least one case, this led to marital breakdown:

We divorced five years ago. It was just... the family just exploded, it was just, it broke down.

Totally broke down.

These are also seen in the PSI data from Table 1. The levels of difficulties with a spouse were reported to have an influence on the stress.

This sense of familial discord contrasts with the harmony reported when carers were on their own on a one-to-one basis with the children. Here the children were described as wonderful and great company:

On a one-to-one he’s fantastic. In a family unit it’s a nightmare when we’re all together [group agree] – an absolute nightmare.

I love being with him on my own you know, we go out places or at home we do cooking, we do lots of stuff. He’s great.

**Isolation.** The families said that the difficulties they experienced with the children led them to become isolated. They were not able to go out socially, and this led to a restricted role that differed from their previous situation (e.g. prior to adoption) also seen in the PSI data (Table 1):

We’re not doing any of the things that we thought we would be doing now when we adopted S. She doesn’t get invited anywhere herself and we don’t go with her anywhere, do we?

This is made worse when the themes overlap; for example, where there is marital breakdown, further increasing the sense of isolation. This in turn was reported as possibly enhancing the risk of depression in carers, which was shown to have an impact from the PSI data.

The other two children can go [to my ex-husband’s home] but you know, his partner doesn’t want R there because of something he may have done, or because of his interaction with their son, so I very rarely [go out], we don’t much get invited to friends’ houses when R is around, very rarely.

**Concerns about the future.** The final theme identified related to concerns about what would happen to the children in later stages of their development. Depending on the age of the child, this varied between anticipated difficulties faced in primary schools to how the child would manage in secondary education. Further longer-term implications as an adult, for example, if the individual would be able to live independently, also caused worry:
The professional said that we had no dreams or hopes for this child and I was putting him down and I said: ‘Yeah, he’ll be really good at maths and when he’s living out on the streets rough, he’ll be able to count how many cardboard boxes he’s got to live in that night.’

The long-term implications, that’s what everybody wants to know, you know what kind of life is the child going to have, what kind of difficulties that YOU are going to face.

Our big issue again is now going to be secondary school. That’s when we’re really, really worried.

**Discussion**

Consistent with our initial hypothesis, these results are similar to findings from other countries. People in the UK do struggle to obtain help for their children (Brown and Bednar, 2003; Brown, Sigvaldason and Bednar, 2005; Brown, Bednar and Sigvaldason, 2007; Brown, Sigvaldason and Bednar, 2007; Caley, Winkelman and Mariano, 2009; Gardner, 2000; Gelo and O’Malley, 2003; Granitas, 2004). They report battles, pressures and concerns, such as the need for respite, which do not seem to be sufficiently addressed even by the universal access to the comprehensive health and social care system that exists in the UK.

**Support and isolation**

Carers reported finding it difficult to care for a child with FASD in many different ways, but these were universally exacerbated by the perceived lack of support and isolation. The initial lack of information about potential difficulties in bringing up the children, and concern for their future, combined to prevent potential parents making informed decisions about adoption. This was associated with the later realisation that their decision increased the stress they experienced. This may well have contributed to the isolation they felt in contrast to the idea of what their lives may have otherwise been.

One important difficulty relates to the amount of accurate information that can be predicted early in the child’s life. Information about a child’s longer-term potential may well be limited in the early years. For example, two children may well develop in different ways even though they were exposed to similar risks. This lack of clarity leaves many carers with a sense that professionals simply cannot advise them adequately. Considering that a recent study showed that up to 30% of a subgroup of children in the foster care system may well have prenatal drug and alcohol exposure (Selwyn and Wijedesa, 2011), this is a potentially serious problem.

Since 2002, the Adoption and Children Act highlights the importance of post-adoption support from social services and health (Department for Children, Schools and Families, 2010). Studies suggest parents of children with FASD still feel unsupported and unskilled (Neil, Cossaar and Young, 2010; Sellick et al, 2007). Helping these parents adapt their child-rearing skills to the needs of children with FASD, and encouraging parents not to blame themselves, may be key to longer-term reduction of stress in the family. It would be hoped that by doing this, the difficulties revealed by the PSI results in this study may be minimised.
Adoption policy

The results further raise questions about the strategies currently used by adoption and foster care services. The benefits of kinship adoption and staying with the family have been documented by several authors (Aldgate and McIntosh, 2006; Children 1st, 2011; Department for Education, 2010; Department for Children, Schools and Families, 2010; Neil, Cossar and Young, 2010; Rushton, 2003; Sellick, 2007; Sellick et al, 2007). The Department for Education (2010) report underscores the complexity of how and why people are adopted and points out the need for continual support.

The benefits of kinship care and staying together as a family (such as feelings of being loved, maintaining relationships with friends and family, and more stable, longer lasting placements) were affirmed by this research, even though there are often difficulties such as limited freedom, financial problems and overcrowding (Department for Education, 2010). A report on fostered children in Scotland, for instance, estimated that 1400 children were in such placements, often with older carers, in particular grandparents (Aldgate and McIntosh, 2006).

Our findings suggest the need for more detailed research and for great care in policy implementation within the specific population of carers for children with FASD. Older carers found the children’s behaviours more difficult to manage and that children were easier to handle in a one-to-one situation, questioning some of the assumptions made about kinship care discussed above. Current policy may well be placing an unintentional burden on the carer. These results are not conclusive but do warrant further exploration in this population.

Secondary disabilities

Longer-term follow-up studies of people with FASD have highlighted difficulties with secondary disabilities and mental health vulnerabilities (Famy, Streissguth and Unis, 1998; Gray and Mukherjee, 2007; Mukherjee, Turk and Hollins, 2006; Streissguth and O’Malley, 2000). However, these can be minimised by good parenting that specifically meets the needs of this groups (O’Connor, Kogan and Findley, 2002). Unfortunately our findings suggest that problems faced by parents as a result of not receiving support to modify parenting techniques combine with a perceived lack of recognition of difficulties by professionals to produce an inauspicious situation. There are opportunities to make a difference, but that requires that these difficulties be addressed.

Limitations

These results cannot be seen as conclusive as this was primarily an exploratory study to identify areas of deficit and guide future more focused research. As the number of participants was small, the power of the quantitative analysis was limited and should therefore be considered as an indicator of trends. Further, the ability to follow up themes identified was not always available as part of this project. In addition, while the original sampling frame was self-selected, randomisation was used for the focus groups. Thus, while the views presented clearly reflect the subgroup who took part, it may not represent the wider population.
Conclusions and recommendations

The results suggest that in the UK, carers of children with FASD feel unsupported and blamed. The difficulties they face are unexpected and could be reduced by more openness and discussion about possible longer-term outcomes. Ongoing post-adoptive support (both emotionally and financially, in keeping with other research) is likely to help. This needs to be balanced with closer investigation into which groups are best placed to bring up these children. The practice of placement with older foster carers and adopters may well need further investigation.

While this is a small-scale study, some recommendations can be suggested from the findings. First, better training for professionals involved with these children. This should be correlated with better information for those looking to adopt, for example at the matching stage of the process. Second, specific access to training for parents around the needs of individuals with FASD, alongside ongoing support from both statutory and charitable organisations would be helpful for families. The outcomes of these would require evaluation to confirm their longer-term benefits. However, the findings here would suggest this may be a way to improve the outcomes faced by these children and their families.

References


Appendix 1. Semi-structured questions used in focus groups

(1) What did you know about FAS before you had a child with the condition?
(2) What has been difficult?
(3) What help have you received?
(4) How easy has it been to get help?
(5) What have you had to do to get this help?
(6) Was it useful?
(7) What support is there for this disorder?
(8) How much do professionals appear to know about this condition?
(9) How does this affect your life?
(10) What positives do you see?
(11) What advice would you give to others?

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Estimation of alcohol content of wine, beer and spirits to evaluate exposure risk in pregnancy: Pilot study using a questionnaire and pouring task in England

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Abstract

Aims: Research has shown varying results regarding safe consumption levels of alcohol during pregnancy. We argued in 2005 that an individual’s inability to accurately predict her alcohol consumption may be one factor influencing risk. In order to re-evaluate within the England, this study sought to assess the current knowledge of the public and of healthcare practitioners.

Design: Both alcohol-knowledge questionnaires and pouring tasks were conducted using standardised ethical-committee-approved methods.

Settings: Different sites across England, including Surrey, London, Oxford and Wigan, where FASD support groups are based.

Participants: Health professionals and the general public, self-selecting in response to advertisement.

Measurements: Frequency data and categorical data was collected and analysed using SPSS version 18.

Findings: In total, 1,265 questionnaires were completed (688 public and 577 professionals). One hundred-forty people completed the pouring task. People’s ability to calculate accurately from strength and volume was within 20% of the accurate figure for units, although with a wide range.

Conclusions: These findings support the hypothesis that when pouring their own drinks, individuals are poor at estimating each drink’s alcohol content. This has implications for public health strategies. Glass size and the level of alcohol concentration have different implications in different countries. For those drinking during pregnancy, however, the message that “no exposure is no risk” remains true.

The debate surrounding a safe level of alcohol consumption in pregnancy continues to draw attention. Since our 2005 U.K. study regarding safe drinking limits in pregnancy (Mukherjee, Hollins, Abou-Salih, & Turk, 2005), other researchers internationally have addressed the issue, but have yet to clarify individual risk. Some studies have suggested that even low levels of exposure carry risks of, for example, mental health problems (Alati et al., 2008; Disney, Iacono, McGue, Tully, & Legrand, 2008; Henderson, Gray, & Brocklehurst, 2007; Patra et al., 2011; Sayal, 2009; Sayal, Heron, Golding, Altai et al., 2007; Sayal, Heron, Golding, & Emond, 2007), while others have shown no evidence of developmental problems in comparison to non-exposed groups (Gray, Mukherjee, & Rutter, 2007; Kelly et al., 2007; Kelly et al., 2010; O’Leary et al., 2010; Onofrio et al., 2007). We argued that in the U.K., individual women’s knowledge of alcohol units, as demonstrated through their consumption levels and higher exposure risk to alcohol, was poor. Further, we argued that if they were pregnant, this increased their risk of having a child affected by prenatal alcohol exposure (Mukherjee et al., 2005).

As part of a wider study of professional and public knowledge about drinking in pregnancy and, more broadly, about Fetal Alcohol Spectrum Disorders (FASD), we...
decided to test our assumption that people were poor at accurately predicting units and poor at estimating the volume they had consumed.

The UK is made up of four nations, each responsible for its own health policy. This research was mainly conducted in England. As a wide range of beverages containing alcohol is available in the UK, alcohol is understood in terms of units. A unit of alcohol is defined as containing 8g (10mls) of absolute alcohol (The Cabinet Office, 2004). The Cabinet Office recommends that for health reasons, people should not exceed a daily maximum intake of 3-4 units of alcohol for men, and 2-3 for women. Binge drinking is defined as consumption of double the daily maximum units: six units for a woman and eight for a man (Department of Health, 1995).

In 2008 an Office of National Statistics (ONS) survey suggested 83% of the population had some awareness of units. What was unclear was how accurately they were able to predict what quantity of a given beverage a unit actually contained (Lader & Goddard, 2006). Two reports highlight changing U.K. alcohol consumption trends. The first showed 21% of men and 14% of women were drinking more than double their recommended daily levels of alcohol per week (Office of National Statistics, 2008), and that people in higher socio-economic groups drank more (Office of National Statistics, 2008). A subsequent survey reported that people were now drinking more at home (Office of National Statistics, 2009). For women this figure was 57%, with 54% claiming to drink at least weekly (Office of National Statistics, 2009). While most women in the study (n = 1,153) had heard about units, it was unclear how this knowledge affected their behavior (Office of National Statistics, 2009).

A series of studies published in Scotland from 2004–2007 attempted to look at this. The first asked each participant in the study to pour a unit of wine, then spirit. The amount poured was more than double the correct volume in 43% of cases for wine, and 55% of cases for spirit (Gill & Donaghy, 2004). Female undergraduate students asked to complete a similar task were found to pour double the original self-reported estimates (Gill, Donaghy, Guise, & Warner, 2007). Despite the addition of a teaching intervention to the pouring task, a group of 297 mixed participants continued to pour double the accurate level, at 2.05 units (Gill & O'May, 2007).

Outside the U.K., similar patterns have been reported. A Dutch study comparing individuals’ ability to estimate standard drink measures (1.5 UK units) for wine and spirits found the group overestimated by 14% and 26% respectively. Women were 7.5% more likely to be inaccurate than men (Lemmens, 2006).

At least two reports have shown that glass size can have an effect on pouring. The first assessed 80 drinks poured in bars, showing that the biggest impact on inaccuracy was the size of the glass rather than its shape (Kerr, Patterson, Koenen, & Greenfield, 2009). A second study, a comparison between shots poured into a small tumbler versus a long slender glass found the shape also had an effect, with 20% more poured into the tumbler (Wasinski & Illersum, 2005). As all these factors were considered to be an influence on exposure risk, we decided to test our hypothesis that people in England were poor at estimating units when given basic information found on many bottles sold in England, and when pouring different drinks, and that this varied by glass size and shape.

METHODS

This study was part of a wider project assessing knowledge about FASD among professionals and the general public in England using questionnaires, focus groups and a series of pouring tasks. Only aspects relating to knowledge of units and the pouring tasks are presented here.

Ethical Considerations

Permission to use unsolicited questionnaires or direct mailing was not granted by the research ethics committee, due to the sensitivity of the questions being asked about FASD. Contact with teenagers and pregnant women was also not allowed, other than in response to indirect advertising. Questionnaires had to be anonymous, preventing some direct comparison between data collected on the questionnaire and the pouring task. Separate public and professional questionnaires were mandated, due to some questions being considered too medical for the general population. Further to address consent issues, an explanation on the title page of the questionnaire, stipulating that people did not have to complete it if they chose not to, was stipulated. Finally, it was insisted that all people who were interested should be allowed to attend education sessions without being required to participate in the actual research. This accounted for some of the discrepancies in questions completed.

Sample

The project was advertised by three national FASD support groups; through direct mail to health departments; through professional networks; and though poster placement in local community centres, churches and private companies with links to the researcher’s organisation. The ethical-committee-approved advertisement highlighted the research project, but also emphasized that the educational question-and-answer session regarding FASD was open to all. Individuals who self-selected to participate were directed via the advert to contact the research team. People working in private or public health services and in direct contact with patients were considered health professionals. All others were allocated to the general public group.

Questionnaire Design

Based upon questions raised in previous research and a smaller pilot conducted by our group (Mukherjee, Hollins, & Turk, 2006), a series of seven questions about alcohol strength and volume were presented (Table 1). Basic information found on many current U.K. bottles containing
alcohol—namely, percentage of alcohol and volume—was provided to assess individuals’ accuracy in calculating the number of units found in each drink. These questions were not exhaustive. We began with a set of 17 questions; balancing the demands of keeping the questionnaire to an appropriate length, accommodating the suggestions of the ethical committee, and answering our specific research question, we arrived at the questions that were eventually used.

**Process**

Separate sessions were conducted for professional and public groups. All participants were invited to attend the research and education session regarding alcohol use in pregnancy, which was presented by one of the authors (RM). The pouring task was completed after a focus group meeting (part of the wider study and not reported here) and before the education session. Questionnaires were completed by all attending the education session, prior to its commencement.

<table>
<thead>
<tr>
<th>Drink volume and type (% alcohol) (N = respondents)</th>
<th>125ml white wine (9%) (N = 1024)</th>
<th>250ml red wine (13%) (N = 1018)</th>
<th>125ml white wine (13%) (N = 1000)</th>
<th>Half pint beer (3.5%) (N = 1012)</th>
<th>Pint strong lager (5%) (N = 1011)</th>
<th>Bottle of alcopop (5%) (N = 982)</th>
<th>50ml vodka (37.5%) (N = 989)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Correct value (units)</td>
<td>1.13</td>
<td>3.25</td>
<td>1.63</td>
<td>0.99</td>
<td>2.84</td>
<td>1.65</td>
<td>1.88</td>
</tr>
<tr>
<td>Mean estimate of units [percentage of actual result]</td>
<td>[106.56]</td>
<td>[82.09]</td>
<td>[114.34]</td>
<td>[80.75]</td>
<td>[112.57]</td>
<td>[153.42]</td>
<td></td>
</tr>
<tr>
<td>95% CI [percentage of actual result]</td>
<td>[1.17 – 1.23]</td>
<td>[2.62 – 2.72]</td>
<td>[1.65 – 1.72]</td>
<td>[1.1 – 1.16]</td>
<td>[2.25 – 2.34]</td>
<td>[1.81 – 1.91]</td>
<td>[2.81 – 2.96]</td>
</tr>
<tr>
<td>Range [percentage of actual result]</td>
<td>[103.94 – 109.18]</td>
<td>[80.54 – 83.65]</td>
<td>[101.5 – 104.5]</td>
<td>[111.14 – 117.53]</td>
<td>[79.08 – 82.43]</td>
<td>[109.40 – 115.65]</td>
<td>[149.40 – 157.44]</td>
</tr>
<tr>
<td>Bottle of alcopop (5%) (N = 982)</td>
<td>[530.97 – 531.91]</td>
<td>[184.62 – 184.72]</td>
<td>[214.72 – 214.82]</td>
<td>[606.06 – 606.12]</td>
<td>[211.27 – 211.32]</td>
<td>[303.03 – 303.05]</td>
<td>[531.91 – 532.93]</td>
</tr>
</tbody>
</table>

**Table 1**

Estimate of the number of units of alcohol contained in various serving sizes and types of alcohol

**Note.** The top line of the table represents the extent of information given to individuals in the questionnaire (CI = Confidence interval)

Individuals were asked to fill in each question to the best of their ability. Pouring tasks were conducted using a standard protocol, described in Box 1. All glasses were visible on a table, and each participant followed the same process of pouring a beverage into the small glass, then the large glass, and finally the tumbler. Each glass was emptied prior to the next being filled.

**Box 1**

Questions asked during pouring tasks set and glasses used, with the type of content to be poured into each glass

- Participants were asked the two following broad questions. The first question was posed and all three glasses poured before asking the second question. Each glass was emptied before the next pour was attempted.
  - You return at the end of a long day at work and pour out a drink, please pour what you would normally pour into each glass.
  - Now please try and pour as accurately as possible one unit of each.
- Three different glasses and two types of alcohol (All purchased from High Street supermarket and coloured water used instead of wine/spirit)
  - 150ml glass 13% wine
  - 350ml glass 13% wine
  - 200ml whisky tumbler 40% malt whisky

Questionnaires were also collected at professional and public conferences across the U.K, where RM was an invited speaker. Finally, for those responding to advertisements but unable to attend sessions, an online questionnaire was available.
**Statistical analysis**

For each value on the knowledge questionnaire, frequency data including means, median, standard deviations, 95% confidence intervals, range for unit estimations and percentage of a unit were calculated. Extreme outliers were excluded, and values of skewness and kurtosis were used to analyse normal distribution of frequency data. Where distributions were not normal, comparisons between the professional and the general public, as well as between genders, were conducted using non-parametric tests. These included the Mann-Whitney U test, as recommended by Field (2009). For the pouring task, frequency data were collected and the mean percentage deviation of accuracy was calculated. Normality was tested using the Kolmogorov-Smirnov test. The Mann-Whitney U test was used to test for significance. The values were compared by gender, age and profession (Field, 2009). Only two groups were compared at any one time; therefore, post hoc corrections were not made. All data were entered and analysed using SPSS version 18.

**RESULTS**

**Sample**

One hundred forty people completed the pouring task and 1,265 questionnaires were completed. Table 2 presents the demographic breakdown of the study. Fifty-seven percent (n = 688) were non-professionals and 83% (n = 897) were female. All adult age groups were represented in the sample, with a mean age of 43 (range 18-75) for the questionnaire and 44 for the pouring task. Table 1 presents the number of participants that completed each question, as well as the findings. Many participants left the alcohol calculation section blank. Despite extreme outliers being excluded (four in total), the data remained non-normally distributed. Table 3 presents the findings from this task. Six people, for personal reasons, declined to pour a glass of whisky. The results from the wider study on knowledge of FASD have been submitted for publication elsewhere; an overall finding was that 86.7% of the general public and 93.8% of professionals had heard about FASD, but only 26.9% and 33.5% of those groups, respectively, could confidently predict what level of alcohol exposure in pregnancy would be considered a clear risk.

