June 2012 Issue 7

FETAL ALCOHOL FORUM©

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

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INTRODUCTION


Susan Fleisher

Those of you who work in the area of Fetal Alcohol and related fields will have noticed that recognition of Fetal Alcohol issues has increased around the world. In this issue of the FETAL ALCOHOL FORUM you will find 143 FASD Research abstracts from 27 countries as well as full articles and recommended books.

Many of our subscribers and supporters have been outraged by the potentially dangerous misleading headlines in the media. Sighting recent Danish studies claim that there is no evidence of alcohol related harm in 5 year old children whose mothers drank in pregnancy. We have included the abstracts of the Danish studies, one of the press articles and a scientific response from leading FASD experts, Professors Theresa Grant and Susan Astley.

As always, we are pleased to begin this issue with original articles written for us by FASD experts: Dr Christine Loock and Osman Ipsiroglu in Canada and Simona Pichini in Italy. We can learn a great deal from Dr Loock's accounts of her FASD journey in Canada and her contribution to the foundation of the Canadian FASD Research Network and FASD partnerships.

Simona Pichini, a leader in FASD in Italy and Spain, shares her professional and personal experience as a scientist and as an adoptive mother of a son with FASD.

Dr Osman Ipsiroglu, a pioneer in sleep research, has contributed a summary of his presentations from two Canadian FASD Conferences: Fetal Alcohol Spectrum Disorder --- *The Power of Knowledge: Integrating Research, Policy and Promising Practice Around the World and Adolescents and Adults with FASD: It's a matter of justice.*

You will see from the abstract titles, we have also included a few studies regarding wider issues related to 'Low-Risk Drinking Guidlelines', transgenerational inherited alcohol conditions, postnatal ethanol exposure, surveys/studies that cover polydrug exposure with alcohol.

As we compile each issue of the FETAL ALCOHOL FORUM we always find the unexpected. In this issue we have found an FASD research collaboration between the Republic of Korea, Pakistan and Saudi Arabia.

NOTE: FASD studies worldwide during the past 6 months.

USA	59
Canada	22
Australia	11
South Africa	5
Denmark	9
Finland	4
Italy	4
Sweden	4
China	3
North Korea	3
New Zealand	3
Russia	3
Germany	2
Spain	2
UK	2
Chile	1
France	1
India	1
Israel	1
Japan	1
Netherlands	1
Pakistan	1
Portugal	1
Saudi Arabia	1
South Korea	1
Switzerland	1
Taiwan	1

We conclude: FASD is a global concern.

Please let us have your feedback at info@nofas-uk.org. To be added to our mailing list click here. Please also let us know if you would like to make a contribution to the next issue of the FETAL ALCOHOL FORUM.

Thank you for your contribution and interest.

Susan Fleisher
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79. **AN EXPLORATION OF THE EXPERIENCES AND PERSPECTIVES OF NEW ZEALANDERS WITH FETAL ALCOHOL SPECTRUM DISORDER**
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82. **PROTECTIVE EFFECT OF [6]-GINGEROL ON THE ETHANOL-INDUCED TERATOGENESIS OF CULTURED MOUSE EMBRYOS**
Yon JM, Baek IJ, Lee SR, Kim MR, Hong JT, Yong H, Lee BJ, Yun YW, Nam SY.
Publication – PubMed
2nd February 2012

83. **ETHANOL EXPOSURE INDUCES UPREGULATION OF SPECIFIC MICRORNAS IN ZEBRAFISH EMBRYOS**
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93. **VITAMIN A, FOLATE, AND CHOLINE AS A POSSIBLE PREVENTIVE INTERVENTION TO FETAL ALCOHOL SYNDROME**
Mark S. Ballard, Muxin Sun, Jenny Ko.
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30th January 2012

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January 2012

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Deng TX, Wang ZX, Gao XQ, Shi YY, Ma ZY, Jin HX, Deng JB.
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5. **ECOGENETIC AND EPIGENETIC INFLUENCES IN FASD**
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6. **AVAILABLE INTERVENTIONS AND SERVICES USED BY CANADIAN ADOLESCENTS WITH FASD AND THEIR FAMILIES**
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7. **HOW TO APPROACH SLEEP PROBLEMS IN CHILDREN WITH FASD: THE 1ST CANADIAN FASD & SLEEP CONSENSUS PAPER**
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8. **RESTLESS LEG SYNDROME (RLS) IN CHILDREN AND YOUTH WITH FASD AND PRENATAL SUBSTANCE EXPOSURE (PSE) – A CLINICALLY MISSED DIAGNOSIS AGGRAVATING THE CHALLENGING BEHAVIOUR?**
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I. THE JOURNEY OF "OUR SUSANNAH": AN OLD SONG WITH NEW MEANINGS

Christine Loock, MD

Written in 1846, the ballad of "Oh! Susannah" is one of the best-known North American songs, again popularized in the 1960s and 70s by our baby boomer generation through such artists as James Taylor and the Byrds. And this year Neil Young will repackage "Susannah" for the 1st track of his new album "Americana". The song is a story of both adversity and hope, in a time of economic, political and social upheaval for families in the US, with poverty, racism, immigration and migration setting the stage for civil war.

The Susannah of our story began her journey in the 1960's and 70s, which was also a time of political and social upheaval in North America, and when the impact of our legacies of immigration, colonization, relocation, and racism on population health were just starting to be understood. "Our Susannah" was born shortly before 20th century researchers "re-conceived" her diagnosis: Lemoine of France (1) and Smith and Jones from Seattle, Washington (2,3), were soon to publish independently their findings about the impact of prenatal alcohol exposure on the fetus. It would take us in North America another 20 years to begin to reliably diagnose Fetal Alcohol Syndrome (FAS), and understand that it was a "Spectrum Disorder (FASD)" and that it wasn't rare or limited to children (4,5).

Our Susannah was born in the Canadian North, weighing just over 5 pounds at term. Her mother, a residential school survivor, had struggled with mental health and addiction issues, which ultimately led to all of her children being removed soon after their births, and her premature death before Susannah reached the age of 10.

Given data that suggests that a stable home environment protects against secondary impacts ("disabilities") related to prenatal alcohol exposure (4), we would have hoped that Susannah's early life would have been supported in government foster care. Sadly, Susannah switched households frequently and was both neglected and abused in her first 8 homes. It wasn't until her later adolescent years that she found a stable and safe home.

Although Susanna felt safe and loved in her last foster home, she began to explore and exert her independence, as many youth do, but having struggled at school, she had little to fall back on when she left for the big city at age 18. She was unable to manage money, find employment or suitable housing or independently care for herself. She began to drink regularly and became dependent upon an abusive partner with addiction and mental health concerns.

Susannah was 25 when she was referred with her two children to our hospital in Vancouver, after having successfully navigated an addiction treatment program during her second pregnancy. She had been cared for in a home for "unwed mothers". But she had drunk heavily with her first child for the first 4 months, until she learned she was pregnant. Susannah then wondered about having FAS herself. She asked if we could assess her.

Despite not having a program for adults, making a diagnosis of FAS for her was relatively easy. You see, she had all the physical features of full FAS, and she had records from being in foster care, which also documented her learning and adaptive difficulties, and her mother's alcohol use during pregnancy. The challenge was how to share Susannah's FAS diagnosis with her social worker, who at the time was trying to help her with housing, parenting and life skills. With her permission, and the guidance and wisdom of practice from the expert psychologist in Seattle, Anne

Streissguth, we were able to disclose the diagnosis in a way that brought more social and financial support to Susannah and her children. Basically, we were advised that in order to parent effectively with FAS, affected individuals require the triad of a strong parent or partner ("alpha") who plans the day to day activities, a supportive network (i.e. supportive peers and places), with personally meaningful activities, for inclusion and participation, and a lastly, strong advocate.

Over the next decade, Susannah managed to reconnect with her last foster family by phone - they lived hours away. She also tried to connect with a peer group, but back then, not many people self identified with FAS. Most challenging for Susannah was that her husband struggled with self-medication for his mental health disorder and, at times, would abuse her, placing the kids at risk. In the end, after a healthy third pregnancy, she voluntarily placed her kids in care for private adoption, because with no known living relatives, she felt she had no other options. She was overwhelmed and isolated. Sadly there were few professionals providing services for adults with FAS. And Susannah's overall cognitive scores made her ineligible to receive community services for intellectual disability. She was "too smart".

Since that time, Susannah's journey has had its ups and downs. She left her husband. She became active in a healing program for persons with early life adversity, and her addictions specialist fortunately knew something about FASD, so that subsequent councilors could adapt and modify her sessions to Susannah's needs. Susannah reconnected with her children, now all adults. And she began to find her voice. And that voice brought her back to our clinical and research table this spring.

Susannah had remembered our names from almost two decades earlier, shortly after placing her kids in care. She had volunteered to participate in our first brain imaging studies of adolescents and adults with FAS (6). This study had been one of our first steps toward moving FASD out of the exclusive domain of paediatrics, as we grew up with our patients (literally), learning with and from our patients and their stories of success and challenges.

Our last 20-year journey with FASD in Canada has also had its ups and downs. We have journeyed alongside many "Susannahs" and "I am Sam"s. We have also come to see that the messages about prevention of FASDs should include not only those in poverty who experienced early life adversity and struggled with addiction, but also the well-educated, well-heeled, professionals who drink to keep up with their peers. Binge drinking has become the norm for women in their reproductive years.

Dr. Geoffrey Robinson and his protégées, Dr. Kwadwo Asante, and Drs. Julie and Bob Conry, were the true early pioneers of our Canadian FASD research and prevention programs(7,8). In the mid 1980s, Dr. Robinson also had the insight to broaden our strategic planning by gathering, both clinical and basic science researchers, interdisciplinary clinicians, economists, educators and social scientists all around a planning table, called the British Columbia "FAS Resource Group". Our goals were to learn more about FASD, to educate, conduct research and to advocate. And to be successful, we needed to plan the evaluation simultaneously with the clinical services. At that time [and still today], population research on FASD incidence and prevalence was required. We knew, even then, that governments do not tend to initiate, but they do respond to the evidence.

Our Canadian approach preventing FAS was guided by a concise manuscript written by an astute social scientist, Janet Waterson from the UK in the early 1990s. She outlined the need for clinical, education, research and health promotion strategies, all linked by intersectoral government coordination. Her approach was elegant, yet simple and clear enough to be drawn using a Ven diagram. (Always remember to keep it simple. You often only have enough time for 3 to 4 points in 5 minutes with harried government decision makers.).

The leadership and tenacity of the original BC FASD Resource group who were joined by a strong, articulate, and passionate group of parents, resulted in focused attention and response from local and national governments and, as a result, the development of a National Advisory Committee and federal government framework for action, followed by the publication of National Guidelines for

Diagnosis of FASD (9-13). A Partnership among 7 Canadian Provinces/Territories emerged to share resources and expertise to enhance FASD prevention and to enhance the care and support strategies for those living with the disability. It soon became clear that gaps in evidence existed and that new research data was critical for effective policy and programming decisions related to FASD. In 2005, The Canada Northwest FASD Research Network was established, through support of the Partnership, to advance clinical and applied quantitative and qualitative research to improve understanding of diagnostics, interventions, surveillance and prevention related to FASD. This information was anticipated to lead to improvements in public policy and programming (14).

Since then, the Research Network's structure has evolved due to the recognition that it would be beneficial to take a leadership role to support the coordination of the relevant research within Canada and along policy-relevant thematic areas that are meaningful to those affected by FASD: Diagnostics, Prevention and Intervention. The Research Network has been very successful at leveraging resources, creating research capacity, translating knowledge into practice, programs and policy and it has also made significant contributions to the field of FASD. Non-partner provinces have repeatedly requested to participate in the Research Network and, in late 2010, The Partnership decided to expand the Research Network nationally and to seek charitable status. Presently, the Canada FASD Research Network is undergoing the processes to become a national charitable organization and has obtained not-for-profit status in British Columbia, its pioneering province.

This unique Partnership model has eliminated the challenges of jurisdictional barriers and access to policy-makers and has provided a small amount of infrastructure support to allow researchers to concentrate on designing and carrying out world-class studies. The Canada FASD Research Network also acts as a hub for networking, collaboration and knowledge translation and exchange related to FASD in Canada.

Conclusion:
Last month, Susannah presented her life story to our community research table, asking that we do something for other "motherless mothers" with FASD, so that they are not lost, forgotten or abused while in alternate "care". Susannah asked about getting connected with other individuals with FASD, who were adults, and parents, like her.

This spring, Susannah participated in the 5th [Canadian] National Biennial Conference on Adolescents and Adults with FASD (It's a Matter of Justice), in Vancouver, BC. To see her smile and laugh, in the closing ceremonies, and perform though song with others, was an unimaginable joy for her and for those of us who have had the privilege to learn and grow, and reconnect with what it really means to have a Fetal Alcohol Spectrum Disorder. Susannah reminds us that she is a person, not a diagnosis, and why we as clinicians, researchers and policy-makers MUST get involved to prevent the intergenerational effects of FASD though education, research, clinical service and advocacy.

Susannah is now joining the FASD Grandparent's Group where she can provide and receive support regarding the everyday challenges of parenting the next generation of children, helping to break the cycle of alcohol use in pregnancy and addressing the added challenges of parenting children with FASD. This group is also interested in new research that incorporates the importance of nurture and the environmental "epigenetic" sequelae of FASD [i.e. prenatal alcohol exposure often overlaps with ensuing early life adversity, as represented by another concurrent "FASD: Family Adversity and Stress Disorders"].

The knowledge that the burden of prenatal and environmental adversity still remains in the "backyards" of developed countries such as Canada, the US, Australia and the UK must give us pause. How can there continue to be ongoing disparity, abuse, exploitation and exclusion of women and children, minorities, and persons with disabilities, in counties, which boast about their historical prosperity and current Olympic prowess (15,16)? Persons with under-recognized disabilities such as FASD remain grossly overrepresented in government custodial care, including foster care and the courts (17). They require the collective and intersectoral actions of strong

advocates, including professionals, parents, partners and politicians, who can collectively add to the shared commitment, will, and passion to champion strategies to prevent the root cause of FASD and to provide effective, sustainable interventions and support for affected individuals.

In Canada, both the professionals and families have learned to work together collaboratively, on an equal basis, with mutual respect and understanding of the strengths that each brings to the table. Things can only improve by society's willingness to listen and honuor the stories of persons who themselves have disabilities such as FASD, allowing them to rewrite their futures, and to compose new words for old songs. This next rendition of "Oh! Susannah" can take on old words with new meaning:

"Oh! Susannah. Don't you cry for me."

Knowledge is power. Listen. Learn. Educate. Evaluate. Advocate.

Refrain [Repeat].