**Table 2**

<table>
<thead>
<tr>
<th>Demographic data from questionnaires and pouring task</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td><strong>Questionnaire</strong></td>
</tr>
<tr>
<td>Number</td>
</tr>
<tr>
<td>Public</td>
</tr>
<tr>
<td>Professional</td>
</tr>
<tr>
<td>Total</td>
</tr>
<tr>
<td>Male</td>
</tr>
<tr>
<td>Female</td>
</tr>
<tr>
<td>Mean age</td>
</tr>
<tr>
<td>Age Range</td>
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<td></td>
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<td></td>
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<td></td>
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</table>

<table>
<thead>
<tr>
<th><strong>Pouring Data</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td><strong>Questionnaire findings</strong></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>When informed of both the volume of liquid and its alcohol content, the majority of participants made estimations of the unit content within 20% of the actual figure. This did not hold true for whisky, where the estimates were, on average, 50% above the expected figure. Further, the range of values observed was wide for all glass sizes and percentage volumes of alcohol. The upper end of this range varied, with overestimates from 184% to 606% of actual figures. There did not appear to be any clear pattern evident from the data. When comparing groups, professionals were more accurate in calculating the actual units of vodka (Mann Whitney U = 104415.5, p = &lt;0.001) and strong lager (Mann Whitney U = 114752.5, p = 0.02). Men were more accurate than women at calculating the number of units in 125 mls of wine at 13% alcohol concentration (Mann Whitney U = 52415, p = 0.05) and in half a pint of beer at 3.5% alcohol concentration (Mann Whitney U = 52415, p = 0.05). There were no other statistically significant differences seen when comparing the different levels of alcohol knowledge by profession, age or gender.</td>
</tr>
</tbody>
</table>

| **Pouring task**                                    |
|                                                      |
| The pouring task (Table 3 and Figure 1) showed a range of findings; the amounts poured varied greatly. Glass size had a significant effect on the outcome of individual “normal” pouring behavior (Wilcoxon Matched pair test z = -9.21, p = < 0.001). The size of the larger glass made some people more cautious when trying to pour a unit, but had the |
Table 3

<table>
<thead>
<tr>
<th>Task requested</th>
<th>N</th>
<th>Median</th>
<th>Range</th>
<th>Comparison with Wilcoxon matched pair analysis Z</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pour a normal drink wine 13%: Small glass [percentage compared to a unit]</td>
<td>140</td>
<td>110 [144.26]</td>
<td>30 – 150 [39.34 – 196.72] IQR = 39.34</td>
<td>-9.21, p &lt; 0.001</td>
<td></td>
</tr>
<tr>
<td>Pour a normal drink wine 13%: Large glass [percentage compared to a unit]</td>
<td>140</td>
<td>90 [118.03]</td>
<td>50 – 355 [65.57 – 465.57] IQR = 44.26</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pour one unit: Small glass wine 13%: [percentage compared to a unit]</td>
<td>140</td>
<td>155 [203.28]</td>
<td>30 – 150 [39.34 – 196.72] IQR = 98.36</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pour one unit: Large glass wine 13%: [percentage compared to a unit]</td>
<td>140</td>
<td>92.5 [121.31]</td>
<td>25 – 190 [32.79 – 249.18] IQR = 52.46</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pour a normal measure of Whisky [percentage compared to a unit]</td>
<td>134</td>
<td>50 [200]</td>
<td>10 – 90 [40 – 360] IQR = 60</td>
<td>-2.56, p = 0.01</td>
<td></td>
</tr>
<tr>
<td>Pour one unit of Whisky [percentage compared to a unit]</td>
<td>140</td>
<td>37.5 [150]</td>
<td>10 – 60 [40 – 240] IQR = 100</td>
<td>-6.94, p &lt; 0.001</td>
<td></td>
</tr>
</tbody>
</table>

Note. Actual expected value for one unit: Wine 13% = 76 ml; Whisky 40% = 25 ml. As results non-parametric Median and range presented only. (Inter Quartile Range for percentage = IQR)

Table 4

<p>| Results showing the difference between gender and population of study regarding accuracy of pouring (combined percentage total difference for all types of drink) |
|------------------------------------------------------------------------------------------|----|--------|----------------|-----------------------------------------------|---------|</p>
<table>
<thead>
<tr>
<th>N</th>
<th>Median</th>
<th>Min</th>
<th>Max</th>
<th>Man Whitney U score</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>30</td>
<td>167.65</td>
<td>69.07</td>
<td>218.58</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>110</td>
<td>156.67</td>
<td>79.18</td>
<td>244.26</td>
<td>1480.50, 0.389</td>
</tr>
<tr>
<td>Public</td>
<td>112</td>
<td>157.24</td>
<td>69.07</td>
<td>244.26</td>
<td>1473.56, 0.623</td>
</tr>
<tr>
<td>Professional</td>
<td>28</td>
<td>159.89</td>
<td>79.18</td>
<td>211.26</td>
<td></td>
</tr>
</tbody>
</table>

Opposite effect on others. There were also significant differences between the amounts people poured when asked to demonstrate their routine pouring behavior and when asked to try and pour measures of alcohol accurate to a unit (Wilcoxon Matched pair tests z = -6.49, p ≤ 0.001). Some, when asked to pour what they would normally pour, poured up to 456% more than a single unit. The skewed nature of the data, with the wide range of volumes, suggested that a few people were particularly poor at estimation. Table 4 shows the comparison by gender and population studied for the combined percentage deviation. No significant findings were seen.

**DISCUSSION**

Consistent with our hypothesis, the results highlight the inability of both members of the general public and health professionals to estimate the alcohol concentration in their drinks and, in some cases, to pour accurately. Unlike other studies, ours found no real gender difference, possibly because of the numbers in each group. Although participants showed much cautiousness about the amount poured, there was a small group who were very bad at pouring accurately, especially when demonstrating their normal pouring behavior with spirits, where much wider ranges were seen with the larger glass. This skewed the findings. Further, this did not seem to be influenced by participants’ broader knowledge of alcohol units. Glass size was also found to have an effect on accuracy in wine pouring, implying that, for a small but significant group at least, smaller glass sizes would decrease health risks.

**Impact on public health**

The harm from alcohol was highlighted as a growing concern in a series of reports published by the World Health Organization (World Health Organization, 2004, 2010, 2011). The annual cost of this harm in the U.K. was estimated to be £2,704 million in a cabinet office report from 2008 (Hiat, 2008).
The increasing size of glasses and the high concentration of alcohol by volume suggest a potentially worrying trend, especially for those least able to accurately estimate consumption. Our findings raise the question of what undermines people’s ability to estimate quantities of alcohol: preconceived ideas, a true lack of understanding, or another reason as yet unidentified? The finding that glass size affected the amounts being poured and the ability of drinkers to accurately predict and pour quantities of alcohol has potential implications for the sizes of glass that are used and sold for different types of drink.

Whilst our results are far too provisional to be conclusive, they do suggest the need for further exploration of the impact of both the size of glasses and the increasing strength of alcohol, in order to guide future policy development in this area.

Impact of knowledge on drinking advice in pregnancy

Whilst these results may be considered provisional, they strengthen our argument from 2005 that due to inaccuracies in their estimates of the quantity of alcohol they consume, and due to uncertain additional environmental risks, women should be advised not to consume alcohol in pregnancy (Mukherjee et al., 2005). Different countries continue to offer different guidance for drinking in pregnancy; for example, the U.S. suggests that no alcohol is safe, whilst the U.K. guidance is that although it is better to avoid alcohol, drinking 1–2 units once or twice a week is unlikely to cause harm (International Centre for Alcohol Policies, 2009). Our results reveal the problem with telling women that they can drink 1–2 units: their knowledge of what a unit is and, for some, their accuracy in identifying one is poor. This advice may well put individuals in a high-risk category without their realising it.

A statement that a behavior is safe should only be made when there is certainty that it is safe for all, especially when risks may lead to a condition with lifelong consequences, such as FASD. Studies have produced mixed findings regarding effects of alcohol consumption on neonatal development, especially at low levels of exposure. Whilst some studies, including some large population-based epidemiological studies, have shown low-level consumption to have limited effect (Alati et al., 2008; Disney et al., 2008; Henderson, Gray et al., 2007; Henderson, Kesmodel, & Gray, 2007; Kelly et al., 2010; O'Leary et al., 2010), others—including similar population-based studies, but also animal research—have shown that there may be the possibility of harm. This is especially, but not exclusively, related to neurodevelopment and later mental health outcomes (Chaudhuri, 2004; Ieraci & Herrera, 2007; Jacobson & Jacobson, 1999; Sayal et al., 2007; Sayal et al., 2009). These same studies confirmed the dose-response relationship with alcohol. High levels of exposure led to the most harm and the greatest expression of the physical characteristics of the disorder.

Methodological criticisms of the studies on both sides of the argument have also been made. For example, it has been argued that the measures used in the epidemiological research were insensitive to change (Sayal, 2009). Others have argued about difficulties in extrapolating animal data to humans (Abel, 2009). This shows that the picture remains unclear.
Our original paper suggested that due to uncertainty about levels of risk, it would be better for pregnant women to avoid alcohol. Our findings from this study would suggest this remains true. Although individual risk cannot be measured, despite all the recent evidence, what is clear is that high exposure relates to high risk and low exposure to low risk. The only truly safe message regarding consumption is that no exposure to alcohol means no risk.

Limitations

This study had some limitations. A self-selected group may not reflect the true level of knowledge of the general public or of most health professionals, due to potential selection bias. There was a gender bias, in that far more women participated. The study does, however, suggest a trend that warrants further investigation: the size and shape of the glasses used may have influenced participants’ pouring behavior. The exact nature of this influence seemed to vary, with the larger glass inducing sometimes recklessness and sometimes caution, but as the same set of glasses was used in all cases, the data is consistent for everyone tested, allowing comparisons to be made. With regards to the questionnaire, it is not known why some people did not complete all the questions. It may be a reflection of a lack of knowledge of how to calculate units, or simply a wish not to participate.

Conclusions

Our results suggest that while providing consumers with information about alcohol units is important, it does not appear to prevent behavior that may lead people to consume more alcohol than they realize. Making smaller glasses available for those least able to pour accurately, and making it easier for people to judge and estimate alcohol content, may reduce this risk. In pregnancy, the importance of this is magnified by continuing uncertainty about the actual level of potential risk that alcohol consumption poses to the fetus. Given the increasing prevalence of FASD in many societies, and our predominantly female participants’ inaccurate assessments of the alcohol content of the drinks they poured, only complete abstinence from alcohol can guarantee that there is no risk to the fetus.

Acknowledgements

Many thanks to E. Riley for comments made on early drafts of the paper and P. Cook for comments regarding statistical presentation of the paper.

REFERENCE


About the Foundation for Alcohol Research and Education

The Foundation for Alcohol Research and Education (FARE) is an independent charitable organisation working to prevent the harmful use of alcohol in Australia. Our mission is to help Australia change the way it drinks by:

• helping communities to prevent and reduce alcohol-related harms
• building the case for alcohol policy reform and
• engaging Australians in conversations about our drinking culture.

Over the last ten years FARE has invested more than $115 million, helped 750 organisations and funded over 1,400 projects addressing the harms caused by alcohol misuse.

FARE is guided by the World Health Organization’s Global Strategy to Reduce the Harmful Use of Alcohol for addressing alcohol-related harms through population-based strategies, problem-directed policies, and direct interventions.
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Foreword

Fetal Alcohol Spectrum Disorders (FASD) is the leading preventable cause of non-genetic, developmental disability in Australia. However, up until recently FASD has been largely overlooked by government.

Australia has now reached a critical juncture, a tipping point if you like, and as is so often the case, the achievements, victories and successes are not the results of the efforts of thousands, but the direct result of the committed efforts of a dedicated few.

We didn’t reach this tipping point easily.

For twenty years, researchers and passionate individuals have worked tirelessly to fill the government policy void, raising awareness of FASD at the state and national level, working on the frontline with those living with FASD and those caring for them.

The success of these combined efforts have resulted in the current House of Representatives Inquiry into FASD which will shortly hand down its findings and recommendations to the Government.

The Foundation for Alcohol Research and Education (FARE) too has played a role. Since 2001, FARE has invested over $2 million into the prevention and treatment of FASD in Australia. Most recently FARE invested half a million dollars into seven projects to address FASD, including the establishment of the first ever diagnostic clinic in Australia. FARE’s efforts have culminated in the preparation of the National Fetal Alcohol Spectrum Disorder Action Plan.

FARE’s Australian Fetal Alcohol Spectrum Disorder Action Plan represents a roadmap for the journey ahead, a costed plan of action that addresses five priority areas: increasing awareness of FASD, increasing diagnostic capability, improved services and support for people with FASD, improved data collection and efforts to close the gap among Aboriginal and Torres Strait Islander peoples.

Throughout the development of the Plan, FARE has had the very real pleasure to work closely with an extremely accomplished group of researchers, doctors, carers, communities and families around Australia.

The Plan has been endorsed by the peak FASD consumer and carer organisation the National Organisation for Fetal Alcohol Syndrome and Related Disorders (NOFASARD) and the Australian FASD Collaboration led by Professor Elizabeth Elliot and Winthrop Research Professor Carol Bower. FARE also consulted widely and acknowledges the support of Australia’s leading FASD experts, whose contribution and cooperation has been critical in the production of this important policy document. These people include:

- Professor Steve Allsop, National Drug Research Institute, Curtin University
- Winthrop Research Professor Carol Bower, Telethon Institute for Child Health Research, Centre for Child Health Research, The University of Western Australia
- Dr Courtney Breen, National Drug and Alcohol Research Centre
- Dr Lucinda Burns, National Drug and Alcohol Research Centre
- Ms Maureen Carter, Nindilingarri Cultural Health Services and Chief Investigator of the Lililwan Project
- Ms Meredythe Crane, Alcohol and other Drugs Council of Australia
- Ms Heather D’Antoine, Menzies School of Health Research, Charles Darwin University
- Professor Heather Douglas, Law School, University of Queensland
- Ms Sharon Eadie, The George Institute for Global Health, University of Sydney Medical School and the Lililwan Project
- Professor Elizabeth Elliott, University of Sydney Medical School, The George Institute for Global Health and Chief Investigator of the Lililwan Project
- Dr James Fitzpatrick, University of Sydney Medical School, The George Institute for Global Health and Chief Investigator of the Lililwan Project
- Dr Kate Frances, National Drug Research Institute, Curtin University
- Ms Adele Gibson, Anyinginyi Health Aboriginal Corporation
We must not forget that the concerning levels of alcohol-related harms in Australia are being driven by the ever increasing availability and affordability of alcohol and the alcohol industry’s aggressive marketing, promotion and advertising efforts. Any significant effort to reduce alcohol-related harms in Australia and address this nation’s drinking culture must be prepared to address those fundamental issues as well.

The Plan acknowledges the current work being undertaken by governments throughout Australia, but also demonstrates the significant work that is still required to ensure that governments addresses the critical gaps that exist in the prevention and management of FASD.

The Hon Nicola Roxon and the Hon Jenny Macklin are to be congratulated for their role in the establishment of the current House of Representatives Inquiry into FASD. It is now up to the Commonwealth Government to seize the momentum, to build on the work of the Inquiry, to take heed of the Inquiry’s findings and recommendations and to listen to Australia’s FASD experts.

I urge the Commonwealth to adopt the Australian Fetal Alcohol Spectrum Disorder Action Plan and ensure this nation’s efforts to effectively address FASD in Australia do not falter.
Plan overview

Fetal Alcohol Spectrum Disorders (FASD) are the leading preventable cause of non-genetic, developmental disability in Australia. Like many other disabilities, people who are born with FASD have the condition for life.

FASD is a non-diagnostic term representing a range of conditions that result from prenatal alcohol exposure. These conditions include Fetal Alcohol Syndrome (FAS), partial FAS, Alcohol-Related Neurodevelopmental Disorder and Alcohol-Related Birth Defects. The primary disabilities associated with FASD are directly linked to the underlying brain damage caused by prenatal alcohol exposure. These can include poor memory, impaired language and communication, poor impulse control and mental, social and emotional delays. In addition to neurological damage the individual may also have physical impairments ranging from subtle facial abnormalities to organ damage.

People with FASD often experience difficulties in day-to-day living. Much of their outward behaviour may appear to others as delinquent or antisocial and this can result in judgments being made about the nature of the person, their behaviour and capability as well as criticism of their parents or carers.

Australia’s response to FASD is at a critical junction. For too long there has been a lack of coordinated action to prevent FASD and assist people affected. Over the last few decades researchers and passionate individuals have worked tirelessly to raise awareness of FASD at local and national levels. This work has often been ad hoc and inconsistently funded and implemented by Australian governments. A concise summary of the history of FASD related activities in Australia is provided in Appendix A.

Currently in Australia:

- One in five women continues to consume alcohol while pregnant after knowledge of pregnancy.
- Health professionals continue to be reluctant to ask women about their alcohol consumption during pregnancy, despite national alcohol guidelines which clearly state that it is best to avoid alcohol altogether during pregnancy.
- Few health professionals are familiar with the clinical features of FAS and there is no standardised Australian FASD diagnostic instrument or clinical guidelines for FASD diagnosis.
- Early intervention options for people with FASD are nonexistent, resulting in the greater likelihood of poorer life outcomes in education and employment.
- Despite the lifelong implications of FASD, getting support is extremely limited and difficult to access.

An Australian FASD Action Plan is now needed to begin to address the extensive gaps in the prevention, early intervention and management of FASD in Australia. The Australian FASD Action Plan 2013-2016 presents actions to be undertaken in three years to start to reduce the numbers of people born with FASD and to help support those currently affected.

The Australian FASD Action Plan includes priority areas that target FASD across the spectrum, from prevention of the condition to management across the lifespan. Each of these areas has clearly defined actions, outputs and targets. The Plan focuses on areas with clear actions and the greatest likelihood of impact in the immediate and short term. These priorities are meant as a starting point. It is recognised that after the initial three years, longer term commitments will be required to ensure progress is sustained over time and that real change is delivered on the ground. A summary of the five priority areas follows.
**Priority Area 1: Increase community awareness of FASD and prevent prenatal exposure to alcohol**

Fundamental to preventing new cases of FASD is the reduction of harmful consumption of alcohol by the general population, and in particular by women during pregnancy. Prevention activities need to target the whole population to raise awareness of the potential risks associated with alcohol consumption during pregnancy and create a supportive environment for women who are pregnant or planning pregnancy to be alcohol-free during this time. This should be done through public education campaigns and mandatory health warning labels on all alcohol products. In addition, targeted prevention initiatives are needed to support women most at risk of having a child with FASD. It is also imperative that all health professionals are able to ask and advise women about their alcohol consumption at any stage of their lives.

1. **Conduct an ongoing national public education campaign about the harms resulting from alcohol consumption during pregnancy.**
   - Funding required: $10.2 million
   Undertake a three year comprehensive public education campaign to raise awareness about the harms associated with alcohol consumption during pregnancy. The campaign should use a range of media, including television, radio, print materials and social media.

2. **Implement mandatory health warning labels on all alcohol products available for sale in Australia.**
   - Funding required: $682,000
   Implement a mandatory, government regulated health warning labelling regime on all alcohol products available for sale in Australia. This regime should be linked to the public education campaign about the harms of alcohol consumption during pregnancy.

3. **Provide specialist support services to pregnant women who have alcohol-related disorders.**
   - Funding required: $3.1 million
   Develop a National Model of Care for women who have alcohol-use disorders with clearly defined referral pathways into treatment. Provide funding for treatment services to develop women-centred practices, with a particular focus on women who are pregnant and develop and evaluate web based interventions to support women who are at risk of alcohol exposed pregnancies.

4. **Educate health professionals on FASD and enable them to routinely ask and advise all women about their alcohol consumption.**
   - Funding is already committed by the Commonwealth Government: $6.1 million
   Publish and distribute the updated Pregnancy Lifescrpts and provide training to health professionals to enable them to routinely ask all women about their alcohol consumption.
Priority Area 2: Improve diagnostic capacity for FASD in Australia

The prevalence of FASD is Australia is believed to be significantly under reported and this is due in part to low diagnosis rates. There is currently no standardised diagnostic instrument and there is limited diagnostic capacity among health professionals in Australia. An evidence-based standardised diagnostic instrument must be implemented, and opportunities for people to be assessed and receive a diagnosis must be provided. Training is also needed for health professionals to both increase their awareness of FASD and facilitate the use of the diagnostic instrument.

2.1 Publish, implement and evaluate the Australian FASD diagnostic instrument.

Funding required: $852,000

Publish and test the draft Australian FASD diagnostic instrument, recently developed by the Australian FASD Collaboration, with funding from the Commonwealth Government. This should be supported by the publication of clinical guidelines on the use of the instrument.

2.2 Establish FASD diagnostic services.

Funding required: $7.3 million

Establish three FASD specific diagnostic clinics across Australia and conduct research into other potential models for delivering FASD diagnostic services in the future. Research to evaluate other FASD diagnostic service models also needs to be undertaken.