References

1. Lemoine P, Harousseau H, Borteyru JP, Menuet JC. Les enfants de parents alcooliques - anomalies observées: à propos de 127 cas. Ouest Med 1968; 21: 476-82.
2. Jones KL, Smith DW, Ulleland CN, Streissguth AP. Pattern of malformation in offspring of chronic alcoholic mothers. Lancet 1973;1(7815):1267-71.
3. Jones KL, Smith DW. Recognition of the fetal alcohol syndrome in early infancy. Lancet 1973;2(7836):999-1001
4. Streissguth A, Barr H, Kogan J, Bookstein F. Primary and secondary disabilities in fetal alcohol syndrome. In: Streissguth AP, Kanter J, editors. The challenge of fetal alcohol syndrome: overcoming secondary disabilities. Seattle: University of Washington Press; 1997. pp 25-39
5. Astley SJ, Bailey D, Talbot C, Clarren SK. Fetal alcohol syndrome (FAS) primary prevention through FAS diagnosis: I. Identification of high-risk birth mothers through the diagnosis of their children. Alcohol Alcohol 2000;35 (5): 499-508.
6. Clark, C, Li, D, Conry, JL, Conry, RF, Loock, C ,Structural and functional brain integrity of fetal alcohol syndrome in nonretarded cases, Pediatrics, 2000, Volume: 105, Issue: 5, Pages: 1096-1099
7. Asante KO, Nelms-Maztke J. Report on the survey of children with chronic handicaps and fetal alcohol syndrome in the Yukon and Northwest British Columbia. Whitehorse: Council for Yukon Indians; 1985.
8. Robinson GC, Conry JL, Conry RF. Clinical profile and prevalence of fetal alcohol syndrome in an isolated community in British Columbia. CMAJ 1987; 137 (3):203-7.
9. Joint statement: prevention of fetal alcohol syndrome (FAS) fetal alcohol effects (FAE) in Canada. Ottawa: Health Canada; 1996.
10. Roberts G., Nanson J. Best practices fetal alcohol syndrome/fetal alcohol effects and the effects of other substance use during pregnancy. Ottawa: Health Canada; 2003.
11. Clarke ME, Tough SC. A national survey regarding knowledge and attitudes of health professionals about fetal alcohol syndrome. Ottawa: Health Canada; 2003
12. Chudley AE, Conry J, Cook JL, Loock C, Rosales T, LeBlanc N. Fetal alcohol spectrum disorder: Canadian guidelines for diagnosis. CMAJ 2005;172(5 Suppl):S1-21.
13. Loock C, Conry J, Cook JL, Chudley AE, Rosales T. Identifying fetal alcohol spectrum disorder in primary care. CMAJ 2005;172(5):628
14. Clarren, SK, Lutke ,J, Sherbuck, M, The Canadian Guidelines and Interdisciplinary Clinical Capacity of Canada to Diagnose Fetal Alcohol Spectrum Disorder, J Popul Ther Clin Pharmacol, 2011; 18 (3):e494-e499
15. Elliott, EJ, Payne, J Morris, A Haan, E, Bower, C, Fetal alcohol syndrome: a prospective national surveillance study, Arch. Dis. Child. September 1, 2008 93:732-737
16. May, PA, Gossage, J P, Kalberg WO, Robinson, LK, Buckley, D, Manning, M, Hoyme, HE, Prevalence and epidemiologic characteristics of FASD from various research methods with an emphasis on recent in-school studies, Developmental Disabilities Research Reviews, 2009;

15(3):176–192
17. Fast DK, Conry J, Loock CA, Identifying fetal alcohol syndrome among youth in the criminal justice system, J Dev Behav Pediatr. 1999 Oct;20(5):370-2.

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II. FETAL ALCOHOL SPECTRUM DISORDER-FASD, MY INVOLVEMENT AS A SCIENTIST AND AS MOTHER

Simona Pichini

My story with FASD started in 2004 in Barcelona at the Hospital del Mar, where I worked at that time as a pharmacotoxicologist.

We had just finished an investigation regarding meconium testing for drug abuse and in 1,209 samples we found a prevalence of prenatal exposure of 2.6% Cocaine, 4.7% heroin and 5.3% cannabis.

It is on record that Hospital del Mar is located between Barceloneta and La Mina, two city areas with a high percentage of poverty, immigrants and local gypsies.

Faced with the results we wondered what the prevalence of prenatal exposure to maternal alcohol would have been in the same population.

Thus we reanalyzed the meconium samples for the presence of fatty acid ethyl esters (FAEEs), the biomarkers of prenatal exposure of alcohol and we found a 45% fetal exposure in this low socio economic status cohort. We thought that this result would serve as an eye opener for Europeans, that gestational alcohol exposure was not only endemic in areas outside Europe, nor was it only in the Mediterranean areas.

We decided to start a broader investigation, also involving my home country of Italy and we started looking for funds for this new study. It was now the end of 2005 and I returned to work in Italy.

I said before that my story with FASD started in 2004, but this is not precisely true. Unknowingly my story with FASD started in 1995, when I adopted a north African newborn male in the San Giovanni Hospital in Rome, Italy. My beautiful boy was a strange baby. No information on his health was given to me except that he had a low birth weight and low serum iron. That was all the health professionals told me. My baby never slept through the night during the first two years of life, but he grew up very well and at three years of age he started to go to kindergarten in Barcelona, Spain where I now worked . The teachers always referred to episodes of angry outbursts for no apparent reason and when he started primary school, the problems increased. His maths and differences in numbers were difficult to understand, the words were written without an end, or without syllables.

His character was very difficult, often angry with no reason. He was an only child and an only grandson, he was pampered and “spoiled” by everyone though he was the centre of my attention and care, it never worked.

When I returned to Italy, it was impossible to find funds for my investigation on prenatal exposure

to maternal alcohol, even though in 2008 I was working with Dr. Luca Morini from Pavia, a young forensic toxicologist in my investigation group. We discovered a new biomarker of prenatal exposure to maternal alcohol: meconium ethylglucuronide.

We developed and validated an analysis method for this new biomarker. We examined meconium samples from two pilot cohorts: one from Barcelona, Spain and one from Reggio Emilia, Italy and we obtained interesting results. Nonetheless, even asking the National Institute of Health and the Ministry of Health, in the Lazio region which includes Rome, no funds could be found for any study regarding prenatal exposure to alcohol.

In 2010 with a group of colleagues from different neonatology wards in Italy, (very good friends as well as colleagues), we decided to go on with our investigation working for free. Dr. Federica Vagnarelli from Santa Maria Nuova Hospital, Reggio Emilia; Dr. Bruno Sacher from Ospedale Snat'Antonio, San Daniele del Friuli; Prof. Paolo Biban from Ospedale Civile Maggiore, Verona, Dr. Massimo Bisceglia from San Giovanni di Dio Hospital Crotone, Prof. Gherardo Rapisardi from Ospedale S.ta Maria Annunziata, Firenze, Dr. Francesco Raimondi from University Hospital "Federico II", Naples and Prof. Luigi Tarani, Policlinico Umberto I, Roma collected meconium samples from babies born in their hospital, Dr. Luca Morini from the Department of Legal medicine of Pavia University and Dr. Emilia Marchei from National Institute of Health, both of them with a fixed term fellowship, analyzed the samples in their free time and Dr. Andrea Pierantozzi, a statistician with no fixed job, performed statistical analysis.

The obtained data was the first objective Italian data, based on the measurement of "old" (FAEEs) and "new" biomarkers (Ethylglucuronide) of prenatal exposure to maternal ethanol.

The overall prevalence of newborns prenatally exposed to maternal ethanol was 7.9% starting from 0% in Verona, 4.0% in San Daniele del Friuli- 4.9% in Naples, 5.0% in Florence, 6.2% in Crotone, up to 10.6% in Reggio Emilia and 29.4% in Rome. Positivity to neonatal biomarkers of exposure was associated with low maternal education level and younger maternal age. Furthermore, the highest percentage of prenatal exposure in the Capital was significantly related to some maternal socio demographic characteristics.

2010 was a really great year for increased awareness of FASD. For the first time a European Conference on FASD was held in Rolduc, the Netherlands and many European investigators could present the results of their studies plus many health and social professionals involved in awareness, formation and information activities could give their contributions.

Diane Black, the chairwoman of the Conference, a scientist personally involved in FASD and a foster mother of three children affected by FASD, decide to create the " European FASD Alliance" to meet the growing need for European professionals and NGOs concerned with FASD to share ideas and work together to raise awareness of FASD problems and address the needs of affected people and families. I was very proud to be included on the Board and for the first time I found people who would listen to me and give me some answers regarding my son.

After many visits to Medical Doctors of all possible specialties and exclusion of attention deficit hyperactivity disorder, dyslexia, major depression, bipolar disorder, finally Prof. Luigi Tarani, a renown dysmorphologist helped me with the diagnosis of FASD. Many signs and behaviours matched with the limited information about his biological parents.