2.3 Implement training for health professionals on the use of the Australian FASD diagnostic instrument.

Funding required: $950,000

Provide training to health professionals on the use of the Australian FASD diagnostic instrument. This should be overseen by a consortium of health peak bodies who will allocate grant funding to train health professionals. In addition a FASD diagnostic training workshop should be developed and rolled out across Australia.

Priority Area 3: Enable people with FASD to achieve their full potential

For people with FASD, their parents and carers, having access to disability support funding, services and early intervention programs results in better outcomes throughout their lives. Fundamental to this is the recognition of FASD as a disability, through the inclusion of FASD in eligibility criteria for disability supports. People with FASD also require access to early intervention services and training resources are needed to support those working with people with FASD in education, employment and criminal justice sectors.

3.1 Support people with FASD, their families and carers.

Economic modelling is required to determine accurate funding estimates.

Recognise FASD as a disability by including FASD in the Impairment Tables for Disability Support Pensions, acknowledging FASD in the National Disability Insurance Scheme and listing FASD in the List of Recognised Disabilities for Carer Payments.

3.2 Improve early intervention options for people with FASD, their families and carers.

Funding required: $1.5 million

Expand the current Better Start for Children with Disability initiative to include FASD and provide funding support to parent and carer organisations to support those who care for people with FASD.

3.3 Treat people with FASD in a socially inclusive manner upon entry into education, employment and if in contact with the criminal justice system.

Funding required: $1,067,000

Develop teaching guidelines for educators on teaching people with FASD, research the employment needs of people with FASD, and train judges and magistrates on increasing their awareness of FASD and of appropriate sentencing options for people with FASD.
Priority Area 4: Improve data collection to understand the extent of FASD in Australia

To provide appropriate services for people with FASD, more information is needed on the prevalence of alcohol consumption during pregnancy and the numbers of people with FASD. Currently little information is available on alcohol consumption during pregnancy and no standardised information is collected once a diagnosis is made. This makes it impossible to know the extent of FASD within Australia and the level of service provision that is required to address this.

4.1 Routinely record women’s alcohol consumption during pregnancy.

Funding is already committed by the Commonwealth Government.

Include standardised questions about alcohol consumption during pregnancy, as part of the Perinatal National Minimum Data Set.

4.2 Standardise data collection on FASD diagnosis.

Funding required: $321,000

Pilot a FASD diagnosis register in one state, as a measure to overcome the current situation where surveillance systems for birth defects and congenital anomalies exist but do not record or report FASD in a standard manner.

4.3 Monitor FASD prevalence through the Australian Paediatric Surveillance Unit.

Funding required: $60,000

Undertake a national surveillance study of FASD using the Australian Paediatric Surveillance Unit to gain updated prevalence figures on FASD.

Priority Area 5: Close the gap on the higher prevalence of FASD among Aboriginal and Torres Strait Islander peoples

FASD is more prevalent among Aboriginal and Torres Strait Islander peoples, with the incidence of FAS being between 2.76 and 4.7 per 1,000 births, which is four times the rate of FAS among the general population. Aboriginal and Torres Strait Islander peoples require culturally appropriate diagnostic and treatment services to assist in preventing new cases of FASD and in supporting people who are affected by FASD.

5.1 Provide support to Aboriginal and Torres Strait Islander peoples to develop community-driven solutions to address alcohol misuse.

Funding is already committed by the Commonwealth Government.

Continue to support the development of community-driven solutions to address alcohol misuse, including community initiated alcohol management plans and restrictions.

5.2 Publish resources on FASD that are culturally appropriate and tailored to different cultural groups within Aboriginal and Torres Strait Islander communities.

Funding required: $1.5 million

Establish a small grants scheme for Aboriginal and Torres Strait Islander communities to adapt FASD resources, being produced by the National Drug Research Institute (NDRI), so that they are locally relevant and culturally appropriate.

5.3 Develop comprehensive community responses to FASD in remote and isolated Aboriginal and Torres Strait Islander communities.

Funding required: $6 million

Support remote and isolated Aboriginal and Torres Strait Islander communities to develop a ‘whole of community’ response to FASD. This will enable them to embed changes in their communities over time.
Overarching principles

The priority areas of the Australian FASD Action Plan should be viewed in the context of a broader set of principles which form the foundation of all actions and targets. These are based on evidence-based practice in the prevention and management of health and social issues.

1. Population health framework

The Australian FASD Action Plan must adopt a population health framework which recognises that FASD and alcohol consumption during pregnancy are part of a complex interplay of biological, social, psychological, environmental and economic factors. It also accepts that the antecedents of FASD are not just a matter of personal responsibility and choice. Broad population-based approaches are needed to reduce alcohol-related harms in the Australian community. Fundamental to the success of reducing the occurrence of prenatal alcohol exposure is reducing the harmful consumption of alcohol in the general population and affecting cultural change of alcohol use in Australia.

2. Whole of government approach

A whole of government approach recognises that people with FASD and their carers require support from a range of sectors, at both the Commonwealth and state and territory levels. Support is required from a range of sectors including; employment, health, education, justice (including police, courts, legal practitioners and correctional services), Indigenous organisations, community services and housing services.

3. Human rights-based approach

The Australian Human Rights Commission recommends that ‘a human rights-based approach’ is needed for FASD and that this approach ‘should underpin all measures to address FASD in order to protect and promote the rights of women, children, families and communities affected by FASD’. A human rights-based approach acknowledges the principles of non-discrimination, participation, inclusion, equity and access. These principles should be inherent in the development of FASD policies and programs.

4. Women-centred practice

‘Women centred practice’ or ‘gender-responsiveness’ are terms that consider the needs of women in all aspects of design and delivery, including the location and accessibility of services, staffing, program development, content and materials. Practically this means that services need to offer a safe environment which is free from violence and which encourages trust. Substance use and heavy alcohol consumption during pregnancy is often seen by child welfare and child protection authorities as abuse or neglect. This contributes to the marginalisation of vulnerable women who fear the loss of custody of their children and therefore feel unable to seek help during their pregnancy. To break the cycle, effective services are needed that link prenatal care, treatment programs and child protection services with other health and social services.
### Australian FASD Action Plan framework

For each priority area, areas for action have been established to guide the work to be undertaken by governments. Indicators of change have also been established to ensure that progress can be measured. These actions need to be adopted in full to help prevent new cases of FASD and to provide support and assistance to people with FASD, their families and carers.

<table>
<thead>
<tr>
<th>Priority area</th>
<th>Areas for Action</th>
<th>Indicators of change</th>
</tr>
</thead>
</table>
| 1. Increase community awareness of FASD and prevent prenatal exposure to alcohol | 1.1 Conduct an ongoing national public education campaign about the harms resulting from alcohol consumption during pregnancy.  
1.2 Implement mandatory health warning labels on all alcohol products available for sale in Australia.  
1.3 Provide specialist support services to pregnant women who have alcohol-related disorders.  
1.4 Educate health professionals on FASD and enable them to routinely ask and advise all women about their alcohol consumption. | • By 2014 an evidence-based (Government regulated) mandatory alcohol pregnancy warning label is applied to all alcohol products sold in Australia.  
• By 2016 there is a 20 per cent reduction in the number of women who report consuming alcohol during pregnancy, based on data from the National Drug Strategy Household Survey (NDSHS).  
• By 2016 there is standardised use of the Australian FASD diagnostic instrument among multi-disciplinary teams of child and maternal health professionals.  
• By 2016 20 per cent of all women are routinely screened around their alcohol consumption using AUDIT-C.  
• By 2016 there is increased awareness by 40 per cent of National Health and Medical Research Council Australian Guidelines to Reduce Health Risks from Drinking Alcohol amongst Australians as measured by National Drug Strategy Household Survey (NDSHS).  
• By 2016 FASD is recognised as a disability and people with FASD are eligible to access disability support services and payments. |
| 2. Improve diagnostic capacity for FASD in Australia | 2.1 Publish, implement and evaluate the Australian FASD diagnostic instrument.  
2.2 Establish FASD diagnostic services.  
2.3 Implement training for health professionals on the use of the Australian FASD diagnostic instrument. |  
| 3. Enable people with FASD to achieve their full potential | 3.1 Support people with FASD, their families and carers.  
3.2 Improve early intervention options for people with FASD, their families and carers.  
3.3 Treat people with FASD in a socially inclusive manner upon entry into education, employment and if in contact with the criminal justice system. |  
| 4. Improve data collection to understand the extent of FASD in Australia | 4.1 Routinely record women’s alcohol consumption during pregnancy.  
4.2 Standardise data collection on FASD.  
4.3 Monitor FASD prevalence through the Australian Paediatric Surveillance Unit. | 5.1 Provide support to Aboriginal and Torres Strait Islander peoples to develop community-driven solutions to address alcohol misuse.  
5.2 Publish resources on FASD that are culturally appropriate and tailored to different cultural groups within Aboriginal and Torres Strait Islander communities.  
5.3 Develop comprehensive community responses to FASD in remote and isolated Aboriginal and Torres Strait Islander communities. |
| 5. Close the gap on the higher prevalence of FASD among Aboriginal and Torres Strait Islander peoples |  
|
Costing the Plan

An Australian FASD Action Plan has been estimated to conservatively cost $37 million in funding over three years outlined in the table below and further detail is provided in Appendix C.

<table>
<thead>
<tr>
<th>Action Area</th>
<th>Year 1</th>
<th>Year 2</th>
<th>Year 3</th>
<th>Total</th>
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</thead>
<tbody>
<tr>
<td>1. Increase community awareness of FASD and prevent prenatal exposure to alcohol</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>1.1 Conduct an ongoing national public education campaign about the harms resulting from alcohol consumption during pregnancy</td>
<td>$4,400,000</td>
<td>$2,900,000</td>
<td>$2,900,000</td>
<td>$10,200,000</td>
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<tr>
<td>1.2 Implement mandatory health warning labels on all alcohol products available for sale in Australia</td>
<td>$306,000</td>
<td>$188,000</td>
<td>$188,000</td>
<td>$682,000</td>
</tr>
<tr>
<td>1.3 Provide specialist support services to pregnant women who have alcohol-use disorders</td>
<td>$244,000</td>
<td>$1,358,000</td>
<td>$1,515,000</td>
<td>$3,117,000</td>
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<td>1.4 Educate health professionals on FASD and enable them to routinely ask and advise all women about their alcohol consumption</td>
<td>Already funded through existing Government commitments</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>2. Improve diagnostic capacity for FASD in Australia</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.1 Publish, implement and evaluate the Australian FASD diagnostic instrument</td>
<td>$195,400</td>
<td>$225,600</td>
<td>$431,000</td>
<td>$852,000</td>
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<td>2.2 Establish FASD diagnostic services</td>
<td>$2,610,000</td>
<td>$2,354,000</td>
<td>$2,354,000</td>
<td>$7,318,000</td>
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<td>2.3 Implement training for health professionals on the use of the Australian FASD diagnostic instrument</td>
<td>-nil</td>
<td>$625,000</td>
<td>$325,000</td>
<td>$950,000</td>
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<tr>
<td>3. Enable people with FASD to achieve their full potential</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3.1 Support people with FASD, their families and carers</td>
<td>Economic modelling required</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3.2 Improve early intervention options for people with FASD, their families and carers</td>
<td>$500,000</td>
<td>$500,000</td>
<td>$500,000</td>
<td>$1,500,000</td>
</tr>
<tr>
<td>3.3 Treat people with FASD in a socially inclusive manner upon entry into education, foster care and if in contact with the criminal justice system</td>
<td>$267,000</td>
<td>$450,000</td>
<td>$350,000</td>
<td>$1,067,000</td>
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<td>4. Improve data collection to understand the extent of FASD in the Australia</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4.1 Routinely record women’s alcohol consumption during pregnancy</td>
<td>Already funded through existing Government commitments</td>
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<td></td>
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<tr>
<td>4.2 Standardise data collection on FASD</td>
<td>$107,000</td>
<td>$107,000</td>
<td>$107,000</td>
<td>$321,000</td>
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<td>4.3 Monitor FASD prevalence through the Australian Paediatric Surveillance Unit</td>
<td>$20,000</td>
<td>$20,000</td>
<td>$20,000</td>
<td>$60,000</td>
</tr>
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<td>5. Close the gap on the higher prevalence of FASD among Aboriginal and Torres Strait Islander peoples</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5.1 Provide support for Aboriginal and Torres Strait Islander peoples to develop community-driven solutions to address alcohol misuse</td>
<td>Already funded through existing Government commitments</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5.2 Fund the publication of resources on FASD that are culturally appropriate and tailored to different cultural groups within Aboriginal and Torres Strait Islander communities</td>
<td>$500,000</td>
<td>$500,000</td>
<td>$500,000</td>
<td>$1,500,000</td>
</tr>
<tr>
<td>5.3 Fund the development of comprehensive community responses to FASD in remote and isolated Aboriginal and Torres Strait Islander communities</td>
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<td>$2,000,000</td>
<td>$2,000,000</td>
<td>$6,000,000</td>
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<td>Sub - total</td>
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<td>$11,207,600</td>
<td>$11,170,000</td>
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Governance Structure

An effective Governance structure should be established for the Australian FASD Action Plan. The Plan should be overseen by a FASD Expert Advisory Committee. This Committee should include at least one representative from each of the following: a FASD consumer and carer group, academics, clinicians, and departmental representation from Department of Health and Ageing (DoHA), Department of Families, Housing, Community Services and Indigenous Affairs (FaHCSIA), Department of Education, Employment and Workplace Relations (DEEWR), State and Territory Health Departments and Justice Departments. There should also be Indigenous representation on the Committee.
Priority Area 1: Increase community awareness of FASD and prevent prenatal exposure to alcohol

Fundamental to reducing prenatal exposure to alcohol, is the reduction of harmful consumption of alcohol in the general population. The 2010 National Drug Strategy Household Survey (NDSHS) found that 11.3 per cent of women consumed alcohol at rates that placed them at risk of alcohol-related harm over a lifetime and 29.8 per cent consumed alcohol at rates that placed them at risk of short term harms.¹⁴

In 2009 the National Health and Medical Research Council Australian Guidelines to Reduce Health Risks from Drinking Alcohol (the Guidelines)⁶ were released. The fourth guideline, on maternal alcohol consumption recommends that ‘not drinking’ is the safest option during pregnancy. However, despite the Guidelines being in place for three years, a report commissioned by FARE in 2012 found that only five per cent of Australians were familiar with the Guidelines.¹⁵

Prevention activities need to encompass the whole of the population and aim to raise overall awareness about the harms associated with alcohol consumption during pregnancy.

1.1 Conduct an ongoing national public education campaign about the harms resulting from alcohol consumption during pregnancy

Despite 30 years of research demonstrating that alcohol consumption during pregnancy can harm the fetus, there has been no concerted and comprehensive effort by the Commonwealth Government to raise awareness of these harms. This is reflected in the proportion of women who consume alcohol during pregnancy.

Recent research published by FARE found that 47.3 per cent of women consumed alcohol while pregnant, before knowledge of their pregnancy and that 19.5 per cent of women continued to consume alcohol even after knowledge of their pregnancy.⁴ A separate study of women’s attitudes towards alcohol consumption during pregnancy in 2006 found that 30 per cent of women intended to consume alcohol in a future pregnancy.¹⁶ The study also found that women are more likely to intend to consume alcohol during pregnancy if they lack knowledge about the harms of alcohol consumption to the unborn child.¹⁶
Internationally, public education campaigns have been shown to increase awareness about the risks of consuming alcohol whilst pregnant and awareness about FASD. In Canada public education campaigns have been in place since 1999. The effectiveness of Canadian efforts to raise awareness of FASD and the harms associated with consuming alcohol while pregnant are reflected in their prenatal alcohol consumption rates which are less than 15 per cent.

The promotion of the Guidelines in Australia has been limited and ad hoc. Since the release of the Guidelines, the Department of Health and Ageing (DoHA) has developed limited communication materials, including a specific brochure for pregnant women. Over 700,000 of these resources (including brochures for adults, parents of young people, wallet cards for young people and a poster targeting pregnant women) have been distributed across Australia. These efforts to promote the Guidelines have been largely ineffective, due in part to the ad hoc nature of the programs and the short term funding for these initiatives.

To increase awareness and understanding of the Guidelines, a national, comprehensive and ongoing public education campaign is required. This campaign should have a particular focus on alcohol consumption during pregnancy. It needs to be appropriately resourced, and funded for the lifespan of the Australian FASD Action Plan. The campaign should include targeted messages for specific groups and promote evidence-based messages at both a national and community level. The campaign should use a broad range of media and ensure that there are specific messages for:

- the general public
- women
- their partners, and
- those identified as being at risk.

The consumption of alcohol by people in the woman’s life, especially partners and extended family, can influence alcohol consumption during pregnancy. Those people also play an important role in supporting women to stop or reduce their alcohol consumption during pregnancy. A 1996 study found that around a third of women would stop or reduce their drinking if their partner also stopped drinking for the duration of the pregnancy, and 38 per cent would drink less if their partner encouraged them to stop or cut back.

At a community level, Medicare Locals should reinforce campaign messages to the general population as well as implement targeted communication messages to high risk individuals and communities. Because of their strong connection to primary health care providers, Medicare Locals are an ideal vehicle to deliver and reinforce educational campaigns about alcohol consumption during pregnancy and raise awareness about FASD.

The total cost of a public education campaign over three years is estimated at $10.2 million. These costs include the cost of producing and running a comprehensive campaign comprising of pamphlets, posters, television and radio advertisements. The initial campaign production in year one is estimated at $4.4 million. The campaign would require maintenance and updating as well as ongoing associated running costs, such as purchase of advertising time on television and radio. The ongoing costs are estimated as $2.9 million per year.

**Action:** Establish and deliver a three year public education campaign, using a range of media, about the harms from alcohol consumption during pregnancy, including specific messages and resources for the general public, women and their partners.

**Funding required:** $10.2 million
1.2 Implement mandatory health warning labels on all alcohol products available for sale in Australia

Internationally, at least 18 countries or territories have introduced laws that require the compulsory use of health warning labels on alcohol products. These countries include France, South Africa, Brazil, Costa Rica, Ecuador, Honduras, Mexico, South Korea and the USA. 22-24 Five countries also have mandated pregnancy labels, either pictorial or text indicating that alcohol should not be consumed during pregnancy (China, France, USA, South Africa and the Russian Federation).25

In Australia, food labels including those on alcohol products are the statutory responsibility of Food Standards Australia New Zealand (FSANZ). At present the alcohol industry has a voluntary consumer information labelling scheme with different products having different messages. Pregnancy warning labels have been developed by DrinkWise, an industry funded social aspects organisation. The Drinkwise labels include two pregnancy labels, which are either text stating ‘it is safest not to drink while pregnant’ or a pictorial silhouette of a woman drinking alcohol with a line through it.25

Following a review of food labelling in Australia and New Zealand, published as Labelling Logic in January 2011, the Legislative and Governance Forum of Food Regulation (convening as the Australia and New Zealand Food Regulation Ministerial Council) decided, in December 2011, to support a mandated pregnancy warning label on alcohol products within two years.26 There is currently no indication of the process the Government intends to follow to move towards this mandatory labelling regime.

In 2009, as part of an application by the Alcohol Advisory Council (ALAC) of New Zealand to FSANZ to implement alcohol health warning labels, a review was commissioned: Alcohol warning labels: evidence of impact on alcohol consumption amongst women of childbearing age. 27 The report found that if labels were adopted in Australia, based upon the available literature, they would have the following potential impacts:

- the majority of female drinkers will have noticed the warnings within two to three years
- younger women and heavier drinkers may notice the warnings more
- of those who notice the labels, approximately 50 per cent will recall the message
- there will be an increase in the number of conversations about the risks of alcohol use during pregnancy, and
- behaviour change may occur if the labels are complemented at point of sale and at other message sources. 28

To contribute to awareness raising and have the greatest potential at changing behaviours, an evidence-based alcohol warning label regime is needed in Australia. The labelling regime should be:

- mandatory so the label appears on all products
- applied consistently across all products so they are visible and recognisable
- include a number of rotating messages focusing on different social and health harms
- developed by health behaviour and public health experts
- regulated and enforced by government, and
- accompanied by a national public education campaign.29

The total cost to Government of implementing a mandatory health warning label regime over three years is $682,000. In the first year these costs total $306,000 and include the label development, administration and enforcement. The annual ongoing cost to Government of mandatory alcohol health warning labels has been estimated at $188,000 per year.13

**Action:** Implement a mandatory, government regulated alcohol health warning label regime for all alcohol products available for sale in Australia.

**Funding required:** $682,000
1.3 Provide specialist support services to pregnant women who have alcohol-related disorders

Women with alcohol or substance misuse issues, who are pregnant and/or parents face particular societal condemnation. Unfortunately these women often delay seeking help or support and this can have serious implications for the mother and the fetus. There are many factors that influence alcohol consumption during pregnancy, including being aware of the pregnancy and being aware of the potential harms of alcohol consumption to the fetus and alcohol dependence.