In 2010, at the time of my Italian multicentre study on the "Assessment of prenatal exposure to ethanol by meconium analysis," my son was 15 years old and the discovery of his "disability" rendered him violent, angry, looking for a culprit to blame for his story and who better than the adoptive mum?

In Italy FASD is an unknown disability as we discovered when we sent a questionnaire to many Italian and Spanish paediatricians and neonatalogists. Nor are social services informed and even less so in general health services. When you tell them your problems they have no solutions and

finally, as in my case, you remain alone with your problem and no solutions.

I'm progressing step by step with daily effort. With Prof. Luigi Tarani, I wrote a brief guide for the diagnosis of FASD, which has been distributed to all the Italian Hospital Neonatology wards and in all the local health assistance services (http://www.iss.it/alco/publ/cont.php?id=73&lang=1&tipo=5). With my son, after many unsuccessful efforts to find an interest, a passion to pull him out of isolation, I hopefully found a way with Karate. A karate team is trying to involve him in all their training, competitions, and initiatives.

It's hard to fight everyday to raise awareness of FASD on a scientific and personal level here in Italy.

I do not have advice for the mothers of FASD children and teenagers. I suggest that they look for strength, for patience, compassion and for feelings which can be supported by real friends and family (when they have one!).

Susan Fleisher of NOFAS-UK and also a parent of an FASD daughter, taught me to remember everyday the beautiful things we have, the beautiful people around us supporting our weakness and to remember our blessings.

Bibliography:

1: Joya X, Friguls B, Ortigosa S, Papaseit E, Martínez SE, Manich A, Garcia-Algar O, Pacifici R, Vall O, Pichini S. Determination of maternal-fetal biomarkers ofprenatal exposure to ethanol: A review. J Pharm Biomed Anal. 2012 Jan 16.

2: Pichini S, Marchei E, Vagnarelli F, Tarani L, Raimondi F, Maffucci R, Sacher B, Bisceglia M, Rapisardi G, Elicio MR, Biban P, Zuccaro P, Pacifici R, Pierantozzi A, Morini L. Assessment of prenatal exposure to ethanol by meconium analysis: results of an Italian multicenter study. Alcohol Clin Exp Res. 2012 Mar;36(3):417-24.

3: Morini L, Falcón M, Pichini S, Garcia-Algar O, Danesino P, Groppi A, Luna A. Ethyl-glucuronide and ethyl-sulfate in placental and fetal tissues by liquid chromatography coupled with tandem mass spectrometry. Anal Biochem. 2011 Nov 1;418(1):30-6.

4: Vagnarelli F, Palmi I, García-Algar O, Falcon M, Memo L, Tarani L, Spoletini R, Pacifici R, Mortali C, Pierantozzi A, Pichini S. A survey of Italian and Spanish neonatologists and paediatricians regarding awareness of the diagnosis of FAS and FASD and maternal ethanol use during pregnancy. BMC Pediatr. 2011 Jun6;11:51.

5: Morini L, Groppi A, Marchei E, Vagnarelli F, Garcia Algar O, Zuccaro P, Pichini S. Population Baseline of Meconium Ethyl Glucuronide and Ethyl Sulfate Concentrations in Newborns of Nondrinking Women in 2 Mediterranean Cohorts. Ther Drug Monit. 2010 Mar 23 32:359-363.

6: Morini L, Marchei E, Vagnarelli F, Garcia Algar O, Groppi A, Mastrobattista L, Pichini S. Ethyl glucuronide and ethyl sulfate in meconium and hair-potential biomarkers of intrauterine exposure to ethanol. Forensic Sci Int. 2010 Mar 20;196(1-3):74-7.

7: Pichini S, Morini L, Marchei E, Palmi I, Rotolo MC, Vagnarelli F, Garcia-Algar O, Vall O, Zuccaro P. Ethylglucuronide and ethylsulfate in meconium to assess gestational ethanol exposure: preliminary results in two Mediterranean cohorts. Can J Clin Pharmacol. 2009 Summer;16(2):e370-5. Epub 2009 Jun 24.

8: Pichini S, Garcia-Algar O, Klein J, Koren G. FAEEs in meconium as biomarkers of maternal drinking habit during pregnancy. Birth Defects Res A Clin Mol Teratol. 2009 Mar;85(3):230;

9: Morini L, Marchei E, Pellegrini M, Groppi A, Stramesi C, Vagnarelli F, Garcia-Algar O, Pacifici R,

Pichini S. Liquid chromatography with tandem mass spectrometric detection for the measurement of ethyl glucuronide and ethyl sulfate in meconium: new biomarkers of gestational ethanol exposure? Ther Drug Monit. 2008 Dec;30(6):725-32.

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III. DE-MEDICALIZING SLEEP: SLEEP ASSESSMENT TOOLS IN THE COMMUNITY SETTING FOR CLIENTS [PATIENTS] WITH FASD & PRENATAL SUBSTANCE EXPOSURE

Summary of the presentations at the 4th International Conference on Fetal Alcohol Spectrum Disorder--The Power of Knowledge: Integrating Research, Policy, and Promising Practice Around the World and 5th National Biennial Conference on Adolescents and Adults with FASD: It's a Matter of Justice. For more detailed information please look at www.chroniccare4sleep.org

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Introduction

The Paediatric Sleep Presentations at the 4th International Conference on Fetal Alcohol Spectrum Disorder--The Power of Knowledge: Integrating Research, Policy, and Promising Practice Around the World and 5th National Biennial Conference on Adolescents and Adults with FASD: It's a Matter of Justice were a source of information for the different kinds of Sleep Problems (SP) and Sleep-related-Problems (SrP) often experienced by many children and adolescents in general, and children and adolescents with fetal alcohol spectrum disorder [FASD] and/or prenatal substance exposure [PSE] in particular [Wengel et al. 2011; Ipsiroglu et al. 2010, 2011a]. At these conferences we had the privilege of sharing our understanding and clinical approach to screening, diagnosis and treatment of SP and SrP in children, youth and young adults with FASD/PSE. Our motto was "individuals are most welcome, but teams prioritized" as conventional sleep assessment concepts have limitations and complex clinical presentations need observations of all involved team members. Thus information exchange with all involved health care professionals is key and remains key in our proposal.

Why? SP & SrP are highly prevalent among children, youth and young adults with FASD/PAE and can lead to a range of other health problems [Stade et al. 2008]. Still, there are many system and knowledge related gaps that block the recognition and treatment of SP & SrP. One frequent reason for delayed sleep onset is Willis-Ekbom Disease, a neurological disorder characterized by an irresistible urge to move one's body to stop uncomfortable or odd sensations, most commonly affecting legs, but also the arms and torso (previously Restless Legs Syndrome) [Allen et al. 2003; Picchietti & Picchietti 2011]. Moving the affected body parts modulates and provides temporary relief from the sensations. This moving/fidgeting behaviour is often interpreted in children, youth and young adults with Willis-Ekbom Disease to be Attention Deficit Hyperactivity Disorder (ADHD) like behaviour, and is often inappropriately medicated as such [Ipsiroglu et al. 2011a]. Additionally, our qualitative and quantitative research in adolescents with an FASD has shown that clinical symptoms and behaviour are often not recognized as sleep related, and caregivers' reports about sleep problems are not given appropriate attention by health care professionals. Children, adolescents and young adults with an FASD are often medicated with melatonin, an over the counter hormone supplement influencing circadian rhythm (Buscemi & Witmans, 2006; Jan et al. 2008; Jan et al. 2010; Jan et al. 2012), others are prescribed psychotropic substances (e.g. selective serotonin re-uptake inhibitors (SSRIs), or antipsychotics) without a critical evaluation of the benefit to potential harm ratio [Ipsiroglu et al. 2011b; Ipsiroglu et al. submitted].

Based on these exciting clinical observations, we started to work on bridging these gaps through a novel bi-directional Communication Strategy that enables team members to structure their observations and follow-up on the interventions (Ipsiroglu et al, 2011c). We also acknowledge the family's need for continuous support throughout the process, especially if some interventions may need additional effort to implement and/or may be initially unsuccessful. In other words, in order to optimize our clinical understanding we need not only consistency, but also the support and observations of involved team members to maintain consistency. We are working to establish this continuity of care concept through knowledge dissemination and collaboration with caregivers, primary care physicians, parent support groups and research partnerships.

In March 2011, the CANADIAN FASD & SLEEP CONSENSUS GROUP reviewed the existing published clinical research exploring the problems and trends of paediatric sleep medicine in regards to screening, diagnosis and treatment of SP and SrP in children, youth and young adults with FASD/PSE, including trends of medication use [Ipsiroglu et al. submitted]. Based on the Canadian health care system's universal services philosophy, the Consensus Group has proposed a 3-Level-Curriculum and is working on standard referral algorithms. The justification is simply that therapeutic measures for SP and SrP are most successful when patients, family members/caregivers, and care teams are all on the same page. Therefore since 2011 all lectures and activities of our working group were designed to help parents and health care professionals embark on the important process of improving wellbeing or quality of life by improving the quality of their child's sleep. Some of our activities are presented on our website www.chroniccare4sleep.org

3-Level-Curriculum

- **Level I** [= Universal Knowledge] *includes* screening with a focus on behaviour, day- and night-time situations and a simple measure for quality-of-life. All health care professionals (HCPs) should be empowered to learn more about SP and SrP to better service patients/clients in health care at the level of care they can offer and help to bridge institutional gaps.
- **Level II** [= Specialized Knowledge] *includes* assessments by HCPs (e.g. occupational/behavioural therapist or community paediatrician) using sleep-logs/-diaries as clinical monitoring/evaluation tools and validated sleep questionnaires, thus requires formal training.
- **Level III** [= Highly Specialized Knowledge] *represents* regional health care services and is the highest level of the curriculum in regards to structured knowledge dissemination.

1st Level: Universal Knowledge or What You Should Know Without Formal Sleep Medicine Training

The Level I concept is based on the Canadian health care system's universal services philosophy and demonstrates the fundamentals of a structured screening and exploration process for SP and SrP. Level I includes an algorithm, composed of two key domains:

a) A **screening questionnaire (discussed below) which focuses on behaviour (including routines) and day- and night-time situations**; and
b) A **measurement tool for wellbeing** (or quality of life).

In addition to these two domains, public health-health based communication strategies are presented on our website. These strategies will include educational material written in language appropriate for families/care providers to apply in their setting.

The screening questionnaire is based on the BEARS concept, a tool for screening the most common SP and SrP in toddlers, preschoolers and school aged children. BEARS does not aim to diagnose, but instead structures an exploration for HCPs and facilitates self-reflection for concerned families and helps them to develop and set goals. Owens and Dalzell adapted the BEARS questionnaire as a teaching algorithm for paediatricians in training and it has been applied in multiple settings. It includes five basic domains, as indicated by the acronym BEARS [Owens & Dalzell, 2005]:

- ***B****edtime Problems*, which explores the going to bed situation;
- ***E****xcessive Daytime Behaviour* which explores the daytime situation (including sleepiness [original version of BEARS] or hyperactive like behaviour, typically exhibited by younger children);
- ***A****wakenings during the night*, which explores the night-time situation;
- ***R****egularity* of sleep/wake cycles which explores the family's routines and lifestyle, as well as
- ***S****noring* and other symptoms of breathing difficulties during sleep. The last domain was changed in favour of ***S****leep Disordered Breathing* since it captures the entire spectrum (with snoring at the low and obstructive sleep apnoea at the higher end of the spectrum).

The question about the **stress** the SP and SrP cause [Sloper & Beresfold 2007], is framed in terms of **wellbeing**, and is asked positively: Questions about the wellbeing of both the child and the main caregiver are measured with a simple wellbeing or Quality of Life (QoL) measurement scale from 0-10 (0 – 100), with 10 (100) being the best QoL one can imagine ('almost in paradise') and 0 being the poorest QoL ('feeling so 'un-alive' that it is like being 'before born or dead'). Asking about wellbeing (QoL) is important in order to:

a) **recognize the urgency** of a SP & SrP related intervention; and

b) **develop a mutual understanding which overcomes systemic communication barriers** and helps HCPs know when to take responsibility and make sure to follow up on the patient and their sleep problems. It becomes even more evident that QoL is significant as a gauge when the caregiver is asked to what degree they think the QoL of both themselves and the child would change if the sleep problems were resolved.

This modified BEARS algorithm, called 'Vancouver-Polar-BEARS' is accessible online and can be used by parents as well as HCPs as a framework for approaching the child's sleep problems in a more structured way [www.chroniccare4sleep.org].

Table 1: The semi-structured interview questions for assessing Sleep Problems and/or Sleep-related-Problems (adapted from Owens & Dalzell, 2005)

Bedtime

Any problems falling asleep or refusing to go to bed? How often/how frequent?

Excessive Daytime Behaviour

Naps or falling asleep during boring activities (e.g., car rides)? "Hyperactivity"? How often/how frequent?

Awakenings During the Night

Waking up frequently during the night? Any complaints/signs of pain? Any sleepwalking, shouting out in sleep, nightmares/terrors, teeth grinding? How often/how frequent?

Regularity and Sleep Duration

Getting enough sleep over the night? How many hours? Differences between weekdays/weekends/holidays? How often/how frequent?

Sleep-Disordered Breathing

Snoring or witnessed breathing gaps; mouth breathing, dry mouth or sore throat, sweating during the night, restless sleep, problems getting up/grumpy in the morning/headache upon waking. How often/how frequent?

Wellbeing Scale (1-10 or 1-100) for the child & caregivers: ***Would you expect any change* in your child's / your wellbeing if sleep problems could be solved?**

Your child's wellbeing

(low) 1 2 3 4 5 6 7 8 9 10 (high)

Change in your wellbeing

(low) 1 2 3 4 5 6 7 8 9 10 (high)

Your wellbeing

(low) 1 2 3 4 5 6 7 8 9 10 (high)

Change in your wellbeing

(low) 1 2 3 4 5 6 7 8 9 10 (high)

Key clinical questions to help:

- Is the sleep of your child ***restorative*** or not?

  IF NOT, can the *non-restorative sleep* be improved with **Sleep Health** measures [e.g. changing lifestyle]?
- Are ***sleep associated situations stressful*** [e.g., going to bed, routines etc.]?

  IF YES, can they be improved with focus on ***Sleep Health*** measures [e.g. changing routines]?
- Imagine ***best*** and ***worst case scenarios***.

This 'exercise' will help to reflect the dimension of the problem spectrum and enable you to be

more specific.

In many cases, screening at Level I and helping parents to focus on Sleep Health measures can be effective in resolving many challenges with collaborative input from structured resources [e.g. developed by our group or others, see Appendix 1] without further *medicalizing* the problem. However, if Sleep Health measures are not effective in treating the child's (family's) sleep problems within a predictable time line, a referral to a health care professional with some formal training is required. Here the use of sleep-logs/-diaries [see Appendix 2] as a first monitoring/evaluation tool will speed up the understanding and further necessary measures.

See Appendix 1: Sleep Health Habits [adapted from the publications Jan et al. 2008 and 2010]
See Appendix 2 : Simple Sleep Overview Log designed for monitoring sleep/wake behaviours and associated mood changes [adapted from Mindell JA, Owens JA, 2009]

2nd Level: Specialized Knowledge or What You Should Know After Some Training

Level II is suggested if Level I is not effective in treating the child's (and in conjunction, family's) sleep problems within a predictable time line. Both *the timeline* and *the need for action* can be defined for the individual patient depending on how much the caregiver feels wellbeing or QoL would be changed if the sleep problem were solved.