Women who have alcohol-use related disorders or are alcohol dependent are most at risk of having a child or multiple children with FASD. Efforts to support these women to reduce or cease their alcohol consumption are crucial in helping to prevent new cases of FASD. These women should also be advised on the contraception options available to them to help prevent unplanned pregnancies.

Factors that influence alcohol consumption during pregnancy include concurrent drug use, mental health problems, physical and sexual violence, and fewer economic resources and opportunities. Women who experience significant disadvantage are more likely to have a child or multiple children with FASD. A study by the University of Washington of 80 birth mothers of children with FASD, found that all women had alcohol use histories, and 63 had a parent with an alcohol problem. The study also found that, of the 80 birth mothers:

- 96 per cent had mental health disorders (post-traumatic stress, depression and anxiety being most common)
- 95 per cent had been physically or sexually abused during their lifetime, and
- 80 per cent lived with men who did not want them to stop drinking during pregnancy.

Women and in particular pregnant women face significant barriers in accessing treatment for their alcohol use. Women account for only 32 per cent of Australia’s alcohol and other drug treatment episodes and men have been the major clients of alcohol and drug treatment services for the last ten years. Subsequently, most treatment programs in Australia and overseas have been designed with men in mind and it is often difficult for services to take into account gender differences in their treatment options and facilities.

For women with alcohol-related disorders, there are often significant issues in their lives that prevent them from seeking treatment. One of the primary difficulties is the lack of childcare options. Few treatment services provide childcare and for some cultural groups it is very difficult for women to leave their homes and/or family responsibilities in order to undertake or seek treatment. Other barriers to treatment include fear of losing custody of children; needing their partner’s permission to attend treatment; fear that
their partner will leave them; stigma and shame that people might identify them as having a problem with alcohol; fear of withdrawal and a belief that they should be able to stop drinking on their own. There is also a lack of services for pregnant women, lack of information about treatment options and lack of priority access. To address the barriers to access and engagement in alcohol and drug treatment services, it is important that these services are modified to better accommodate the needs of women.

There is growing potential for women to access support through online alcohol assessments and interventions. These have been shown to have a positive effect on the levels of alcohol consumption by low-income women. This was regardless of whether the women received personalised feedback or general information about alcohol’s health impacts and FASD. Another study showed that over half of women who were deemed at risk of having an alcohol-exposed pregnancy (i.e. any alcohol consumption in the previous 30 days and were not using reliable contraception) were no longer at risk after enrolling in and completing the self-guided online change intervention. A further example is the use of a ‘parent supporter in alcohol, drugs and addiction’ on the popular website Netmums in the UK. This ‘parent support’ was provided by Swanswell’s substance misuse workers to answer questions relating to alcohol and other drugs.

It is important that women who are at high-risk of an alcohol exposed pregnancy are referred to appropriate services. The most effective way to ensure that this occurs is through the development of a model of care in each state and territory. The West Australian Department of Health, Child and Youth Health Network Model of Care for FASD outlines that clear referral pathways are needed between GPs, maternity and newborn services and alcohol and other drug services to ensure comprehensive support for all pregnant women, including those in rural and remote regions. The WA Model of Care also highlights the need to develop protocols for multi-disciplinary inter-sectoral approaches to support pregnant women with alcohol use disorders over their life course.

The development of the West Australia Model of Care in 2010-2011 was led by a project officer (0.6FTE*) with three development meetings held. These were: an implementation planning meeting; a project control group meeting, to which experts provided their time in kind; and a large forum with 100 people in attendance. The cost of the project officer and various meetings is estimated as being $60,000.

* FTE – Full time equivalent position
Western Australia is now developing an implementation plan for the Model of Care, which is due for publication in early 2013. This plan will outline the roles and responsibilities for each Government agency. These actions have been negotiated with and assigned to each agency and the plan will include measures for implementation and reporting mechanisms. This is a complex process requiring system-wide change. The development of the implementation plan has involved more than 60 organisations and engagement strategies across the state.

It is recommended that a National Model of Care be developed, with each state and territory establishing their own implementation plans.40 The total cost of developing a National Model of Care and state-based implementation plans is estimated at $517,000. The National Model of Care is estimated to cost $146,000 over three years. This includes a series of workshops in each state and territory with relevant authorities ($48,000), salaries for project officers to oversee the drafting and promotion ($98,000). Each state-based implementation plan is expected to cost $53,000, made up of salaries ($44,000), a consultation workshop ($6,000) and promotion ($3,000). The development of the implement plans in each state and territory would need to be overseen by a working group.

The total cost of making alcohol and drug treatment services more suitable for women and pregnant women with alcohol-use disorder, is estimated at $2.1 million over three years. This consists of a scoping study in the first year and a small grants funding round in the second and third years. The scoping study is estimated to cost $100,000, consisting of quantitative and qualitative research with alcohol and other drug treatment providers and focus groups with pregnant women. This is costed at $35,000 with project implementation (led by a full time project officer for 12 months) estimated at $65,000. A total of $2 million should also be committed to the small grants funding round to improve specialist support to pregnant women with alcohol-use disorders. These grants would be capped at $100,000, with up to $1 million being available in each year. This would allow 20 services over two years to adopt women-centred practice.

The total cost of developing, testing and evaluating an online intervention program for women who are planning pregnancy, pregnant and/or parents would be $500,000 over three years. This includes $100,000 for website development, $200,000 for counselling support, $100,000 for project management and promotion. A further $100,000 should be dedicated to the evaluation of the program.

‘There are many factors that influence alcohol consumption during pregnancy, including being aware of the pregnancy and being aware of the potential harms of alcohol consumption to the fetus and alcohol dependence.’

Actions:

• Develop state and territory based models of care for women who have alcohol use disorders with clearly defined referral pathways into treatment ($517,000).

• Provide funding to alcohol and drug treatment services to allow them to develop women-centred practices, with a particular focus on women who are pregnant ($2.1 million).

• Develop and evaluate an online intervention program to support women at risk of alcohol exposed pregnancies ($500,000).

Funding required: $3.1 million over three years.
1.4 Educate health professionals on FASD and enable them to routinely ask and advise all women about their alcohol consumption

Australian women consider health professionals to be the best source of information regarding their pregnancy. Women are often willing to make changes to their lifestyle, diet and alcohol consumption if advised to do so, and pregnancy can be a ‘teachable moment’ or a critical window of opportunity for change. However health professionals are often reluctant to discuss alcohol consumption with women due to fear of upsetting the woman, time pressures or their own discomfort. A national poll carried out by FARE in 2012 found that just over a third of the mothers surveyed could recall having had a healthcare professional raise with them the harms associated with alcohol consumption (37 per cent).

It is vitally important that all health professionals, including General Practitioners (GPs) are trained to ask women about their alcohol consumption. Every time a health professional sees a woman, there is potential to prevent a new case of FASD and provide a consistent message on the harms of alcohol consumption during pregnancy.

An Australian feasibility study, Asking QUestions about Alcohol in pregnancy (AQUA), examined the questions that health professionals should ask about alcohol consumption during pregnancy. The study found that women should be screened for their alcohol intake using a validated instrument which includes an assessment of consumption patterns and instructions for the practitioner on how to interpret and discuss the information with the woman. The study concluded that a mechanism for this already exists through the Lifescripts – Advice for Healthy Living project at DoHA.

Lifescripts are used by GPs to address lifestyle risk factors across the population, such as smoking, nutrition, alcohol consumption and physical inactivity. Lifescripts are a national initiative, funded and developed by DoHA and supported and promoted by the Australian General Practice Network. Lifescripts were first introduced in the 2003-04 DoHA budget with an investment of $4.3 million towards their development. In 2007 the Government invested further funding to maximise the uptake of the program.
The Pregnancy Lifescr ipt was developed in 2007 to assist women in having healthy pregnancies.45 This script had a special focus on alcohol consumption during pregnancy and was designed for use by GPs and practice nurses. During 2010-2011 the Pregnancy Lifescr ipt s were reviewed and updated versions were scheduled to be released in August 2011. To date these have not been published. The Lifescr ipt resources also include posters for doctors’ waiting rooms, patient brochures and assessment and prescription pads for use by the GP.

The total cost of the Lifescr ipt s program has been $5.5 million from 2003-04 to 2010-11. The Government has already committed this funding to the Lifescr ipt s program.

The total cost of training health professionals on delivering information on alcohol consumption over three years is $650,450. DoHA has provided funding to FARE to work with health professional bodies to develop appropriate training to raise awareness of the Guidelines among health professionals, and to encourage them to discuss alcohol consumption with all consumers.46

**Actions:**

- Publish and distribute the updated Pregnancy Lifescr ipt s to GPs, to encourage discussions about alcohol consumption during pregnancy ($5.5 million already committed by Government for the complete Lifescr ipt s program).

- Provide training to GPs and other relevant health professional bodies on how best to raise the issue of alcohol consumption with consumers, particularly with pregnant women ($650,450 already committed by Government).

**Funding already committed by Government:** $6,150,450
FASD is often described as an ‘invisible’ disability due to the underlying brain damage caused by prenatal alcohol exposure. This alcohol exposure can result in a variety of problems including difficulties with speech and language; impairment of vision and hearing; organ damage and difficulty with judgement, reasoning and behaviour.1 Most people who are born with FASD do not display some or any of the physical traits that are characterised by the condition.2 Even FAS, which is commonly associated with abnormal facial features, may be difficult to diagnose and assess in newborns and across different racial groups.

Obtaining a diagnosis of FASD can improve an individual’s opportunities in life. A diagnosis can allow an understanding of the specific deficits affecting that individual, which in turn can facilitate communication between health professionals, educators, families and carers on effective interventions and the appropriate supports needed.47,48

However a diagnosis should never be an endpoint. The process to confirm a diagnosis should also identify the appropriate health care, education, and service needs of the individual and the families/carers.49

2.1 Publish, implement and evaluate the Australian FASD diagnostic instrument

Australia currently has no screening and diagnostic instrument for FASD. When diagnosing FASD in Australia, health professionals rely upon a combination of overseas diagnostic instruments, including the:

- FASD Canadian Guidelines for diagnosis50
- ‘University of Washington 4-Digit Diagnostic code’51
- Center for Disease Control Guidelines: ‘Fetal Alcohol Syndrome: Guidelines for referral and diagnosis’ in the USA.49

Canada is the only country that has nationally consistent diagnostic guidelines. These guidelines have facilitated consistent diagnostic practice across the country and allowed for comparable data on FASD to be collected and monitored over time.52,53

In Australia, in 2010, the Commonwealth Government provided $450,000 in funding for the development of a ‘Screening and Diagnostic Instrument for FASD in Australia’. The funding was allocated to the Australian FASD Collaboration, which involved researchers from across the country5 and was led by Professor Elizabeth Elliott and Winthrop Research Professor Carol Bower. The FASD Collaboration undertook considerable work to develop a national diagnostic instrument for FASD and submitted a final report to DoHA in May 2012.54 The report included a systematic literature

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1 The Australian FASD Collaboration is made up of the following researchers: Lead Investigators: Professor Elizabeth Elliott (University of Sydney); Winthrop Research Professor Carol Bower (Telethon Institute of Child Health Research); Senior Consultants: Dr Lucinda Burns (National Drug and Alcohol Research Centre); Ms Heather D’Antoine (Menzies School of Health Research); Ms Maureen Carter (Nindilingarri Cultural Health Services); Dr James Fitzpatrick (Sydney Medical School); Associate Professor Jane Halliday (Murdock Children’s Research Institute); Ms Lorian Hayes (University of Queensland); Associate Professor Jane Latimer (George Institute for International Health, Sydney Medical School); Ms Anne McKenzie (University of Western Australia); Ms Sue Miers (National Organisation for Fetal Alcohol Syndrome and Related Disorders); Dr Raewyn Mutch (WA Department of Health); Dr Colleen O’Leary (Curtin University of Technology and Telethon Institute for Child Health Research); Ms Jan Payne (Telethon Institute for Child Health Research); Dr Elizabeth Peardon (University of Sydney); Ms Elizabeth Russell (Russell Family Fetal Alcohol Disorders Association); Dr Amanda Wilkins (WA Department of Health); Ms Heather Jones (Telethon Institute for Child Health Research) and Dr Rochelle Watkins (Telethon Institute for Child Health Research).
A review on screening and diagnostic assessment as well as an examination of FASD screening programs and diagnostic guidelines from across the world. The report also included a summary of consumer and community input into the design and implementation of screening and diagnosis for FASD in Australia.54,55

The diagnostic instrument developed now requires evaluation in a range of clinical environments across Australia prior to its implementation. Detailed guidelines on its use and resources for health professionals also need to be developed.

In total the cost of finalising and evaluating the diagnostic instrument would be $852,000.56 Based on costs from the previous developmental work, it is estimated that the evaluation and finalisation of the diagnostic instrument would cost a further $562,000 over two and a half years. This includes a national consultation and expert review process ($25,000) and pilot testing ($85,000). The remainder would be spent on salaries of $452,000.

The development of training resources on the diagnostic instrument would run in parallel with the evaluation of the instrument in the third year and is estimated at $290,000 over one year. This includes $180,000 on salaries, $35,000 on the development of resources, $30,000 on production costs and $45,000 in evaluation.56

**Action:** Publish the Australian FASD diagnostic instrument and develop guidelines for its use.

**Funding required:** $852,000 over three years.

### 2.2 Establish FASD diagnostic services

An improvement of FASD diagnosis rates would result in people with the condition receiving greater assistance and support, while also improving awareness of FASD among the Australian community. Once the Australian FASD diagnostic instrument and guidelines are published, the assessment and diagnosis of individuals via multi-disciplinary health teams needs to occur. A FASD diagnosis is determined through a multi-disciplinary approach with assessments undertaken by a range of health professionals including paediatricians, clinical psychologists, occupational therapists, speech and language therapists, physiotherapists and social workers.

There can be considerable stigma associated with a FASD diagnosis and the communication of this diagnosis with the individual, family and carers requires particular sensitivities. For a biological mother, a diagnosis of FASD for her child may be very confronting. It is important that support is available to the family and carers to understand the diagnosis and cope with the changes to their lives that it entails.

Four possible FASD diagnostic service models are explained in further detail below.

#### Service model one: specific dedicated FASD diagnostic clinics

Australia has one dedicated FASD diagnostic clinic, which is funded by FARE. This clinic opens fortnightly and is based at the Children’s Development Unit, within The Children’s Hospital at Westmead in Sydney.57

Children (aged 0 to 16 years) who are referred to the clinic undergo a comprehensive assessment consisting of full history and medical checks as well as assessments in developmental and/or neuropsychology issues, speech and language, as well as occupational and physiotherapy developmental issues. As part of the diagnosis, children are photographed for analysis of facial features and referred on for other investigations such as brain scans, genetic testing and hearing and vision assessments where necessary.57 In this model the child is initially seen by a paediatrician and then referred to the other specialists for further tests. To make a
diagnosis the multi-disciplinary team reviews the results from all of the assessments and recommends a final diagnosis.

This model allows for specialist teams to focus on the diagnosis of FASD and would result in teams of health professionals specifically trained in the diagnosis of FASD. The two limitations of this model are that firstly it takes considerable time for the child to complete all of the assessments. They are only referred onto the next assessment when the first is completed. This means that assessments could take a number of months to complete. Secondly, dependent on hospital policy, this model would only be able to accept patients up to the age of 16 as the clinic sits within the remit of a children's hospital.

\[\text{An improvement of FASD diagnosis rates would result in people with the condition receiving greater assistance and support, while also improving awareness of FASD among the Australian community.}\]

Service model two: Using existing child development services to diagnose FASD

The second service model uses existing Child Development Services, usually located in hospitals to assess children for FASD. These services exist across Australia, although they are known by different names in different states\(^5\) (e.g. in South Australia these services are called Early Childhood Intervention Programs). There is also a lack of consistency on what conditions and age ranges of children that the different programs will assess.\(^5\)

In Western Australia there are 11 Child Development Centres across the state that provide a range of supports for children (up to 16 years of age) who have or are at risk of developmental difficulties. The services are made up of multi-disciplinary teams including speech pathologists, occupational therapists, paediatricians and medical officers, physiotherapists, social workers and clinical psychologists.\(^5\)

In 2010, the Western Australian Government committed $49.7 million to improve access to child development services across the state\(^6\) and recommended that the existing ‘Child Development Service: West Perth and State’ located at Princess Margaret Hospital undertake screening and diagnosis of children with FASD with joint assessments between health agencies and other services.\(^38\)

For adolescents and adults who are unable to be assessed through Child Development Services, the West Australian Model of Care for FASD recommends that:

- adolescents be assessed by the Child and Adolescent Mental Health Service and Complex Attention and Hyperactivity Disorders Service in WA
- adult clients should be seen through the Neuro-Psychiatric Service of the Adult Mental Health Service.\(^38\)

Funding for this model would be dependent upon the state or territory in which the model was being applied and the existing services structures.

This model allows for the use of existing services to diagnose FASD. These services already utilise a multi-disciplinary approach so the health professionals have the skills and experience to undertake the work. One concern with this model is that these services are already over-stretched and have lengthy waiting periods.

Service model three: Creating FASD diagnostic teams to target at-risk communities

A third model for diagnosis in Australia is the approach that was used in Marulu: the Lililwan Project in the Fitzroy Valley of Western Australia. This model may be more appropriate for rural and remote communities. As part of the Lililwan project all children between the ages of seven and eight were assessed by a specialist multi-disciplinary team that travelled to the community.\(^6\)

\(^5\) Across Australia these are known as: ‘Child Development Units’, ‘Child Development Centres’, ‘Developmental Assessment clinics’, ‘Child Development Clinics’ and ‘Child and Adolescent Mental Health Services’.
was also gathered on early life trauma based on questions from the *Australian Longitudinal Study of Indigenous Children 2008*. This model saw the multi-disciplinary team assess the children at the same time, rather than complete separate assessments over a number of weeks or months.62

The cost of this model would be dependent upon the number of children in the community in which this model would be applied. Along with philanthropic funding, *Marulu: the Liliwan Project* received $1.7 million from the Department of Families, Housing, Community Services and Indigenous Affairs (FaHCSIA) and DoHA63 in 2010.

While this model allows for FASD diagnostic assessments for whole communities to be carried out at the same time, it may only be appropriate for high-risk communities due to the coordination and travel expenses for the specialists. Key to the success of this model is the need for it to be adapted to the specific community to ensure the cultural appropriateness of the approach.

**Service model four: A local remote/regional diagnostic team**

The experience in the Fitzroy Valley in Western Australia highlights that over time it may be possible to develop health services based in remote regions that are able to assess and diagnose children with FASD. This would require trained health professionals who live in the region and are able to travel to remote areas.

Community Health Services are located throughout Australia and usually employ social workers, occupational therapists, speech therapists, psychologists, paediatricians and specialist alcohol and other drug or mental health workers. It is possible that the staff working for these services could be trained to become FASD diagnosis teams. This would build the capacity of local services.

In Western Australia the Pilbara Community Drug Service Team, based in South Hedland provides outreach services to Port Hedland, Karratha, Onslow, Tom Price and Newman and is one of 12 community-based alcohol and other drug programs that cover the state. The Pilbara team also runs the *Alcohol and pregnancy: what are the risks?* initiative in conjunction with the Pilbara Health Partnership.64 This provides counselling and education for communities throughout the Pilbara region on alcohol and pregnancy. It is possible that a team like this could be broadened to include a range of health disciplines necessary to undertake FASD diagnosis work.

It is important that this model be flexible enough for different areas and local configurations. It is also possible that in time this service model could be provided using tele-health services. Tele-health uses high-speed broadband to deliver services via video link to consult with a patient, and the rapid transfer of files including scans to enable health professionals to participate in a virtual environment without travelling long distances.65 The Apunipima Cape York Council has successfully undertaken its first tele-health consultation in June 2012 with three patients located in Mossman, a township located approximately 90 kilometres north of Cairns.66

This model would allow for FASD diagnostic assessments to be undertaken within existing services, by local people who already work and live in the community. However, the model relies heavily on the skills, training and capacity of professionals working at the
Community Health Services in undertaking diagnostic assessments and requires significant long term funding to embed practices and become sustainable.

Overview of models

These different models provide a range of options for diagnosing FASD in rural, remote and urban settings. It is important that as these models are implemented, that work is undertaken to examine the cost effectiveness of each model and to determine the effectiveness of different models in different locations e.g. rural versus urban settings, specialist clinic versus existing Child Development Services.