Children, youth and young adults with neurodevelopmental disorders/disabilities and multiple health problems, such as those with FASD/PSE or other neurodevelopmental disorders/disabilities, such as Autism Spectrum Disorder (ASD), may often need to be moved to level II quickly. Their SP & SrP are often complex and may require a more in-depth and multidisciplinary approach.

The referral to Level II will usually be made by a general practitioner, though sometimes the referral will be initiated by community-based HCPs. Based on our clinical experience and discussions in the consensus task force groups, we suggest that the report generated at Level II will be addressed not only to the *general practitioner* but also to *all involved key members of the community-based health care team* in order to overcome systemic communication barriers (the consent for distribution of the report should be generated under these premises).

Level II includes a more in-depth assessment by an experienced HCP (e.g. community paediatrician, occupational or behavioural therapist) with a special interest in sleep and some formal training, as would be defined by the specific professional and academic institutions that are in collaboration with institutions in the field of paediatric sleep (e.g. Canadian Sleep Society) [Ipsiroglu et al. 2009].

At the current stage for Level II, we suggest agreement on a 'common denominator concept': all HCPs with various backgrounds should be familiar with the standardized use of:

a) sleep logs and diaries as clinical monitoring tools in order to evaluate interventions; and
b) the validated "Children's Sleep Habits Questionnaire" (CSHQ, Owens 2000); and
c) "Pediatric Sleep Questionnaire" (PSQ, Chervin 2000), which will help to define a comprehensive sleep history.

Presently opinions differ on how these tools, such as the CSHQ and PSQ, should be used. One option is for caregivers to fill out the questionnaire before the appointment, while another option is to conduct semi-structured qualitative interviews in an individualized setting, which can empower parents to bring up their own notions and concerns. The understanding gained from qualitative studies is in favour of the latter, but manpower related restrictions also need to be considered.

An ideal situation is to use both methods: using validated questionnaires as a 'starting point' to be reviewed and discussed during a semi-structured interview/conversation. Another way would be to use the questionnaires for semi-structuring the interview. Our experience is that the automated use of questionnaires leads to a categorical diagnosis and reduces the ability for exploration of

functional presentations, e.g. ‘challenging sleep/wake behaviours’.

Explorations of challenging day- and night-time behaviours in the clinical setting (over the day, in an office) may give limited insight. Video recordings of ‘stressful’ situations in the ‘natural setting’ (e.g. at home or at school) can add significant value to the clinical assessment.

With the comprehensive assessment, the HCP at Level II will take on the role of a clinical navigator in a complex chronic care system.

Thus we also suggest clinical attention to *family ecology* (Lucyshyn & Albin 1993; Lucyshyn et al. 1997). Family ecology explores, amongst other factors, the strength of the child and the family versus the stress that the child’s condition places on the entire family, as well as the available resources to restore the individual caregiver and/or the family’s capacity to cope with the situation. The goal is not to overwhelm the family with additional therapeutic recommendations, but to explore and acknowledge the situation; and determine how to optimize the caregiver and/or the family’s capacity.

The family ecology assessment is not a checklist, but a framework for a semi-structured qualitative interview with exposed and vulnerable clients. This helps to develop a therapeutically sustainable relationship and assesses the family’s situation, including readiness or desperation, and helps the HCP to design the next steps. Due to the therapeutic relationship based on exploration and listening, trust is built. This approach could prevent the involvement of child protection services in cases where the caregiver is reluctant to fully disclose the stress and frustration of caring for a child with a complex condition (Stockler et al, 2012). A respectful exploration of the child’s history and social and cultural context can add significant value in understanding the particularities of the child’s sleep/health/mental health problems and in working with parent(s)/caregiver(s) to find individually tailored solutions.

Table 2: The semi-structured interview questions for assessing Family Ecology (adapted from Lucyshyn and Albin 1993; Lucyshyn et al 1997) and individualizing therapeutic measures.

What would you characterize as strengths of your family?

What are your sources of stress?

- What is the effect of your child’s problem behaviors on you as a parent?
- What is the effect of your child’s problem behaviors on the family as a whole?
- What are other sources of stress in the family?

What formal or informal resources have you used to help improve the situation, e.g.

- respite care
- participation in a parent support group or
- help with childcare and household chores by other family members?

What are your sources of social support, e.g.

- someone with whom you discuss problems and find solutions,
- someone with whom you do leisure activities,
- someone who validates your worth as a person?

What are your goals for your child and family?

The use of objective measures to assess sleep was discussed in the consensus meetings and task force groups. There is an agreement to place the use of diagnostic tools such as pulse-oximetry screening, unattended sleep studies, actigraphy and home-based-overnight-video-sleep-studies to Level III. However, there was also agreement that within Level II the structured use of home-based-overnight-video-sleep-studies and/or videos taken and provided by parents may have utility for increasing clinical understanding and optimizing available history.

3rd Level

Analogous to Service Level III in the health care system, Level III of our *III-Level-Sleep-Curriculum* represents regional health care services and is the highest level of the curriculum in regards to structured knowledge dissemination, and requires an extensive integrated infrastructure and trans-disciplinary thinking.

Level III includes the involvement of a fully trained sleep specialist who is able not only to *screen*, but also to *assess* and *diagnose* sleep disorders with the use of available screening and diagnosis tools, such as [Wise et al. 2011; Witmans & Young, 2011]:

- pulse-oximetry screening,
- unattended sleep studies,
- actigraphy,
- home-based-overnight-video-sleep-studies,
- Electroencephalogram (EEG),
- Polysomnography (PSG), and/or
- a Multiple Sleep Latency Test (MSLT).

In Canada, this level is usually located in the quaternary care setting. However, it has to be stated that even in university hospitals the training of the professionals working at this level has not been coordinated and is discipline/sub-speciality viewpoint-driven. As resources are limited, the necessary discussion has possibly been avoided, creating a tremendous health care management problem.

There is an agreement among sleep medicine focused health care professionals that structuring Levels I and II will also help to implement community based quality control not only for these two levels, but also for Level III medicine, which will help to guarantee continuity of care.

Furthermore, given the Canadian health care system's pyramidal structure, the most common sleep problems can be solved at Level I and II, with each HCP involved in screening and assessing sleep problems acting as the gate keeper for the next level. This approach avoids unnecessary and upsetting interventions for the child as well as unnecessary additional costs to the health care system [Miller et al. 2004, 2009].

Further tasks and definitions of a Level III curriculum also need to be determined at this point, as they may vary between different health care authorities and are certainly influenced by regional and geographic differences. Workshops such as these support addressing these quality and continuity of care issues by opening the discussion for public/health professionals' review. Bi-directional Knowledge Dissemination activities will embed the understanding of a community based quality control of medical services in chronic care management.

Applying the suggested *Best Practice Approach*

A major reason why SP and/or SrP are so often unrecognized and ineffectively treated is because screening and self-monitoring tools were lacking. The screening and self-monitoring tools suggested for use at Level I structure the discussion about when and how SP and/or SRP should be approached. Furthermore, the tools presented at Level II enable HCP with some training to go further and assess (possibly diagnose) SP and/or SrP at the community level. We strongly believe that before starting medication for ADHD-like behaviour and/or psychotropic medication for challenging behaviour a sleep assessment should be done.

On the list below, check off which habits you already have and which ones you would like to have. Start with easy going ones. Check also which habits you consider to be a challenge, thus you don't want to focus on and mark them, too.

Recommendations for Sleep Health Habits for Children with Neurodevelopmental Disabilities such as FASD/Prenatal Substance Exposure

Sleep Health Habits	☺ Yes, we already do this!	☺☺ Yes, we think this would help!	😐 Not right now… This won't work!
Your child maintains a regular wake-up and bedtime every day, including weekends (maximum deviation of 1 hour). Regularity (of both sleep times **and** meal times) encourages your child's body cycles to be coordinated. Maintaining a regular wake-up time is most important for our sleep-wake cycle.	☐	☐	☐
Your child is exposed to sunshine [bright light] during the day. Exposing children to sunshine during the day, after their wake time, particularly in the morning, may help them sleep better at night. Bright light helps the body to produce melatonin (a natural sleep hormone) that promotes better sleep and mood.	☐	☐	☐
Your child's day has a balance of activity and rest. The planning of daily activities is important. Children benefit from structure and routine as well as balanced patterns of both activity and rest during the day and night. Recognizing the relationship between daytime activity and sleep promotion is important.	☐	☐	☐
Your child only takes short naps in the early afternoon. Daytime naps should be geared around the child's age and development. We recommend that a nap should not be taken after 3:00 pm.	☐	☐	☐
As a general rule, your child does only quiet activities in the last hour prior to going to bed. Calming activities include well-structured routine behaviours, such as quiet baths, listening to stories and/or lullabies. Vigorous activities may stimulate your child and afterwards it may take them several hours to relax. Having a bath before bedtime with a bright light, shallow water, and many toys can make the bath exciting rather than calming. Story-telling could have a calming influence, but unfamiliar stories or books with loud sounds (e.g. animal noises) may be stimulating. A bath with deeper water, dim lights and soft familiar songs, may calm.	☐	☐	☐
Your child eats / drinks only light healthy foods / beverages before going to bed. Your child does not eat food during the night. Overeating before bedtime can interfere with your child's sleep. Light healthy snacks such as cheese and crackers (protein and carbohydrates, like a diabetic snack) or oatmeal are recommended. Avoid food and drinks with caffeine (e.g. hot chocolate, chocolate cookies, energy drinks, cola) 4 to 6 hours before falling asleep. Allowing regular night mealtimes quickly teaches the body to wake up during the night because it needs to "be fed". If required your child may drink water.	☐	☐	☐
Your child's TV, DVD, computer game (screen) time is limited. Screen time creates an excess of stimuli and should be avoided at night. However, a favourite and familiar DVD following dinner may be calming for some, but it could result in over-stimulation for others.	☐	☐	☐

Sleep Health Habits (cont.)	☺ Yes, we already do this!	☺☺ Yes, we think this would help!	😐 Not right now… This won't work!
You have a regular bedtime routine with your child; including story time (see also social stories). A series of regular activities, carried out in the same sequence (for example, changing into pyjamas, brushing teeth, going to the toilette, and turning off the lights) allows the body to prepare for going to sleep. Your bedtime routines should not be longer than 30 minutes. Encourage your child to complete part of the bedtime routine independently (e.g. let your child turn off the lights), this strengthens his/her sense of control and independence.	☐	☐	☐
You recognize your child's cue for tiredness. When children become tired they will exhibit some of their bedtime routine activities (e.g. rubbing eyes, taking off socks). When you recognize your child is getting tired help them to get fully ready for sleep. Even slightly dozing off in the late afternoon or at night time can affect quality of sleep during the night.	☐	☐	☐
You put your child to bed while drowsy but still awake in the same place where they sleep all night. Take your child to bed when he/she is awake and then leave the room before they fall asleep. Otherwise your child will constantly associate falling asleep with your presence.	☐	☐	☐
Your child is in bed by 7:00/8:00/9:00 pm depending on his/her age and needs. Children need different amounts of sleep, depending on their ages and individual needs. We recommend that children between the ages of 5-10 go to bed no later than 9:00.	☐	☐	☐
Your child's bed is ONLY used for sleeping. You NEVER send your child to bed as punishment. This causes the bed to become associated with thoughts and activities (e.g. homework and school) that prevent sleep. In turn, your child will come to associate the bed, and the act of going to bed, with punishment.	☐	☐	☐
You let your child turn off the lights Let your child turn off the lights by him/herself. This strengthens their sense of control and independence.	☐	☐	☐
You do not turn on a bright light if you console your child at night or if your child gets out of bed. A bright light tends to cause your child's body to wake up and influences his/her inner clock. Additionally, your child may learn to associate light with comfort and consolation, and darkness with solitude and distress. The bedroom should be totally dark; however, some anxious children with fears of the dark might benefit from a dim night light. Try not to turn on any lights in your child's bedroom at night and try to eliminate all noise sources. *Bright light* = time to wake up *Dark* = time to sleep	☐	☐	☐
Do not smoke in your home because smoking disrupts your child's sleep.	☐	☐	☐

Adapted from Jan JE, Asante KO, Conry JL, Fast DK, Bax MCO, Ipsiroglu OS, Bradbury E, Loock CA, Wasdell MB (2010) Sleep Health Issues for Children with FASD: Clinical Considerations? International Journal of Pediatrics, Article ID 639048, 7 pages, doi:10.1155/2010/639048] and Jan JE, Owens J, Weiss M, Johnson K, Wasdell MB, Freeman R, Ipsiroglu OS. Sleep Hygiene for Children with Neurodevelopmental Disabilities (2008). Pediatrics 122/6 pp. 1343-1350 (doi:10.1542/peds.2007-3308)

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RESEARCH ABSTRACTS

Wiley Online Library – BJOG: An International Journal of Obstetrics and Gynaecology
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1. THE EFFECT OF DIFFERENT ALCOHOL DRINKING PATTERNS IN EARLY TO MID PREGNANCY ON THE CHILD'S INTELLIGENCE, ATTENTION, AND EXECUTIVE FUNCTION

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ABSTRACT

Objective: To conduct a combined analysis of the estimated effects of maternal average weekly alcohol consumption, and any binge drinking, in early to mid pregnancy on general intelligence, attention, and executive function in 5-year-old children.

Design: Follow-up study.

Setting: Neuropsychological testing in four Danish cities 2003–2008.

Population: A cohort of 1628 women and their children sampled from the Danish National Birth Cohort.

Methods: Participants were sampled based on maternal alcohol consumption during early pregnancy. At age 5 years, the children were tested for general intelligence, attention, and executive function. The three outcomes were analysed together in a multivariate model to obtain joint estimates and P values for the association of alcohol across outcomes. The effects of low to moderate alcohol consumption and binge drinking in early pregnancy were adjusted for a wide range of potential confounding factors.

Main outcome measures: Wechsler Preschool and Primary Scale of Intelligence—Revised (WPPSI-R), the Test of Everyday Attention for Children at Five (TEACh-5), and the Behavior Rating Inventory of Executive Functions (BRIEF) scores.

Results: Multivariate analyses showed no statistically significant effects arising from average weekly alcohol consumption or any binge drinking, either individually or in combination. These results replicate findings from separate analyses of each outcome variable.

Conclusions: The present study contributes comprehensive methodological and statistical approaches that should be incorporated in future studies of low to moderate alcohol consumption and binge drinking during pregnancy. Furthermore, as no safe level of drinking during pregnancy has been established, the most conservative advice for women is not to drink alcohol during pregnancy. However, the present study suggests that small volumes consumed occasionally may not present serious concern.

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2. THE EFFECTS OF LOW TO MODERATE PRENATAL ALCOHOL EXPOSURE IN EARLY PREGNANCY ON IQ IN 5-YEAR-OLD CHILDREN

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ABSTRACT
Objective: To examine the effects of low to moderate maternal alcohol consumption during early pregnancy on children's intelligence (IQ) at age 5 years.

Design: Prospective follow-up study.

Setting: Neuropsychological testing in four Danish cities 2003–2008.

Population: A cohort of 1628 women and their children sampled from the Danish National Birth Cohort.

Methods: Participants were sampled based on maternal alcohol consumption during pregnancy. At 5 years of age, children were tested with the Wechsler Preschool and Primary Scale of Intelligence—Revised (WPPSI-R). Parental education, maternal IQ, maternal smoking in pregnancy, the child's age at testing, gender, and tester were considered core confounding factors, whereas the full model also controlled for maternal binge drinking, age, BMI, parity, home environment, postnatal smoking in the home, health status, and indicators for hearing and vision impairments.

Main outcome measures: The WPPSI-R.