In the first instance, three FASD clinics based on model one should be developed throughout Australia. These clinics should be based on the FASD Diagnostic Clinic at The Children’s Hospital at Westmead. The total cost of developing and operating the three diagnostic clinics over three years is estimated to be $3.6 million. This is based on the funding provided to the FASD Diagnostic Clinic at The Children’s Hospital at Westmead, which has received $184,000 for a 12 month pilot project. Of this, salaries account for $151,000, which consist of a General Paediatrician (0.1 FTE), Developmental Paediatrician (0.2 FTE), Clinical Psychologist (0.1 FTE), Speech Pathologist (0.1 FTE), Occupational Therapist (0.1 FTE), Physiotherapist (0.1 FTE) and Administration Officer (0.1 FTE).

This clinic is currently funded to operate one day per fortnight, and to be truly effective it would be preferable for any Dedicated FASD clinic to operate at least one day per week. It is therefore reasonable to double the current funding of the Westmead Clinic to provide an effective service. It is believed that at least three such Specific FASD clinics are required across Australia. Using the doubled costs of the Westmead clinic as a guide (approximately $400,000 per year), three clinics would require $1.2 million per annum.

Research on the other service models also needs to be undertaken. Paediatric Registrar Dr James Fitzpatrick, with the The George Institute for Global Health has recently developed a funding proposal for a trial of paediatric and child health care being delivered by a multi-disciplinary team in Fitzroy Valley schools. This model would see health and education professionals coordinating FASD diagnosis and management within the school system. There is strong support from health and education partners to trial this project from 2013-2015. Private sector funding has been secured to undertake community consultation and pilot the model on a small scale.

The model is similar to service model four and is estimated to cost $1.8 million over three years. This is comprised of $705,000 in the first year, (including $73,000 in set up costs) and $577,000 in the following two years. The operation costs in all three years include three members of staff (one clinical researcher, one coordinator and an Aboriginal Health Worker) estimated at $467,000 per year, administration and management ($40,000), promotion and resources ($5,000 per year), a team vehicle ($100,000 including purchase, maintenance and repair) and research and evaluation ($50,000 per year).

A similar project should be established in another region, allowing two research projects to take place. The total estimated cost of two research projects to implement and evaluate different FASD diagnostic service models would be $3.7 million over three years. This would allow for each project to be awarded $1.8 million over three years.

Actions:

- Establish FASD specific diagnostic clinics operating for one day per week, in three locations across Australia ($3.6 million).
- Undertake two research projects to establish and evaluate different FASD diagnostic service models in three locations ($3.7 million).

Funding required: $7.3 million over three years.

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FTE - Full time equivalent position
2.3 Implement training for health professionals on use of the Australian FASD diagnostic instrument

Greater knowledge, awareness and understanding of FASD is needed among health and medical professionals to improve the diagnosis of FASD. In particular a training and implementation plan should be developed alongside the guidelines for the Australian FASD diagnostic instrument, in order to train health and medical professionals on its use.

Health professionals have a key role to play in improving the diagnosis of FASD; however few health professionals are aware of the condition or feel equipped to manage patients with FASD. In a study of 1,143 health professionals in Western Australia in 2005, only 12 per cent were able to correctly identify all four essential features of FAS and only two per cent considered that they were properly skilled to manage an individual with FAS. When a similar study was conducted with paediatricians (n=132), it found that only 18.9 per cent identified all essential diagnostic features for FAS and that while 76.5 per cent had suspected a case was FAS they had not diagnosed it.

GPs also have an important role to play in the diagnosis and prevention of FASD. Most people’s health care needs and information starts with a health professional, often in a consultation setting with a GP. This is also the public’s preferred method for receiving information about their health concerns. A Western Australian study in 2005 found that only 20 per cent of GPs surveyed could correctly identify the four essential features of FAS. A third of GPs (35 per cent) had suspected that a child had FAS but did not make a diagnosis. GPs most often requested education materials for themselves (75 per cent), their patients (69 per cent) and diagnostic checklists and referral resources to assist them.

Most (82 per cent) of the health and medical professionals in the West Australian survey agreed that an early diagnosis would improve the treatment plans for the child but did not make a diagnosis or refer the child as they were concerned that the diagnosis would stigmatise both the child and their family. This is unfortunate as early diagnosis of FASD can improve the overall life outcomes for the individual and enable families and carers to access disability support services otherwise unavailable to them.
To date, training opportunities on FASD have been limited. The Russell Family Fetal Alcohol Disorders Association (RFFADA), in conjunction with Training Connections Australia, currently offers ten modules on FASD for alcohol and other drug workers; mental health workers; teachers and the criminal justice sector.\textsuperscript{70} Other initiatives have included the Drug and Alcohol Services South Australia’s guide for midwives on FASD in 2006. This Guide outlines key information on FASD, strategies on how to ask women about their alcohol consumption during pregnancy and how to identify FASD in children.\textsuperscript{71} The Drug and Alcohol Office in Western Australia also produces resources for health professionals on FASD and the prevention of prenatal alcohol exposure.\textsuperscript{41}

The West Australian Government, through the Model of Care, has acknowledged the importance of training health professionals in FASD and recommends training at: under-graduate; post-graduate levels and in-service training programs for:

- antenatal and maternity care providers including midwives, GPs, obstetricians and others
- child health nurses and school health nurses
- child development service providers including allied health professionals
- paediatricians and neonatologists
- Aboriginal health workers
- health promotion officers, and
- drug and alcohol service providers.\textsuperscript{58}

The training of Australian health professionals in FASD needs concerted effort and Australia could learn from programs in the USA that were developed to systemically train health and medical professionals about FASD. In 2009 the Center for Disease Control and Prevention (CDC) developed the ‘FASD Competency-Based Curriculum Development Guide for Medical and Allied Health Education and Practice’\textsuperscript{72} and funded five university-based FASD Regional Training Centres to implement this training. Each of the five training centres received between $200,000 and $350,000 (USD) per year, to a total of $4.5 million over three years.\textsuperscript{73}

In 2010 the Royal Australian College of General Practitioners (RACGP) received $409,000 to administer a small grants funding round to train providers to deliver accredited psychological skills training to GPs.\textsuperscript{74} The RACGP awarded 37 grants with each grant delivering a minimum of 20 hours training to 13 participants (on average). A total of 490 people were trained through these grants.

The Commonwealth Government could fund a similar model to train health professionals on the use of the Australian FASD diagnostic instrument. This could be done through the development of a consortium body to oversee the implementation of this training and distribute grant funding. The consortium would consist of representatives from the peak bodies involved in FASD diagnosis. This includes the Paediatric College within the Royal Australasian College of Physicians, the Royal Australian College of General Practitioners and relevant allied health peaks (e.g. Australian Psychological Society, Australian Agency for Social Workers, Occupational Therapists Australia, Physiotherapists Australia and Speech Pathology Australia). This consortium would then disburse small grants funding to training providers to deliver training on use of the Australian FASD diagnostic instrument.

‘The training of Australian health professionals in FASD needs concerted effort and Australia could learn from programs in the USA that were developed to systemically train health and medical professionals about FASD.’

The total cost of these training grants is estimated at $650,000 over two years and would commence once the diagnostic instrument and guidelines have been published. This funding is based on 500 health
professionals being trained at a cost of $1,000 each, and $150,000 towards program management and funds for the consortium.

In addition to the small grants training, it is recommended that five hands-on training workshops with health professionals on FASD diagnosis and management be provided across Australia.75 These training courses would be led by overseas diagnostic experts and include an overview on FASD, case scenario diagnostic evaluations and hands-on practice sessions. The training would run for three days.

The total cost of these training workshops is estimated at $300,000 and would take place in the second year of the Australian FASD Action Plan, once the Australian FASD diagnostic instrument has been published. Funding for the training course is based on $35,000 being available for international and domestic flights for the diagnostic experts. The remainder of the funding ($265,000) would be divided between venue and catering costs of $150,000 (based on $30,000 per course – for a three day course in five jurisdictions*). The remaining $115,000 would go towards the development of course materials, management and administration.

**Actions:**

- Train health professionals in the use of Australian FASD diagnostic instrument by funding a small grants round for training providers to train health professionals, overseen by a consortium of relevant health peak bodies ($650,000).
- Carry out five practical FASD diagnostic training workshops across Australia, led by international FASD experts ($300,000).

**Funding required:** $950,000 over two years.

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*These figures are based on $10,000 per day per training course, made up of $1,500 for catering, $2,000 for audio visual hire and $6,500 for venue hire. Total of three day course is $30,000 in each jurisdiction.
A landmark Commonwealth Government report in 2009 called: ‘Shut out: The experiences of people with disabilities and their families in Australia’ stated that “people with disabilities may be present in the community but most do not enjoy full participation in it.” This is apparent for people with FASD. Unfortunately FASD is not consistently identified as a disability in Australia. As a result people with FASD, their families and carers struggle to access assistance from social services, education and training, justice and health agencies.

The Productivity Commission’s report into ‘Disability Care and Support’ in Australia, published in 2011 found people with disabilities and their families are both managers and advocates of their conditions, as they have to deal with concurrent service providers, government agencies and health professionals. This echoes the frustration expressed by families and carers of people with FASD on the lack of knowledge on FASD by service providers and of the need to educate professionals themselves on the condition. This often leads to more stress for the family, especially when personal experiences are ignored or minimised.

Access to disability support services and early intervention programs are crucial in preventing the development of secondary disabilities for people with FASD. Secondary disabilities (such as mental health issues, alcohol and drug problems, disrupted schooling, lack of employment and incarceration) can occur when FASD is undiagnosed or misunderstood. Similar to other disabilities, access to early intervention services will result in better outcomes for the individual throughout their life.

### 3.1 Support people with FASD, their families and carers

Across Australia, people with FASD, their families and carers have difficulties in accessing disability support services and funding. Many are precluded due to lack of diagnosis from a health professional or because FASD is excluded from eligibility criteria. There are also differences in the supports available to children and adults and differences between state and territory services.

For adults in Australia access to the Disability Support Pension is determined through the Social Security Act 1991 (Cth) and the application of the ‘Tables for the Assessment of Work-related
It is unfortunate that FASD has not been recognised in these initiatives and it is imperative that FASD be recognised in the new National Disability Insurance Scheme (NDIS). The creation of the NDIS was recommended by the Productivity Commission as a mechanism to fund long-term care and support (but not income replacement) for people with significant disabilities.78

The Commonwealth Government is currently working with the states and territories to design the scheme, which is scheduled to be rolled out in mid-2013 and in place by 2018-19.83 Between now and 2018 it is important that people with FASD, their families and carers are adequately represented in NDIS eligibility criteria and that changes are made to existing services and initiatives to recognise FASD.

Determining the costs of extending disability services and carers allowances to include FASD is difficult because of the uncertainty in FASD prevalence figures in Australia. Economic modelling is required to determine accurate estimates of the number of people who may be eligible. This modelling should be undertaken by Treasury and FaHSCIA.

‘Across Australia, people with FASD, their families and carers have difficulties in accessing disability support services and funding.’

Impairment’ (hereafter called Impairment Tables). These tables79 have been revised under the Social Security and Other Legislation Amendment Bill 2011 (Cth) and individuals have been assessed under these tables since January 2012.

FASD, has, for the first time been recognised in these Impairment Tables under ‘Table 9: Intellectual Function’ but there is a qualification that the individual has a low IQ. This is unfortunate as although FAS is associated with lower IQ, 75 to 80 per cent of people with FASD have IQs within the normal range.77 This, therefore, precludes most people with FASD. To amend this situation FASD should be listed as a condition under ‘Table 7: Brain Function’ alongside “a person with Autism Spectrum Disorder who does not have a low IQ.”80

For children with FASD little support is available. From 2008 to 2011 the Commonwealth Government invested $190 million into the ‘Helping Children with Autism’ package. This allowed for the funding of individual assistance packages for children with autism or any other pervasive developmental disorder (though excluding FASD), their families and carers.81 The ‘Helping Children with Autism’ package involved cross government working with DoHA, FaHCSIA and the Department of Education, Employment and Workplace Relations (DEEWR) to deliver the program.

People who care for children under 16 with a disability can access carers payments. Children with disabilities who automatically qualify for these payments are those recognised in the ‘List of Registered Disabilities.’82 FASD is not currently included in this list and is not adequately covered by any other disabilities on the list. It is critical that this situation is changed and that FASD be added to this list.

Actions:

• For adults: recognise FASD as a condition under Table 7: Brain Function in the Impairment Tables for the Disability Support Pension as well as in Table 9: Intellectual Disability in the Impairment Tables.f
• For carers of children under 16: recognise FASD in the ‘List of Recognised Disabilities.’
• Recognise FASD in the National Disability Insurance Scheme.

Funding required: Economic modelling required by Treasury and FaHCSIA to determine numbers of people who may be eligible.

1 There will be cost in adding people with FASD to these Impairment Tables but it is impossible to quantify the number of people who will qualify to receive Disability Support Pensions as the number of adults with FASD is unknown.
3.2 Improve early intervention options for people with FASD, their families and carers

In order to reach their full potential, children with FASD require the same level of access to early intervention services as children with other disabilities. The Commonwealth Government recommended in its best practice guidelines for Autism Spectrum Disorders that a child receive a minimum of 20 hours a week of early intervention services for two or more years in order to make major gains. Children with FASD need comparable levels of service provision. The CDC in the USA recommended that early intervention services are needed for children from birth to three years of age to help in the development of language, walking, and interaction with others.

To achieve the same level of access to early intervention services as that received by other children with disabilities, it is imperative that FASD is recognised in the Commonwealth Government’s ‘Better Start for Children with Disability’ initiative.

The ‘Better Start for Children with Disability’ initiative started in 2011 and provides assistance to eligible children up to the age of 13 years. This assistance includes:

- up to $12,000 in funding for early intervention services and treatments
- assistance for children who live in outer-regional, rural or remote locations to access services
- a treatment and management plan to be developed and covered through Medicare, and
- funding for up to 20 allied health services up to the age of 15 years (provided a treatment and management plan is in place before the age of 13 years).

The Commonwealth Government has provided $122 million to the ‘Better Start for Children with Disability’ initiative over four years (commencing in 2011).

While early diagnosis of FASD is associated with fewer secondary disabilities, the Canadian Paediatric Society and the CDC in the USA both state that FASD intervention programs should not be dependent on a formal diagnosis. This is because the ‘window of opportunity’ for dealing with behavioural abnormalities and preventing secondary disabilities is often missed.

In the USA, in 2009, five intervention programs for people with FASD were assessed by the Interventions for Children with Fetal Alcohol Spectrum Disorders Research Consortium. It found that successful early intervention programs for FASD had the following elements:

- a component of education or training for parents that was built into the program
- giving explicit instructions to the child when learning new skills, rather than the child learning through observation and processing of information alone, and
- programs and techniques were integrated into existing community services, such as special education, therapy or counselling services that the child was already attending.

Other strategies such as educational, psycho-social, and pharmacological approaches that include nutrition and physical

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Eligible children are those who are under six and have a diagnosis of: Down syndrome, cerebral palsy, Fragile X syndrome; or a moderate or greater vision/hearing impairment including deafblindness. These categories were determined by the effectiveness of early intervention programs to be able to prepare these children for school.
therapies are being studied. However the evidence-base for these programs is limited and they require further testing and evaluation.

People who care for those with FASD also need access to support. In 2002 the Victorian Order of Nurses for Canada undertook a four year project on parenting strategies for children with FASD. The main outcome of this project was the development of the ‘Let’s Talk FASD’ guidelines. These guidelines were shaped by the first-hand experiences of people living with FASD and are a collation of parent-driven strategies to care for children and adults with FASD.

In Australia support to parents and carers of people with FASD is provided by two organisations. These are RFFADA and the National Organisation for Fetal Alcohol Syndrome and Related Disorders (NOFASARD). In 2012 NOFASARD received $750,000 over three years from DoHA to continue to support people with FASD and those who care for them. Continued funding is needed to support these organisations and to expand these services into other states and territories. This funding should match the funding provided to NOFASARD.

The total cost of funding two other organisations to provide support to parents and carers of people with FASD is estimated at $1.5 million over three years. This is based on the current funding being provided to NOFASARD and its extension into two other jurisdictions.

### 3.3 Treat people with FASD in a socially inclusive manner upon entry into education, employment and if in contact with the criminal justice system

People with FASD often face a range of health, social and educational difficulties. Approximately 85 per cent of children with FASD do not live with their birth parents and are at risk of multiple foster placements. In the USA, it is estimated that 70 per cent of children in foster care are affected by prenatal alcohol exposure to some degree. Unfortunately similar data are not available in Australia.

FASD can directly diminish a child’s success at school. The underlying brain damage can limit how well a person with FASD may process information, understand and meet the expectations set for them. For example: concentrating in a classroom; sitting still for long periods of time and transferring learning from one situation to apply to another can be extremely difficult for people with FASD.

### Actions
- Expand current Better Start for Children with Disability Initiative to include FASD.
- Extend funding support to parents and carers organisations that currently exist and expand these into other states and territories ($1.5 million).

**Funding required:** $1.5 million over three years.

**Funding already committed by Government:** $122 million to the Better Start for Children with Disability and $750,000 to NOFASARD.

‘In order for people with FASD to fulfil their potential there needs to be improvements to the education, employment and criminal justice systems in Australia in recognising FASD as an issue and responding appropriately.’

In a traditional school environment, people with FASD struggle to meet these expectations, causing frustration for both the student and teacher. Teaching methods that aim to change the behaviour of a person with FASD are likely to fail, not due to a lack of the individual’s desire to change, but their ability to be able to do so. By
continually failing to meet expectations, people with FASD develop a sense of failure which can lead to depression and other mental health issues.\textsuperscript{3,2}

Being able to obtain and maintain employment is also difficult for people with FASD. A study of adults with FASD conducted in 1996 by the University of Washington found that:

- 50 per cent of respondents had trouble finding employment
- 60 per cent had difficulty maintaining employment
- 18 per cent had achieved independent living but most had employment problems, and
- 80 per cent had difficulty in managing money and decision making.\textsuperscript{77}

From the above it is apparent why people with disrupted school experiences, unstable home environments and difficulty obtaining employment may become involved with the criminal justice system. Additionally people with FASD are often vulnerable to exploitation and often guided into criminal behaviour.\textsuperscript{96} In a prison environment an individual with FASD can be used as a scapegoat, be negatively influenced by their peers, victimised and exposed to more serious criminal elements.\textsuperscript{77} Correctional facilities and the criminal justice system are not generally prepared to identify or address the needs of individuals with FASD within the overall offender population.

Across Canada and the USA the situation is changing. Significant work has been undertaken to train teachers and address employment and criminal justice issues. In the education sector, British Columbia has produced a \textit{Manual of Policies, Procedures and Guidelines for Special Education Services}\textsuperscript{97} and in Alberta similar guidelines exist with strategies on how to teach students with FASD.\textsuperscript{98} In the USA, the National Organization for Fetal Alcohol Syndrome (NOFAS) has created an education curriculum for teachers from kindergarten to grade 12 on FASD. This gives age-appropriate information on alcohol and provides opportunities for teachers to integrate information on FASD into the standard education curriculum.\textsuperscript{99} The Florida Department of Education has also produced a resource guide for educators on FASD.\textsuperscript{100}

A Churchill Fellowship undertaken by Kym Crawford (an education specialist) on the Canadian experience of addressing FASD found that the key issues were that:

- People working in education, including principals, teachers, education assistants and Aboriginal and Torres Strait Islander education officers, need professional development training on how to educate students with FASD.
- Current specialist support services should be expanded to provide assistance for students with FASD. This includes FASD being an indicated group within the West Australian ‘Schools Plus’ framework, and
- State government education departments need to work closely with diagnostic services (when these exist) to develop a support system immediately after diagnosis. This could be similar to the service provided for students with Acquired Brain Injury (ABI), where specialist teachers liaise with those making the diagnosis and make appropriate adjustments to the student’s educational program to accommodate the specific needs of the individual.\textsuperscript{101}

Pilot programs have also taken place in the USA and Canada to help people with FASD achieve success in employment. From 2009 to 2010 the Alberta Government in Canada, ran pilot projects in the towns of Medicine Hat and Cold Lake. The project at Medicine Hat aimed to improve the employability and life skills of people with FASD through employment coaching. The Cold Lake project developed transition plans for young people moving from youth to adult services and produced tools for employment support agencies to work with people with FASD.\textsuperscript{102} These projects and others in the USA have found that key structural components are needed when employing people with FASD. This includes awareness by the employer and other members of staff about FASD and an understanding that an individual’s behaviour and abilities may change on a day-to-day basis.\textsuperscript{103,2}

Also in the USA and Canada there is a growing body of information about appropriate sentencing options for people with FASD. In British Columbia, the John Howard Society of Central and South Okanagan
has developed a Gateway Mentoring Program that provides one-to-one mentorship to people with FASD involved in or at risk of involvement in the Criminal Justice System. The program offers crisis intervention, life skills training, access to further community supports, and promotion of healthy lifestyles. In Manitoba the FASD Youth Justice Program, established in 2004, provides people accused of a crime with an opportunity to receive an assessment for FASD prior to sentencing. This program developed a checklist called S.T.O.P (Systematic Tell-tales of the Problems) which is a series of ‘red-flags’ that court and justice officials use to trigger a referral for diagnosis. These flags include:

- repeated history of ‘failure to comply’
- lacking empathy
- disrupted or poor school experiences
- being unable to connect actions with consequences
- appearing unaffected by past punishments
- committing crimes that are opportunistic rather than planned
- committing offences that involve risky behaviour for little gain or involvement in gang crime, and
- having superficial relationships and friendships.

In Australia, in 2011 with funding from FARE, the Queensland University and Collaboration for Alcohol Related Developmental Disorders (formerly FASD Research Network) undertook a survey of all judges and magistrates in Queensland to ascertain levels of awareness of FASD and the impact of FASD on their practice. In total 49 members of the Queensland judiciary completed the survey. Of these, 80 per cent reported they had ‘heard’ of FASD and 75 per cent thought that FASD was relevant to their work in the legal profession. However 82 per cent had never sent an accused person for a FASD diagnosis or assessment as they “did not know where to send the person.” Additionally 85 per cent wanted more information and guidelines on how to appropriately sentence a person with FASD.

In order for people with FASD to fulfil their potential there needs to be improvements to the education, employment and criminal justice systems in Australia in recognising FASD as an issue and responding appropriately.

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h Collaboration for Alcohol Related Developmental Disorders (formerly FASD Research Network) is made up of the following researchers: Dr Tom Burne (Queensland Brain Institute), Dr Suyinn Chong (Queensland Institute of Medical Research), Associate Professor Gail Garvey (Menzies School of Health Research), Ms Lorian Hayes (National Indigenous Australian Foetal Alcohol Syndrome Education Network), Ms Diane Malbin (Fetal Alcohol Syndrome Consultation, Education and Training Services Inc), Professor David Pow (Royal Melbourne Institute of Technology), Mrs Anne Russell (Russell Family Foundation for Fetal Alcohol Disorders), Dr Stephen Stalnis, Brisbane Youth Detention Centre, Professor Emma Whitelaw (Queensland Institute of Medical Research).

The Network also includes the following researchers from the University of Queensland: Dr Rosa Alati, Ms Amanda Barnett, Dr Tracey Björkman, Professor Paul Colditz, Professor Heather Douglas, Dr Simon Finningan, Associate Professor Glenda Gobe, Professor Wayne Hall, Dr Janet Hammill, Professor Noel Hayman, Professor Wendy Hoy, Professor Murray Mitchell, Associate Professor Karen Moritz, Dr Leith, Moxon-Leister, Dr Peter Nixon, Ms Coraile Ober, Dr Margo Pritchard, Associate Professor Stephen Rose, Dr James Scott and Ms Megan Williams.
To achieve the best educational outcomes DEEWR should work in conjunction with state and territory education departments to develop education standards to support children with FASD. DEEWR should also develop national training guidelines on teaching people with FASD, based on guidelines that exist in Canada and the USA. Functional assessments should be undertaken by schools to shape decisions about educational goals for the individual. It should also be recognised that the education assistance for one person may not work for another.

To help find suitable employment, people with FASD are able to access Disability Employment Services (DES), funded by DEEWR. These services were introduced by the Commonwealth Government in 2010 to provide tailored advice for job seekers with a disability to enable them to secure suitable employment. Regrettably no information is available on the numbers of people with FASD who have been able to use these services or what the outcome has been. More research is needed in Australia to understand the needs of people with FASD in seeking employment and of those who are employed.

For the criminal justice sector the research undertaken by Queensland University recommended that corrective services screen for FASD when preparing pre-sentencing reports using the S.T.O.P checklist. Also NOFAS in the USA recommend that the criminal justice system help people with FASD by:

- educating judges, lawyers and correctional officers on FASD
- establishing screening tools and procedures to identify FASD among those entering the juvenile justice or adult criminal justice system, and
- utilising alternative sentencing options for people with FASD.

Determining the costs of how the education, employment and criminal justice systems can adequately respond to FASD is difficult. This is predominantly due to the current lack of awareness and knowledge of FASD as an issue within these sectors. To ensure the current situation changes the following are required:

- Develop teaching guidelines for educators on how to teach people with FASD.
- Undertake research into the employment needs of people with FASD, and
- Pilot a training program for judges and magistrates on FASD.

The total cost of developing teaching guidelines is estimated at $800,000 over three years, based on development costs of $100,000 per state. Producing the teaching guidelines would require oversight by a project officer to prepare the guidelines and a consultation process on how to incorporate the teaching strategies into the curriculum. This process should be piloted by one State Government, supported by DEEWR before being expanded into other jurisdictions.

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1 People who are eligible to access the DES Employment Support Services are recognised within the Disability Services Act 1986. This includes people with a disability that:
   a) is attributable to an intellectual, psychiatric, sensory or physical impairment or a combination of such impairments;
   b) is permanent or likely to be permanent, and
   c) results in:
      i. a substantially reduced capacity of the person for communication, learning or mobility, and
      ii. the need for ongoing support services.
The total cost of undertaking research into the employment needs of people with FASD is estimated to cost $200,000 over two years. This research would investigate the barriers and challenges that people with FASD face in seeking and maintaining employment. The research would consist of a postal survey of people with FASD, their parents and carers (n=2,000) and four focus groups. The quantitative and qualitative components of the research are estimated to cost $70,000 and the project implementation (led by a full time project officer for 18 months) is estimated at $130,000.109

Research from the Queensland University clearly indicates that knowledge by judges and magistrates on FASD is poor and that they require and are willing to participate in training on FASD. However the best model to deliver this training across Australia is currently unknown. It is therefore proposed that a pilot be undertaken to develop a training model for judges and magistrates on FASD. This pilot would include the development of a training course for judges and magistrates in Queensland (building on the Queensland University project), implementation and evaluation of this training and the development of a training model that could be used throughout Australia. This pilot could also be carried out in consultation with the Australasian Institute of Judicial Administration (AIJA) which is a membership body for judges, magistrates, tribunal members and court administrators in Australia.110

The total cost of the training pilot for judges and magistrates is estimated at $67,000 over one year. This is based on running four training courses, with up to 15 participants per course (total of 60 participants) and includes training materials, workbooks and assessment information, to a total of $8,000.1 The research component of the study is estimated to cost $50,000 which would evaluate the training and develop a training model that could be rolled out across Australia.

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Actions:

- Develop teaching guidelines for educators on how to teach people with FASD ($800,000).
- Undertake research on the employment needs of people with FASD ($200,000).
- Pilot a training program for judges and magistrates on FASD in one state and develop a training model to be rolled out across Australia ($67,000).

Funding required: $1,067,000 over three years.

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1 There are a total of 39 District Court Judges and 85 Magistrates in Queensland, total 124. Therefore running four training courses with 15 people per training would equate to 50 per cent of the District Court Judges and Magistrates in Queensland receiving training on FASD. Training costs provided by RFFADA.
The prevalence of FASD in Australia is largely unknown and believed to be significantly underreported. Australia lacks standardised data on alcohol consumption during pregnancy and on the number of people with FASD. Without this information, Australia will continue to be unclear about the true extent of FASD within the Australian community.

4.1 Routinely record women's alcohol consumption during pregnancy

There are significant gaps in knowledge about levels of alcohol consumption during pregnancy among Australian mothers. At present, the NDSHS provides the best source of information about alcohol consumption during pregnancy Australia-wide. However, the survey only asks questions about the reduction or cessation of alcohol consumption during pregnancy and not about how much alcohol is being consumed or at what point in the pregnancy.

In 2011, following a review of maternity data in Australia by the Australian Institute of Health and Welfare (AIHW), it was recommended that items on maternal alcohol consumption be added to the Perinatal National Minimum Dataset (Perinatal NMDS). The Perinatal NMDS currently consists of 23 mandatory questions with data supplied by state and territory governments. It includes items on the demographics of the mother and the baby and contains two items relating to tobacco use but no questions about alcohol consumption during pregnancy.

The feasibility of adding additional questions to the Perinatal NMDS is currently being examined by the AIHW as part of a scoping study focused on improving the data collection on FASD in Australia. This study commenced in 2010 and the initial indications highlight inherent difficulties in adding new questions to the Perinatal NMDS. This is due to concerns about the privacy of data collected and reticence by staff to ask pregnant women about their alcohol consumption. The study is due to be published at the end of 2012 and has received $212,082 in funding over two years from DoHA.

Some states and territories, as part of their perinatal data sets, do collect information on alcohol consumption during pregnancy, although not all data collected is published. For example:

- In Tasmania, the most recent data (2009) shows that 11 per cent of women consumed alcohol during pregnancy. Maternal alcohol consumption was more prevalent among older women, especially those aged between 35-39 years (16 per cent). Additionally, 9.3 per cent of babies born to mothers who consumed alcohol during pregnancy had lower birth weights, compared to 6.5 per cent for women who did not consume alcohol during pregnancy.

- Northern Territory data from 2006 showed that alcohol was consumed by 10.9 per cent of the women during their pregnancy. The prevalence of alcohol consumption during pregnancy was higher for Indigenous women (14 per cent at the first visit and 8 per cent at 36 weeks’ gestation) than non-Indigenous mothers (8 per cent and 4 per cent).

- In both Queensland and the Australian Capital Territory, women are asked about their alcohol consumption during pregnancy; however, this data has not been published. The Australian Capital Territory added questions to the Midwives Data Collection Form in 2002 and

- In Victoria, data collection on maternal alcohol consumption will commence in 2012 and form part of the Victoria Perinatal Data Collection.
Further information about women’s alcohol consumption during pregnancy has come from ‘Growing up in Australia: The Longitudinal Study on Australian Children’ (LSAC) and the first wave of the ‘Footprints in time: Longitudinal Study on Indigenous Children’ (LSIC).

As part of the LSAC, mothers from two cohorts (known as cohorts B and K) were asked about their alcohol consumption during pregnancy. For those born between 1999 and 2000 (K cohort), 27.6 per cent of mothers reported drinking alcohol at some stage of their pregnancy. While for those born between 2003 and 2004 (B cohort), 37.6 per cent of women reporting consuming alcohol while pregnant. The timing of these questions corresponds with changes to the NHMRC Guidelines on maternal alcohol consumption and suggests that consumption levels were higher when the ‘NHMRC Australian Alcohol Guidelines: Health Risks and Benefits,’ were released in 2001, which allowed for small amounts of alcohol consumption during pregnancy.

The first wave of data from LSIC in 2009 showed that 22 per cent of Aboriginal and Torres Strait Islander women consumed alcohol while pregnant. However, the majority (79 per cent) drank less when pregnant than prior to pregnancy. In order to improve our understanding of alcohol consumption during pregnancy, it is critical that these studies continue to ask questions about maternal alcohol consumption patterns.

It is crucial that the Perinatal NMDS includes mandatory questions about alcohol consumption during pregnancy. In 2011 the National Perinatal Epidemiology and Statistics Unit (NPESU) of AIHW stated that considerable work is needed to develop consistent definitions and standardisations on alcohol consumption. It also recommended that the national maternity data collection should align with existing data capture arrangements as part of the Maternity Information Matrix, which is a repository of data collected from across Australia on maternity care.

The addition of nationally agreed questions on smoking status to the Perinatal NMDS took four years. This commenced in 2006 with a national data development program. Adding these items to the Perinatal NMDS required strong Commonwealth Government leadership. That same leadership is now required to develop consistent and standardised questions on alcohol consumption during pregnancy. These questions should be included as mandatory items in the Perinatal NMDS and reported on nationally.

The development of standardised data for monitoring alcohol consumption during pregnancy should be overseen by the National Perinatal Data Development Committee (NPDDC). This committee reviews and recommends data items for inclusion on the Perinatal NMDS. The NPDDC is an advisory committee to the NPESU, of AIHW. This action does not require additional funding as it is part of the responsibilities of the AIHW National Perinatal Epidemiology and Statistics Unit to design and implement.

**Action:** Introduce standardised questions about alcohol consumption during pregnancy as part of the Perinatal National Minimum Data Set.

**Funding already committed by Government:** As part of the AIHW National Perinatal Epidemiology and Statistics Unit.
4.2 Standardise data collection on FASD

Currently when a child or adult receives a diagnosis of FASD there are no recording or reporting mechanisms for that diagnosis. The AIHW is currently undertaking a scoping study on improving data collection and reporting of FASD in Australia. Unfortunately no information is currently available and the study is due to be published at the end of 2012.112

However, surveillance systems do exist across Australia for other birth defects and congenital anomalies.119 Congenital anomalies are present from birth, are diagnosed either prenatally, at birth, or within the first few years of life. FASD is considered to be a congenital anomaly as the baby is born with the condition and the harm is caused prenatally.

Currently congenital anomalies are reported through the Australian Congenital Anomalies Monitoring System (ACAMS). The ACAMS is used to detect changes in the frequency of birth defects and can help families and carers access support services. Unfortunately a National Minimum Data Set for congenital anomalies does not exist and notification periods of birth defects to ACAMS vary across the country. This ranges from prenatal diagnosis up to 15 years of age in one state.119k,l

The committee responsible for the development of the National Minimum Data Set for Congenital Anomalies is comprised of representatives from the states and territory governments, Commonwealth Government and key medical bodies.11 National Congenital Anomalies Steering Committee is comprised of representatives from state and territory governments, DoHA, AIHW, the National Centre for Classification in Health, the Human Genetics Society of Australasia/Royal Australian College of Physicians joint committee on newborn screening, the Australian Association of Paediatric Surgeons, the Royal College of Physicians and the Australian Paediatric Surveillance Unit

While ACAMS may offer a method to capture notifications of FASD in time, this is dependent on the development of a National Minimum Data Set for Congenital Anomalies.103 Once the Australian FASD diagnostic instrument and clinical guidelines have been completed and published, then data on FASD should be included in ACAMS. There should be clear definitions and reporting mechanisms to be followed by all jurisdictions and national reporting by the AIHW.

Another option is to establish separate birth defects registers on FASD in each state. Similar registers already exist for cerebral palsy across Australia including the Queensland Cerebral Palsy Register (QCPR). The QCPR was established in 2005 with the Queensland Government providing $90,000 in that year.21 Since 2005 detailed data on cerebral palsy has been collected through the register, allowing for a better understanding of the prevalence of the condition over time. A similar register could be progressed for FASD with a pilot study in one jurisdiction to establish a FASD Register. This could then be rolled out across Australia.

The total cost of establishing a FASD Register in one state is estimated at $321,000 over three years. This is based on the funding provided to establish the QCPR with inflation. This estimate of $107,000 per year to be predominantly spent on salaries and management ($98,000). Salaries include one Clinical Epidemiologist and two senior research assistants.75 Some funds would also be needed to promote and develop the register, estimated at $9,000.

**Action:** Pilot the establishment of a FASD diagnosis register in one state.

**Funding required:** $321,000 over three years.

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1 For Tasmania, Queensland and Australian Capital Territory diagnosis can be done prenatally, New South Wales notification period is at one year of age and for Victoria up to 15, Western Australia up to 6 and South Australian notifications can be made up to 5 years of age.
2 The Northern Territory data does not currently align with the format required by ACAMS and this is not reported on.
3 National Congenital Anomalies Steering Committee is comprised of representatives from state and territory governments, DoHA, AIHW, the National Centre for Classification in Health, the Human Genetics Society of Australasia/Royal Australian College of Physicians joint committee on newborn screening, the Australian Association of Paediatric Surgeons, the Royal College of Physicians and the Australian Paediatric Surveillance Unit.
4.3 Monitor FASD prevalence through the Australian Paediatric Surveillance Unit

As outlined throughout this document Australia lacks accurate data on the prevalence of FASD in the community. Once the Australian FASD diagnostic instrument is published and data is being collected and collated nationally, it should be possible to monitor the prevalence of FASD through existing surveillance units.

Two studies are currently taking place that will help provide information about the prevalence of FASD in Australia. These include the *Marulu: the Lililwan Project* in the Fitzroy Valley of Western Australia, and a longitudinal birth cohort study called the ‘*Triple B Study: Bumps, babies and beyond*’. This study is coordinated by National Drug and Alcohol Research Centre (NDARC) and is collecting information on 1,800 to 2,000 Australian families to examine a wide range of factors that affect the health and development of children and families. The project has a key focus on examining the impact of substance use by pregnant women and their partners during the prenatal period of infant development and future family functioning.

The first national surveillance study for FAS in Australia took place between 2001 and 2004 using the Australian Paediatric Surveillance Unit (APSU) to record cases of FAS identified during that time period.94 The APSU provides active surveillance for prospective, national identification and study of children (under 15 years of age) with uncommon conditions of childhood, including rare infectious and vaccine preventable diseases, genetic disorders, child mental health problems, and rare injuries. Report cards are sent each month to 1,330 paediatricians and other child health clinicians. Clinicians then indicate if they have seen a child with any of the conditions listed. Clinicians report details on demographics, diagnosis, treatments and short-term outcomes for their patients back to APSU.22

The APSU national surveillance study found a limited number of cases of FAS, though it was hypothesised that this was due in part to only 19 per cent of paediatricians being able to correctly identify the diagnostic features of FAS. With systematic training of medical and health professionals to raise awareness of FASD (outlined in sections 1.3 and 2.3) it is likely that reporting rates of FASD would increase over time.

Using the APSU, the systematic surveillance of FASD should be able to be achieved. This system would appear to be the most appropriate mechanism to ascertain the prevalence of FASD within the population over time. It is recommended that the APSU national surveillance study be repeated.75 This would allow for comparison of data and to ascertain any improvements in awareness of the condition since that time.

The total cost to repeat a National Surveillance study of FASD is estimated at $60,000 and would run over three years. The study would need to develop a new protocol to cover more conditions within the FASD spectrum, as the previous study only covered FAS. The study would be run by APSU and would cover staff costs in undertaking the study.75

Action: Repeat the National Surveillance study of FASD using the Australian Paediatric Surveillance Unit.

Funding required: $60,000 over three years.
Aboriginal and Torres Strait Islander peoples are disproportionately affected by FASD with the incidence of FAS being between 2.76 and 4.7 per 1,000 births, in contrast to only between 0.6 and 0.68 per 1,000 live births in the general population. The National Indigenous Drug and Alcohol Committee (NIDAC) outline in their 2012 report ‘Addressing fetal alcohol spectrum disorder in Australia’ that most of our knowledge and research of FAS and FASD, to date, has been through studies conducted on Indigenous communities that are already known to have high levels of alcohol consumption.

NIDAC outlines that FASD should not be seen as a problem unique to Indigenous Australians or Indigenous people but that higher prevalence estimates of FAS in Indigenous communities are due to factors such as socioeconomic status, disadvantage, drinking patterns and diet.

A key challenge in adequately managing FASD among Aboriginal and Torres Strait Islander peoples is that approximately 26 per cent of Aboriginal and Torres Strait Islander people live in remote or very remote areas, making diagnosis and management of FASD difficult. Aboriginal and Torres Strait Islander peoples require culturally appropriate diagnostic and treatment services to assist them in preventing new cases of FASD and to provide support to those who are currently affected, their families and their carers.

5.1. Provide support to Aboriginal and Torres Strait Islander peoples to develop community-driven solutions to address alcohol misuse

The life expectancy of Aboriginal and Torres Strait Islander peoples remains 12 years lower for men and ten years lower for women than the non-Indigenous population. Chronic diseases continue to significantly contribute to morbidity and mortality and, unfortunately alcohol and other drug use continues to be both a consequence and a contributor to this gap in health and social equality between Indigenous and non-Indigenous Australians.

Indigenous Australians are more likely to abstain from alcohol (23 per cent) compared to non-Indigenous Australians (17 per cent) but those Aboriginal and Torres Strait Islander people who do drink, drink at higher risk levels for both the short and long term. In certain communities where alcohol use is pervasive, anecdotal reports suggest that as well as health and social harms caused by alcohol, the prevalence of FASD is likely to be high. For example, the Director of a Queensland preschool and kindergarten stated to the House of Representative Standing Committee on Aboriginal and Torres Strait Islander Affairs that around 80 per cent of the children at the school were showing symptoms of FASD.

To begin to redress the inequality in Indigenous life expectancy, COAG, in November 2008 agreed to a National Partnership Agreement of $1.6 billion to close the life expectancy gap between Indigenous and non-Indigenous Australians within a generation. This has become known as the ‘Closing the Gap’ Strategy. The Commonwealth Government also established a Closing the Gap Clearinghouse within the AIHW, which in 2011 released a report titled ‘What works to overcome Indigenous disadvantage.’ This
report reviewed available evidence and found that the key factors for success in Indigenous community-based alcohol and substance-abuse programs were strong leadership, strong community engagement, appropriate infrastructure and use of a paid workforce to ensure long-term sustainability. In addition adequate resourcing and planning of interventions was important. To be effective, research evidence suggests that interventions should:

- have the support and be controlled by local communities
- be designed specifically for the needs of particular communities and subgroups within them
- be culturally sensitive
- have adequate resourcing and support
- be resourced to cater for clients with complex needs
- provide ongoing care
- achieve an appropriate balance between broad-based and substance specific services, and
- be part of a planned, integrated set of interventions.

The research also found that where Indigenous communities lack capacity partnering with non-Indigenous organisations can occur and be successful if there are agreements for the local community to take full control within an agreed timeframe.

A powerful example of this has been the Marulu strategy in the Fitzroy Valley of Western Australia which began in 2008. This Strategy has been recognised by Australia’s Social Justice Commissioner who described Marulu and the Lililwan study in particular as “an example of researchers reciprocating both the spirit and intent of the community by working to address the challenges of FASD in genuine partnership done where research is done with the community and not just about the community.”

The Marulu Strategy contains elements that AIHW has found are necessary for alcohol strategies to be effective. These are a comprehensive approach that:

- addresses the underlying social determinants
- prevents or minimises the uptake of harmful alcohol use
- provides safe acute care for those who are intoxicated
- provides treatment for those who are dependent, and
- supports people who are affected by FASD or those whose harmful alcohol and other drug use has left them disabled or cognitively impaired.

Aboriginal and Torres Strait Islander peoples need culturally specific prevention, intervention and management strategies that are supported and controlled by local communities. Funding for communities to be able to implement alcohol and substance abuse management plans should come from the Commonwealth Government’s initiative called ‘Breaking the Cycle of Alcohol and Drug Abuse in Indigenous Communities Activity’. A total of $20 million over three years (from 2011-12 to 2013-14) has been committed to this initiative which covers four regions in South Australia, Queensland, New South Wales and Western Australia.

Notably the Breaking the Cycle initiative does not cover the Northern Territory which is covered by the Stronger Futures in the Northern Territory Act 2012 Cth which came into effect on 16 July 2012, replacing the previous Northern Territory National Emergency Response Act 2007. This legislation is not without controversy, though it is focused on strengthening the current alcohol management plans, continuing current alcohol restrictions and strengthening penalties for illicit sales of alcohol. The Commonwealth Government has committed $3.4 billion to Stronger Futures. However this is not only for alcohol measures.

The total amount of funding committed by the Commonwealth Government to these initiatives is over $3.4 billion and this funding should be used to support the implementation of community-driven solutions to alcohol misuse across Australia.

**Action:** Continue to support the development of community-driven solutions to alcohol misuse and support community-led alcohol restrictions where appropriate.

**Funding already committed by Government:** $20 million for Breaking the Cycle of Alcohol and Drug Abuse in Indigenous Communities Activity and commitments to Stronger Futures.
5.2. Publish resources on FASD that are culturally appropriate and tailored to different cultural groups within Aboriginal and Torres Strait Islander communities

As part of the ‘Closing the Gap’ Strategy, the Commonwealth Government established an Indigenous Chronic Disease Package and, through this package DoHA has undertaken developmental market research to understand Indigenous peoples’ awareness and knowledge of healthy lifestyles. The market research undertaken in 2010 found that there is a prevailing belief among Aboriginal and Torres Strait Islander people that chronic diseases are an unavoidable and expected part of life. This is often compounded by the common and fatalistic view that negative health behaviours are both inherited and socially learnt by Aboriginal and Torres Strait Islander peoples. The research found that public education campaigns should aim to increase awareness on the links between risk factors and chronic disease by promoting the actions that can be taken and by appealing to known motivators such as the desire to look after one’s health for the sake of one’s family.

The research also showed that people in remote and very remote areas have a number of unique needs related to communications and physical distance. In particular the more isolated an Indigenous community, the greater likelihood that English will be only a second or third language. It was recommended that local campaigns need to make greater use of visual communications and local languages. The research recommended that messages used in public education campaigns:

- use简单 clear language devoid of jargon
- use familiar and/or local Indigenous people
- apply Aboriginal English
- apply a narrative, storytelling approach, and
- use visual themes applying Indigenous imagery and art work.

There are already organisations and programs working to provide education and awareness of FASD in Aboriginal and Torres Strait Islander communities. This includes the National Indigenous Australian Foetal Alcohol Syndrome Education Network (NIAFASEN). NIAFASEN provides education and awareness programs to Aboriginal and Torres Strait Islander communities on alcohol, other drug use and FASD. NIAFASEN undertakes training with parents, community members and health professionals on FASD across Australia and facilitates FASD prevention programs.

Other examples include the ‘The Strong Spirit Strong Future - Promoting Healthy Women and Pregnancies Project’ in Western Australia that was established in July 2010. This project developed a suite of culturally secure Aboriginal FASD initiatives and resources. A forum was also held to gain input and guidance of senior Aboriginal professionals and Aboriginal community members on the project.

In September 2011 the Anyinginyi FASD Project commenced, located in Tennant Creek, Northern Territory. The initial focus of the project has been to identify existing services and programs and develop a library of resources to be used within the community. The project has also developed Pregnancy Pamper Packs to be distributed by health professionals to all pregnant women with information on alcohol. Anyinginyi has worked with local young people to create a hip hop song called “Strong Baby, Strong Life!”
It is important that the knowledge gained through previous campaigns and education projects are distributed across Australia so that campaigns do not start from scratch. This could be done through existing clearinghouses such as the AIHW Closing the Gap Clearinghouse or Australian Indigenous HealthInfoNet Clearinghouse.

DoHA has also funded National Drug Research Institute (NDRI), to undertake research on developing culturally appropriate resources on alcohol use during pregnancy and FASD. This research aims to develop resource templates on FASD and on the harms associated with alcohol consumption during pregnancy. These templates will then be used by Aboriginal and Torres Strait Islander communities to develop their own culturally appropriate and locally relevant resources on FASD.134 NDRI was awarded close to $700,000 over two years to develop these templates and the project is due to be completed by December 2012135.

The research by NDRI should be seen as an initial phase in the development of culturally appropriate and local relevant resources. Once the project is completed it is critical that further funding is committed so that communities can develop and adapt these templates to local circumstances. A small grants funding scheme, to be developed and administered by DoHA, will achieve this.

The total cost of implementing and adapting the resources from the NDRI project into local communities is estimated at $1.5 million over three years. This would be administered through a small grants scheme for communities to develop FASD resources based on the NDRI templates. Capping the small grants at $500,000 per year and $20,000 per grant (spent on production and development costs) could allow 25 organisations each year to develop culturally appropriate FASD resources based on the NDRI templates.135

**Action:** Fund the provision of a small grants scheme for Aboriginal and Torres Strait Islander communities to adapt the resources produced by the NDRI on FASD and alcohol consumption during pregnancy.

**Funding required:** $1.5 million over three years.

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5.3. Develop comprehensive community responses to FASD in remote and isolated Aboriginal and Torres Strait Islander communities

One of the key barriers to improving health care outcomes for Aboriginal and Torres Strait Islander peoples is poor access to primary health care services, due in part to location or transport problems in accessing the service as well as a lack of culturally sensitive services.

Close to 80 per cent of discrete Indigenous communities are located more than 50 kilometres from a hospital and 50 per cent are located more than 25 kilometres from a community health centre.136 Some areas of Australia also have acute shortages of health professionals or accommodation to house locum health professionals.136 These factors need to be taken into consideration in allocating resources for the management of FASD in these communities.

Two potential service models for FASD diagnosis that may be applicable to Aboriginal and Torres Strait Islander peoples have already been outlined in Priority Area 2: ‘Service model three: Creating FASD diagnostic teams to target at-risk communities’ and ‘Service model four: A local remote/regional diagnostic team’ (see pages 25-26).

Key to the success of either model is ensuring that it is culturally appropriate to the specific community where it is being implemented. The application and testing of different service models is now needed. This could be part of separate feasibility studies or as part...
of comprehensive community response, such as the Marulu Strategy, in Fitzroy Valley, Western Australia.

An example of a comprehensive community response to FASD has been the FASD prevention program by the Ord Valley Aboriginal Health Service (OVAHS) in East Kimberley region of Western Australia. Similar to Marulu this program was started in response to the local Aboriginal community’s concerns about the risks of maternal alcohol use.\(^{137}\)

The OVAHS program has five target groups. The first being women attending antenatal appointments. These women are given information on FASD, advice on their alcohol consumption and one-on-one counselling. These assessments are now an accepted part of routine antenatal care by the community.

The second target group is women of child-bearing age who are engaged through local services such as schools, crisis centres and at weekly community stalls and events. These women receive information on contraception, alcohol and FASD. The other target groups include local Aboriginal men and the wider community. Importantly a number of the nursing and medical staff at OVAHS completed the FASD diagnostic training course at Washington University in the USA, allowing them to undertake diagnostic assessments.

Evaluation of the program has shown it to be effective in creating a ‘whole of community’ response to FASD.\(^{137}\) Similar programs could take place in other areas.

The costs associated with funding comprehensive community responses to FASD in remote communities is difficult to estimate. The OVAHS program received $480,300 for one year from the Commonwealth Government. Using this figure, a grant funding round could be established for communities to develop a ‘whole of community response’ to FASD. Similar to the Breaking the Cycle of Alcohol and Drug Abuse in Indigenous Communities Activity grants, participants could be invited to apply, capped at four regions\(^{130}\).

The total cost of providing a grant funding round to establish ‘whole of community responses’ to FASD is estimated at $6 million over three years. This would encompass four communities receiving grants of $1.5 million over three years to establish and embed community responses to FASD. Each community grant would be expected to address the following five groups: pregnant women, women of child bearing age, local men, health professionals and other staff and engagement with the local community, including services and councils to fully embed the program.

**Action**

Establish a grant funding round for four Aboriginal and Torres Strait Islander communities to develop and embed a ‘whole of community’ response to FASD, including diagnosis.

**Funding required:** $6 million over three years.
Beyond the first three years of the Australian FASD Action Plan

Beyond the initial three years of this Australian FASD Action Plan priorities should be determined by evaluating what has been achieved and by using the following criteria:

• Impact: those strategies which demonstrate the best impact in reducing harms; disability; mortality and economic costs. This includes impacts on people with FASD as well as families and communities.

• Improvability: those strategies which can close the gap between current practice and evidence-based practice and through improvements are likely to result in changes in numbers of people affected.

• Inclusiveness: those strategies that have the greatest population reach; this includes age groups, ethnicity and gender and is focused on equality and equity.

It is also important that priorities and activities are reviewed and evaluated to demonstrate if they have been effective. A strategy to evaluate the success of the Australian FASD Action Plan should be developed and timed to commence with the start of the Plan.
Appendices

Appendix A: History of FASD in Australia

Across the world, the relationship between maternal alcohol consumption and negative child outcomes has been recognised for a considerable time, with Dr William Sullivan in 1899 suggesting that alcohol was causally related to negative birth and life outcomes. In 1973 the term ‘Fetal Alcohol Syndrome’ was coined by paediatric dysmorphologists Dr Kenneth Jones and Dr David Smith from the University of Washington. Since that time research and clinical experience has expanded to describe a broader spectrum of prenatal alcohol exposure effects. The USA and Canada have led the world on efforts to prevent new cases of FASD, to understand its effects and to provide support and assistance to those affected.

Australian efforts to recognise and manage FASD issues have lagged behind these countries. When FASD programs have been developed, they have often been ad hoc and inconsistently applied across states and territories. This has meant that new programs have been established with few opportunities for extension or expansion or learning from previous initiatives.

The profile raising of FASD in Australia is due to the passion and concerted efforts of people who have been affected by FASD, as well as key researchers and health professionals. Some of the key milestones achieved as a result of their efforts are included below.

1978 -80s **Four case studies on Fetal Alcohol Syndrome in Australia were published**

These studies substantiated international research findings that heavy alcohol exposure in utero is associated with FAS.

1999 **National Organisation for Fetal Alcohol Syndrome and Related Disorders established**

The National Organisation for Fetal Alcohol Syndrome and Related Disorders (NOFASARD) was established. This was the first organisation in Australia with the aim of raising awareness of FASD and providing assistance to people living with FASD, their families, carers and support workers.

2001 **Publication of Cape York Justice Study (Fitzgerald Report)**

The Queensland Government commissioned Justice Fitzgerald to study the causes, nature and extent of alcohol misuse in the Cape York region and relationship with the law. In the report Justice Fitzgerald found alcohol to be the chief precursor to violence, crime, injury and ill health to the people in the Cape York region and noted the “alarming results...of maternal alcohol consumption on the newborn.” Recommendations were made for programs such as “Strong Mothers, Strong Babies, Strong Culture” to be undertaken to improve early childhood development and pregnancy outcomes.


In 1999 the National Expert Advisory Committee on Alcohol (NEACA) recognised the need for a scientific literature review on FASD during the development of the National Alcohol Strategy 2001-2003/04. The occasional paper was developed through this and a national workshop identified in a list of priorities in the document.
2002 National Workshop on Fetal Alcohol Syndrome
This workshop was convened by the Australian National Council on Drugs and NEACA and brought together researchers, clinicians, service providers and individuals with a particular interest and/or expertise in FASD. It aimed to raise the national profile of FAS among professionals and contribute to policy approaches to address FAS.146

2005 Publication of ‘Alcohol and pregnancy: a mother’s responsible disturbance’
This book, written by Elizabeth Russell uncovers the circumstances that lead to both her children being diagnosed with FASD.147 This book was followed in 2007 with ‘Alcohol and pregnancy: no blame no shame’ and ‘Strategies for Employment Service Specialists’ also by Elizabeth Russell.

2006 Application to Food Standards Australia New Zealand (FSANZ) for warning labels
The Alcohol Advisory Council of New Zealand (ALAC) made a submission to FSANZ to make changes to the existing food codes so that all alcohol products would be required to have health and safety messages warning about the dangers of consuming alcohol while pregnant.148

2006 Intergovernmental Committee on Drugs Working Party on FASD established
The Intergovernmental Committee on Drugs (IGCD) established a Working Party on FASD. The Working Party initiated and supported research into FASD, including examination of the economic impact of FASD, including services and treatment for FASD. A log of FASD-related activities in Australia was compiled and a monograph produced. The monograph was completed in 2009 and released in 2011.

2007 Russell Family Fetal Alcohol Disorders Association (RFFADA) established
The Russell Family Fetal Alcohol Disorders Association (RFFADA) aims to support parents and carers of children and adolescents with FASD. In 2010 RFFADA established a partnership with Training Connections Australia to develop and deliver training on FASD to a variety of audiences around Australia.

2011 National Indigenous Corporation for Fetal Alcohol Syndrome Education Network (NICFASEN) established
Founded by Lorian Hayes, NICFASEN has provided education on FASD to over 40 Aboriginal and Torres Strait Islander communities across Australia.

2011 Inquiry into FASD by the House of Representatives Social Policy and Legal Affairs Standing Committee
In November 2011 the Minister for Families, Housing, Community Services and Indigenous Affairs, The Hon Jenny Macklin MP and the then Minister for Health and Ageing, The Hon Nicola Roxon MP requested that the Committee inquire into and report on the incidence and prevention of FASD in Australia. The committee is due to present its report to Parliament in 2012.

Since 2009 the Commonwealth Government has invested $2.5 million into FASD specific programs and research.149 This includes the development of the FASD Monograph, funding for the initial phases of development of the Australian FASD diagnostic instrument and investment into the first comprehensive assessment of FASD prevalence in an Australian Community, known as Marulu: The Lililwan Project.

State and territory governments have also funded some work on FASD. In 2006 the Drug and Alcohol Service in South Australia produced a guide for midwives on FASD.71 In 2010 the Western Australian Government Child and Youth Health Networks developed a Model of Care for FASD.58
**Appendix B: Acronyms**

<table>
<thead>
<tr>
<th>Acronym</th>
<th>Description</th>
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<tbody>
<tr>
<td>ABI</td>
<td>Acquired Brain Injury</td>
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<tr>
<td>ACAMS</td>
<td>Australian Congenital Anomalies Monitoring System</td>
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<tr>
<td>AHMAC</td>
<td>Australian Health Ministers Advisory Council</td>
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<td>AIHW</td>
<td>Australian Institute of Health and Welfare</td>
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<td>AJWA</td>
<td>Australasian Institute of Judicial Administration</td>
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<td>ALAC</td>
<td>Alcohol Advisory Council (of New Zealand)</td>
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<td>APSU</td>
<td>Australian Paediatric Surveillance Unit</td>
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<tr>
<td>AQUA Project</td>
<td>Asking QUESTions about Alcohol in pregnancy</td>
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<tr>
<td>CDC</td>
<td>Centers for Disease Control and Prevention (USA)</td>
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<td>COAG</td>
<td>Council of Australian Governments</td>
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<td>DEEWR</td>
<td>Australian Government Department of Education, Employment and Workplace Relations</td>
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<td>DES</td>
<td>Disability Employment Services</td>
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<td>DoHA</td>
<td>Australian Government Department of Health and Ageing</td>
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<tr>
<td>FaHCSIA</td>
<td>Australian Government Department of Families, Housing, Community Services and Indigenous Affairs</td>
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<td>FARE</td>
<td>Foundation for Alcohol Research and Education</td>
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<td>FAS</td>
<td>Fetal Alcohol Syndrome</td>
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<td>FASD</td>
<td>Fetal Alcohol Spectrum Disorders</td>
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<td>FASDRN</td>
<td>FASD Research Network</td>
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<td>FSANZ</td>
<td>Food Standards Australia New Zealand</td>
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<tr>
<td>GP</td>
<td>General Practitioner</td>
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<td>HTA</td>
<td>Health Technology Analysts</td>
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<tr>
<td>ICD-9-BPA</td>
<td>British Paediatric Association Classification of Diseases</td>
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<td>ICD-9</td>
<td>International Classification of Diseases, 9th Revision</td>
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<tr>
<td>LSAC</td>
<td>Growing up in Australia: The Longitudinal Study on Australian Children</td>
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<td>LSIC</td>
<td>Footprints in time: Longitudinal Study on Indigenous Children</td>
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<td>NDARC</td>
<td>National Drug and Alcohol Research Centre</td>
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<td>NDIS</td>
<td>National Disability Insurance Scheme</td>
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<td>NDISP</td>
<td>National Disability Insurance Scheme Pathways and Supports</td>
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<td>NDRI</td>
<td>National Drug Research Institute</td>
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<td>NDSHS</td>
<td>National Drug Strategy Household Survey</td>
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<td>NICFASEN</td>
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<td>NHMRC Guidelines</td>
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<td>NOFASARD</td>
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<td>NOFAS</td>
<td>National Organization for Fetal Alcohol Syndrome (USA)</td>
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<td>NPDCC</td>
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<td>NPESU</td>
<td>National Perinatal Epidemiology and Statistics Unit of AIHW</td>
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<td>OVAHS</td>
<td>Ord Valley Aboriginal Health Service</td>
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<td>Perinatal NMDS</td>
<td>Perinatal National Minimum Dataset</td>
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<td>QCPR</td>
<td>Queensland Cerebral Palsy Register</td>
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<td>Royal Australian College of General Practitioners</td>
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<tr>
<td>RFFADA</td>
<td>Russell Family Fetal Alcohol Disorders Association</td>
</tr>
<tr>
<td>S.T.O.P.</td>
<td>Systematic Tell-tales of the Problems, used by the Criminal Justice system in the USA as flags for referral for FASD diagnosis</td>
</tr>
</tbody>
</table>
Appendix C: Detailed breakdown of funding for each Priority Area

Priority Area 1: Increase community awareness of FASD and prevent prenatal exposure to alcohol

1.1 Conduct an ongoing national public education campaign about the harms resulting from alcohol consumption during pregnancy.

Establish and deliver a three year public education campaign, using a range of media, about the harms from alcohol consumption during pregnancy, including specific messages and resources for the general public, women and their partners: $10.2 million over three years.

<table>
<thead>
<tr>
<th>Breakdown of funding required</th>
<th>Year 1</th>
<th>Year 2</th>
<th>Year 3</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Campaign development</td>
<td>$2,000,000</td>
<td></td>
<td></td>
<td>$2,000,000</td>
</tr>
<tr>
<td>Printing, pamphlets and posters</td>
<td>$700,000</td>
<td></td>
<td></td>
<td>$700,000</td>
</tr>
<tr>
<td>Development of television adverts</td>
<td>$800,000</td>
<td></td>
<td></td>
<td>$800,000</td>
</tr>
<tr>
<td>Development of radio adverts</td>
<td>$500,000</td>
<td></td>
<td></td>
<td>$500,000</td>
</tr>
<tr>
<td>Running the campaign</td>
<td>$2,400,000</td>
<td>$2,900,000</td>
<td>$2,900,000</td>
<td>$8,200,000</td>
</tr>
<tr>
<td>Purchase of advertising time</td>
<td>$2,000,000</td>
<td>$2,000,000</td>
<td>$2,000,000</td>
<td>$6,000,000</td>
</tr>
<tr>
<td>PR agency fees</td>
<td>$400,000</td>
<td></td>
<td></td>
<td>$1,200,000</td>
</tr>
<tr>
<td>Update campaign (25% of total production costs)</td>
<td>$500,000</td>
<td>$500,000</td>
<td></td>
<td>$1,000,000</td>
</tr>
<tr>
<td>Total</td>
<td>$4,400,000</td>
<td>$2,900,000</td>
<td>$2,900,000</td>
<td>$10,200,000</td>
</tr>
</tbody>
</table>

1.2 Implement mandatory health warning labels on all alcohol products for sale in Australia.

Implement a mandatory, government regulated alcohol health warning label regime for all alcohol products available for sale in Australia: $682,000 over three years.

<table>
<thead>
<tr>
<th>Breakdown of funding required</th>
<th>Year 1</th>
<th>Year 2</th>
<th>Year 3</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Implementation and enforcement</td>
<td>$264,000</td>
<td></td>
<td></td>
<td>$264,000</td>
</tr>
<tr>
<td>Administration</td>
<td>$42,000</td>
<td>$11,000</td>
<td>$11,000</td>
<td>$64,000</td>
</tr>
<tr>
<td>Auditing</td>
<td>$88,000</td>
<td>$88,000</td>
<td></td>
<td>$176,000</td>
</tr>
<tr>
<td>Dealing with complaints and enquiries</td>
<td>$44,000</td>
<td>$44,000</td>
<td>$88,000</td>
<td></td>
</tr>
<tr>
<td>Research</td>
<td>$45,000</td>
<td>$45,000</td>
<td></td>
<td>$90,000</td>
</tr>
<tr>
<td>Total</td>
<td>$306,000</td>
<td>$188,000</td>
<td>$188,000</td>
<td>$682,000</td>
</tr>
</tbody>
</table>

* All figures taken from Health Technology Analysts report13
1.3 Provide specialist support services to pregnant women who have alcohol-related disorders.

Total funding required: $3.1 million over three years:

- Develop state and territory based models of care for women who have alcohol use disorders with clearly defined referral pathways into treatment ($517,000).
- Provide funding to alcohol and drug treatment services to allow them to develop women-centred practices, with a particular focus on women who are pregnant ($2.1 million).
- Develop and evaluate an online intervention program to support women at risk of alcohol exposed pregnancies ($500,000).

<table>
<thead>
<tr>
<th>Breakdown of funding required</th>
<th>Year 1</th>
<th>Year 2</th>
<th>Year 3</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>National Model of Care</td>
<td>$44,000</td>
<td>$58,000</td>
<td>$44,000</td>
<td>$146,000</td>
</tr>
<tr>
<td>Project Officer drafting (6 months)</td>
<td>$44,000</td>
<td></td>
<td></td>
<td>$44,000</td>
</tr>
<tr>
<td>Project Officer analysis (6 months)</td>
<td></td>
<td>$44,000</td>
<td></td>
<td>$44,000</td>
</tr>
<tr>
<td>Consultation meetings ($6,000) in each state (eight)</td>
<td>$48,000</td>
<td></td>
<td>$48,000</td>
<td></td>
</tr>
<tr>
<td>Promotion and resources</td>
<td>$10,000</td>
<td></td>
<td>$10,000</td>
<td></td>
</tr>
<tr>
<td>Development of state-based implementation plans</td>
<td></td>
<td></td>
<td>$371,000</td>
<td>$371,000</td>
</tr>
<tr>
<td>Project officer drafting (6 months)</td>
<td></td>
<td></td>
<td>$44,000</td>
<td>$44,000</td>
</tr>
<tr>
<td>State consultation meeting</td>
<td></td>
<td>$6,000</td>
<td></td>
<td>$6,000</td>
</tr>
<tr>
<td>Promotion and resources</td>
<td></td>
<td>$3,000</td>
<td></td>
<td>$3,000</td>
</tr>
<tr>
<td>Total per State</td>
<td>$53,000</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total for National Model of Care and state implementation plans</td>
<td>$44,000</td>
<td>$58,000</td>
<td>$415,000</td>
<td>$517,000</td>
</tr>
<tr>
<td>Grants funding for specialist support</td>
<td>$100,000</td>
<td>$1,000,000</td>
<td>$1,000,000</td>
<td>$2,100,000</td>
</tr>
<tr>
<td>Scoping study and small grants funding</td>
<td>$100,000</td>
<td></td>
<td>$100,000</td>
<td></td>
</tr>
<tr>
<td>Project officer</td>
<td>$65,000</td>
<td></td>
<td>$65,000</td>
<td></td>
</tr>
<tr>
<td>Qualitative and quantitative research</td>
<td>$35,000</td>
<td></td>
<td>$35,000</td>
<td></td>
</tr>
<tr>
<td>Small grants funding 10 grants x $100,000 each</td>
<td>$1,000,000</td>
<td>$1,000,000</td>
<td>$2,000,000</td>
<td></td>
</tr>
<tr>
<td>Online intervention program to support women at risk of alcohol exposed pregnancies</td>
<td>$100,000</td>
<td>$300,000</td>
<td>$100,000</td>
<td>$500,000</td>
</tr>
<tr>
<td>Website development</td>
<td>$100,000</td>
<td></td>
<td></td>
<td>$100,000</td>
</tr>
<tr>
<td>Counselling support</td>
<td>$200,000</td>
<td></td>
<td></td>
<td>$200,000</td>
</tr>
<tr>
<td>Project management and promotion</td>
<td>$100,000</td>
<td></td>
<td></td>
<td>$100,000</td>
</tr>
<tr>
<td>Evaluation</td>
<td>$100,000</td>
<td></td>
<td></td>
<td>$100,000</td>
</tr>
<tr>
<td>Total</td>
<td>$244,000</td>
<td>$1,358,000</td>
<td>$1,515,000</td>
<td>$3,117,000</td>
</tr>
</tbody>
</table>

* This figure excludes Western Australia which already has a Model of Care for FASD.
*b Excludes Western Australia
1.4 Educate health professionals on FASD and enable them to routinely ask and advise all women about their alcohol consumption.

Total funding already committed by Government: $6,150,450:

• Publish and distribute the updated Pregnancy Lifescrptsp to GPs, to encourage discussions about alcohol consumption during pregnancy ($5.5 million already committed by Government for the complete Lifescrcripts program).

• Provide training to GPs and other relevant health professional bodies on how best to raise the issue of alcohol consumption with consumers, particularly with pregnant women ($650,450 already committed by Government).
Priority Area 2: Improve diagnostic capacity for FASD in Australia

2.1 Publish, implement and evaluate the Australian FASD diagnostic instrument.

Publish the Australian FASD diagnostic instrument and develop guidelines for its use: $852,000 over three years.

<table>
<thead>
<tr>
<th>Breakdown of funding required*</th>
<th>Year 1</th>
<th>Year 2</th>
<th>Year 3</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diagnostic instrument evaluation</td>
<td>$195,400</td>
<td>$225,400</td>
<td>$141,000</td>
<td>$562,000</td>
</tr>
<tr>
<td>Salary costs</td>
<td>$170,400</td>
<td>$170,600</td>
<td>$111,000</td>
<td>$452,000</td>
</tr>
<tr>
<td>National consultation</td>
<td>$25,000</td>
<td></td>
<td></td>
<td>$25,000</td>
</tr>
<tr>
<td>National trial and evaluation</td>
<td></td>
<td>$55,000</td>
<td>$30,000</td>
<td>$85,000</td>
</tr>
<tr>
<td>Diagnostic training resources</td>
<td></td>
<td></td>
<td>$290,000</td>
<td>$290,000</td>
</tr>
<tr>
<td>Salary costs</td>
<td></td>
<td></td>
<td>$180,000</td>
<td>$180,000</td>
</tr>
<tr>
<td>Resource development</td>
<td></td>
<td></td>
<td>$35,000</td>
<td>$35,000</td>
</tr>
<tr>
<td>Resource production costs</td>
<td></td>
<td></td>
<td>$30,000</td>
<td>$30,000</td>
</tr>
<tr>
<td>Evaluation</td>
<td></td>
<td></td>
<td>$45,000</td>
<td>$45,000</td>
</tr>
<tr>
<td>Total</td>
<td>$195,400</td>
<td>$225,600</td>
<td>$431,000</td>
<td>$852,000</td>
</tr>
</tbody>
</table>

2.2 Establish FASD diagnostic services.

Total funding required: $7.3 million over three years:
- Establish FASD specific diagnostic clinics operating for one day per week, in three locations across Australia ($3.6 million).
- Undertake three research projects to establish and evaluate different FASD diagnostic service models in three locations ($3.7 million).

<table>
<thead>
<tr>
<th>Costs of one FASD specific clinic</th>
<th>Per year</th>
</tr>
</thead>
<tbody>
<tr>
<td>Staffing</td>
<td>$308,000</td>
</tr>
<tr>
<td>Equipment</td>
<td>$36,000</td>
</tr>
<tr>
<td>Educating health professionals</td>
<td>$9,000</td>
</tr>
<tr>
<td>Promotion</td>
<td>$8,000</td>
</tr>
<tr>
<td>Clinic room rental</td>
<td>$30,000</td>
</tr>
<tr>
<td>Management costs</td>
<td>$9,000</td>
</tr>
<tr>
<td>Total clinic operating cost</td>
<td>$400,000</td>
</tr>
<tr>
<td>Three clinics ($400,000 x 3)</td>
<td>$1,200,000</td>
</tr>
<tr>
<td>Three clinics over three years</td>
<td>$3,600,000</td>
</tr>
</tbody>
</table>
### Cost of research into other diagnostic models

<table>
<thead>
<tr>
<th>Cost Description</th>
<th>Year 1</th>
<th>Year 2</th>
<th>Year 3</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Staff costs (three staff)</td>
<td>$467,000</td>
<td>$467,000</td>
<td>$467,000</td>
<td>$1,401,000</td>
</tr>
<tr>
<td>Vehicle for use by team (includes registration, maintenance, repairs)</td>
<td>$70,000</td>
<td>$15,000</td>
<td>$15,000</td>
<td>$100,000</td>
</tr>
<tr>
<td>Administration</td>
<td>$40,000</td>
<td>$40,000</td>
<td>$40,000</td>
<td>$120,000</td>
</tr>
<tr>
<td>Promotion and resources</td>
<td>$5,000</td>
<td>$5,000</td>
<td>$5,000</td>
<td>$15,000</td>
</tr>
<tr>
<td>Research and evaluation</td>
<td>$50,000</td>
<td>$50,000</td>
<td>$50,000</td>
<td>$150,000</td>
</tr>
<tr>
<td>Set up costs</td>
<td>$73,000</td>
<td>$0</td>
<td>$0</td>
<td>$73,000</td>
</tr>
<tr>
<td><strong>Total clinic cost</strong></td>
<td><strong>$705,000</strong></td>
<td><strong>$577,000</strong></td>
<td><strong>$577,000</strong></td>
<td><strong>$1,859,000</strong></td>
</tr>
<tr>
<td><strong>Two research projects</strong></td>
<td><strong>$1,410,000</strong></td>
<td><strong>$1,154,000</strong></td>
<td><strong>$1,154,000</strong></td>
<td><strong>$3,718,000</strong></td>
</tr>
</tbody>
</table>

#### 2.3 Implement training for health professionals on use of the Australian FASD diagnostic instrument.

Total funding required: $950,000 over two years:

- Train health professionals in the use of Australian FASD diagnostic instrument by funding a small grants round for training providers to train health professionals, overseen by consortium of relevant health peak bodies ($650,000).
- Carry-out five practical FASD diagnostic training workshops across Australia, led by international FASD experts ($300,000).

### Breakdown of funding required

<table>
<thead>
<tr>
<th>Breakdown Description</th>
<th>Year 1</th>
<th>Year 2</th>
<th>Year 3</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small grants round</td>
<td>$325,000</td>
<td>$325,000</td>
<td>$650,000</td>
<td></td>
</tr>
<tr>
<td>Consortium establishment and program management</td>
<td>$75,000</td>
<td>$75,000</td>
<td>$150,000</td>
<td></td>
</tr>
<tr>
<td>Training program (500 health professionals x $1000 each over two years)</td>
<td>$250,000</td>
<td>$250,000</td>
<td>$500,000</td>
<td></td>
</tr>
<tr>
<td>Five FASD diagnostic training courses</td>
<td>$300,000</td>
<td>$300,000</td>
<td>$300,000</td>
<td></td>
</tr>
<tr>
<td>International and Domestic flights for experts</td>
<td>$35,000</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Venue and catering costs across the five courses (see below)</td>
<td>$150,000</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Development of course materials, printing, management and administration</td>
<td>$115,000</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>$625,000</strong></td>
<td><strong>$325,000</strong></td>
<td><strong>$950,000</strong></td>
<td></td>
</tr>
</tbody>
</table>

### Cost per training course

<table>
<thead>
<tr>
<th>Cost Description</th>
<th>Per day</th>
<th>Three day training course</th>
<th>Five training courses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Venue costs</td>
<td>$6,500</td>
<td>$19,500</td>
<td>$97,500</td>
</tr>
<tr>
<td>Catering</td>
<td>$1,500</td>
<td>$4,500</td>
<td>$22,500</td>
</tr>
<tr>
<td>Audio visual</td>
<td>$2,000</td>
<td>$6,000</td>
<td>$30,000</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>$10,000</strong></td>
<td><strong>$30,000</strong></td>
<td><strong>$150,000</strong></td>
</tr>
</tbody>
</table>
Priority Area 3: Enable people with FASD to achieve their full potential

3.1 Support people with FASD, their families and carers: Economic modelling required.

3.2 Improve early intervention options for people with FASD, their families and carers.
Extend funding support to parents and carers organisations that currently exist and expand these into other states and territories: $1.5 million over three years.

<table>
<thead>
<tr>
<th>Breakdown of funding required</th>
<th>Year 1</th>
<th>Year 2</th>
<th>Year 3</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extend funding support to parents and carers organisations</td>
<td>$500,000</td>
<td>$500,000</td>
<td>$500,000</td>
<td>$1,500,000</td>
</tr>
<tr>
<td>Total</td>
<td>$500,000</td>
<td>$500,000</td>
<td>$500,000</td>
<td>$1,500,000</td>
</tr>
</tbody>
</table>

3.3 Treat people with FASD in a socially inclusive manner upon entry into education, employment and if in contact with the criminal justice system.

Total funding required: $1,067,000 over three years:
- Develop teaching guidelines for educators on how to teach people with FASD ($800,000).
- Undertake research on the employment needs of people with FASD ($200,000).
- Pilot a training program for judges and magistrates on FASD in one state and develop a training model to be rolled out across Australia ($67,000).

<table>
<thead>
<tr>
<th>Breakdown of funding required</th>
<th>Year 1</th>
<th>Year 2</th>
<th>Year 3</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pilot the development of teaching guidelines in one state</td>
<td>$100,000</td>
<td>$350,000</td>
<td>$350,000</td>
<td>$800,000</td>
</tr>
<tr>
<td>Project officer</td>
<td>$78,000</td>
<td></td>
<td></td>
<td>$78,000</td>
</tr>
<tr>
<td>Consultation and printing</td>
<td>$22,000</td>
<td></td>
<td></td>
<td>$22,000</td>
</tr>
<tr>
<td>Expand development of teaching guidelines into other states</td>
<td></td>
<td>$350,000</td>
<td>$350,000</td>
<td>$700,000</td>
</tr>
<tr>
<td>Research into employment needs</td>
<td>$100,000</td>
<td>$100,000</td>
<td></td>
<td>$200,000</td>
</tr>
<tr>
<td>Quantitative and qualitative research components (over two years)</td>
<td>$35,000</td>
<td>$35,000</td>
<td></td>
<td>$70,000</td>
</tr>
<tr>
<td>Project implementation (full time research officer 18 months)</td>
<td>$65,000</td>
<td>$65,000</td>
<td></td>
<td>$130,000</td>
</tr>
<tr>
<td>Pilot training for judges and magistrates</td>
<td>$67,000</td>
<td></td>
<td></td>
<td>$67,000</td>
</tr>
<tr>
<td>Four training courses (fees $2,000 per course)</td>
<td>$8,000</td>
<td></td>
<td></td>
<td>$8,000</td>
</tr>
<tr>
<td>Travel and accommodation for trainer</td>
<td>$5,000</td>
<td></td>
<td></td>
<td>$5,000</td>
</tr>
<tr>
<td>Venue hire and catering costs</td>
<td>$4,000</td>
<td></td>
<td></td>
<td>$4,000</td>
</tr>
<tr>
<td>Evaluation of training and development of training model to be rolled out</td>
<td>$50,000</td>
<td></td>
<td></td>
<td>$50,000</td>
</tr>
<tr>
<td>Total</td>
<td>$267,000</td>
<td>$450,000</td>
<td>$350,000</td>
<td>$1,067,000</td>
</tr>
</tbody>
</table>
Priority Area 4: Improve data collection to understand the extent of FASD in Australia

4.1 Routinely record women’s alcohol consumption during pregnancy: Funding already committed by Government as part of the AIHW National Perinatal Epidemiology and Statistics Unit.

4.2 Standardise data collection on FASD
Pilot the establishment of a FASD diagnosis register in one state: $321,000 over three years.

<table>
<thead>
<tr>
<th>Breakdown of funding required</th>
<th>Year 1</th>
<th>Year 2</th>
<th>Year 3</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Staffing</td>
<td>$90,000</td>
<td>$90,000</td>
<td>$90,000</td>
<td>$270,000</td>
</tr>
<tr>
<td>Promotion</td>
<td>$9,000</td>
<td>$9,000</td>
<td>$9,000</td>
<td>$27,000</td>
</tr>
<tr>
<td>Management cost</td>
<td>$8,000</td>
<td>$8,000</td>
<td>$8,000</td>
<td>$24,000</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>$107,000</strong></td>
<td><strong>$107,000</strong></td>
<td><strong>$107,000</strong></td>
<td><strong>$321,000</strong></td>
</tr>
</tbody>
</table>

4.3 Monitor FASD prevalence through the Australian Paediatric Surveillance Unit.
Repeat the National Surveillance study of FASD using the Australian Paediatric Surveillance Unit: $60,000 over three years.

<table>
<thead>
<tr>
<th>Breakdown of funding required</th>
<th>Year 1</th>
<th>Year 2</th>
<th>Year 3</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Development of study protocol and staffing costs within APSU</td>
<td>$20,000</td>
<td>$20,000</td>
<td>$20,000</td>
<td>$60,000</td>
</tr>
</tbody>
</table>
Priority Area 5: Close the gap on the higher prevalence of FASD among Aboriginal and Torres Strait Islander peoples

5.1. Provide support to Aboriginal and Torres Strait Islander communities to develop community-driven solutions to address alcohol misuse: Funding already committed by Government.

5.2. Publish resources on FASD that are culturally appropriate and tailored to different cultural groups within Aboriginal and Torres Strait Islander communities.

Fund the provision of a small grants scheme for Aboriginal and Torres Strait Islander communities to adapt the resources produced by the NDRI on FASD and alcohol consumption during pregnancy: $1.5 million over three years.

<table>
<thead>
<tr>
<th>Breakdown of funding required</th>
<th>Year 1</th>
<th>Year 2</th>
<th>Year 3</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small grants funding scheme</td>
<td>$500,000</td>
<td>$500,000</td>
<td>$500,000</td>
<td>$1,500,000</td>
</tr>
<tr>
<td>25 grants provided each year up to $20,000 each</td>
<td>$20,000 x 25 grants</td>
<td>$20,000 x 25 grants</td>
<td>$20,000 x 25 grants</td>
<td>$20,000 x 25 grants</td>
</tr>
</tbody>
</table>

5.3 Develop comprehensive community responses to FASD in remote and isolated Aboriginal and Torres Strait Islander communities.

Establish a grant funding round for four Aboriginal and Torres Strait Islander communities to develop and embed a ‘whole of community’ response to FASD, including diagnosis: $6 million over three years.

<table>
<thead>
<tr>
<th>Breakdown of funding required</th>
<th>Year 1</th>
<th>Year 2</th>
<th>Year 3</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st community</td>
<td>$500,000</td>
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<tr>
<td>2nd community</td>
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<tr>
<td>3rd community</td>
<td>$500,000</td>
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<tr>
<td>4th community</td>
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</tr>
<tr>
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<td>$2,000,000</td>
<td>$2,000,000</td>
<td>$6,000,000</td>
</tr>
</tbody>
</table>
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