Results: No differences in test performance were observed between children whose mothers reported consuming between one and four or between five and eight drinks per week at some point during pregnancy, compared with children of mothers who abstained. For women who reported consuming nine or more drinks per week no differences were observed for mean differences; however, the risks of low full-scale IQ (OR 4.6; 95% CI 1.2–18.2) and low verbal IQ (OR 5.9; 95% CI 1.4–24.9) scores, but not low performance IQ score, were increased.

Conclusions: Maternal consumption of low to moderate quantities of alcohol during pregnancy was not associated with the mean IQ score of preschool children. Despite these findings, acceptable levels of alcohol use during pregnancy have not yet been established, and conservative advice for women continues to be to avoid alcohol use during pregnancy.

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3. THE EFFECT OF ALCOHOL BINGE DRINKING IN EARLY PREGNANCY ON GENERAL INTELLIGENCE IN CHILDREN

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ABSTRACT

Objective: To examine the effects of binge alcohol consumption during early pregnancy, including the number of binge episodes and the timing of binge drinking, on general intelligence in 5-year-old children.

Design: Follow-up study.

Setting: Neuropsychological testing in four Danish cities 2003–2008.

Population: A cohort of 1617 women and their children sampled from the Danish National Birth Cohort.

Methods: Participants were sampled on the basis of maternal alcohol consumption during pregnancy. At 5 years of age the children were tested with six subtests from the Wechsler Preschool and Primary Scale of Intelligence – Revised (WPPSI-R). Parental education, maternal IQ, prenatal maternal smoking, the child's age at testing, the gender of the child, and tester were considered core confounding factors, whereas the full model also controlled for prenatal maternal average alcohol intake, maternal age, maternal pre-pregnancy body mass index (BMI), parity, home environment, postnatal parental smoking, health status, and indicators for hearing and vision impairment.

Main outcome measure: WPPSI-R.

Results: There were no systematic or significant differences in general intelligence between children of mothers reporting binge drinking and children of mothers with no binge episodes, except that binge drinking in gestational weeks 1–2 significantly reduced the risk of low, full-scale IQ (OR 0.54; 95% CI 0.31–0.96) when adjusted for core confounding factors. The results were otherwise not statistically significantly related to the number of binge episodes (with a maximum of 12) and timing of binge drinking.

Conclusions: We found no systematic association between binge drinking during early pregnancy and child intelligence. However, binge drinking reduced the risk of low, full-scale IQ in gestational weeks 1–2. This finding may be explained by residual confounding.

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4. THE EFFECTS OF LOW TO MODERATE ALCOHOL CONSUMPTION AND BINGE DRINKING IN EARLY PREGNANCY ON SELECTIVE AND SUSTAINED ATTENTION IN 5-YEAR-OLD CHILDREN

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ABSTRACT

Objective: The aim was to examine the effects of low to moderate maternal alcohol consumption and binge drinking in early pregnancy on children's attention at 5 years of age.

Design: Prospective follow-up study.

Setting: Neuropsychological testing in four Danish cities 2003–2008.

Population: A cohort of 1628 women and their children sampled from the Danish National Birth Cohort.

Methods: Participants were sampled based on maternal alcohol consumption during pregnancy. At 5 years of age, the children were tested with the recently developed Test of Everyday Attention for Children at Five (TEACh-5). Parental education, maternal IQ, maternal smoking in pregnancy, the child's age at testing, gender, and tester were considered core confounding factors, whereas the full model also controlled the following potential confounding factors: maternal binge drinking or low to moderate alcohol consumption, age, body mass index (BMI), parity, home environment, postnatal smoking in the home, child's health status, and indicators for hearing and vision impairments.

Main outcome measures: TEACh-5 attention scores.

Results: There were no significant effects on test performance in children of mothers drinking up to 8 drinks per week compared with children of mothers who abstained, but there was a significant association between maternal consumption of 9 or more drinks per week and risk of a low overall attention score (OR 3.50, 95% CI 1.15–10.68). No consistent or significant associations were observed between binge drinking and attention test scores.

Conclusions: The findings suggest an effect of maternal consumption of 9 or more drinks per week on attention functions in children, but the study detected no effects of lower levels of maternal consumption and no consistent effects of maternal binge drinking.

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5. THE EFFECTS OF LOW TO MODERATE ALCOHOL CONSUMPTION AND BINGE DRINKING IN EARLY PREGNANCY ON EXECUTIVE FUNCTION IN 5-YEAR-OLD CHILDREN

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ABSTRACT

Objective: To examine the effects of low to moderate maternal alcohol consumption and binge drinking in early pregnancy on children's executive functions at the age of 5 years.

Design: Follow-up study.

Setting: Neuropsychological testing in four Danish cities 2003–2008.

Population: A cohort of 1628 women and their children sampled from the Danish National Birth Cohort.

Methods: Participants were sampled based on maternal alcohol drinking patterns during early pregnancy. When the children were 5 years old, the parent and teacher forms of the Behaviour Rating Inventory of Executive Function (BRIEF) were completed by the mothers and a preschool teacher. Parental education, maternal IQ, prenatal maternal smoking, the child's age at testing, and the child's gender were considered core confounding factors. The full model also included maternal binge drinking or low to moderate alcohol consumption, maternal age, parity, maternal marital status, family home environment, postnatal parental smoking, pre-pregnancy maternal body mass index (BMI), and the health status of the child.

Main outcome measures: The BRIEF parent and teacher forms.

Results: Adjusted for all potential confounding factors, no statistically significant associations between maternal low to moderate average weekly consumption and BRIEF index scores were observed. In adjusted analyses, binge drinking in gestational week 9 or later was significantly associated with elevated Behavioural Regulation Index parent scores (OR 2.04, 95% CI 0.33–3.76), and with the risk of high scores on the Metacognitive Index assessed by the teacher (OR 2.06, 95% CI 1.01–4.23).

Conclusions: This study did not observe significant effects of low to moderate alcohol consumption during pregnancy on executive functioning at the age of 5 years. Furthermore, only weak and no consistent associations between maternal binge drinking and executive functions were observed.

Read Full Article,
http://onlinelibrary.wiley.com/doi/10.1111/j.1471-0528.2012.03397.x/abstract

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PubMed, Anal Bioanal Chem.
2012 Jun 6.

6. A NEW METHOD FOR QUANTIFYING PRENATAL EXPOSURE TO ETHANOL BY MICROWAVE-ASSISTED EXTRACTION (MAE) OF MECONIUM FOLLOWED BY GAS CHROMATOGRAPHY-MASS SPECTROMETRY (GC-MS)

Cabarcos P, Tabernero MJ, Alvarez I, Miguez M, Fernández P, Bermejo AM.
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ABSTRACT

Ethanol is a legal and widely available substance. There are health and social consequences associated with its abuse. One of the most important problems is related to alcohol consumption during pregnancy. In fact, prenatal ethanol exposure can be associated with fetal alcohol spectrum disorder (FASD), a term used to describe a wide range of potentially lifelong effects that include physical, mental, behavioral, and learning disabilities. Fatty acid ethyl esters (FAEEs), which are non-oxidative metabolites of ethanol, are currently used as biomarkers of direct ethanol consumption in different matrices, including hair, blood, skin surface, and meconium. Analysis of these compounds in meconium reveals exposure to alcohol during the second and third trimesters of pregnancy. An important finding for evaluation of gestational ethanol exposure is the fact that FAEEs do not cross the placenta. Because they accumulate in the fetal gut from approximately the 20th week of gestation until birth, this provides a wide window of detection of chronic exposure to alcohol. The sum of the concentrations of all the FAEEs, with a cutoff of 2 nmol g(-1) or 600 ng g(-1) meconium, has been recommended as evidence of maternal alcohol use. We introduce a novel technique to quantify ethyl myristate, ethyl palmitate, ethyl stearate, and their deuterated analogues (as internal standards, IS) in meconium using microwave-assisted extraction (MAE) coupled with gas chromatography-mass spectrometry (GC-MS). Limits of detection and quantification were 50 and 100 ng g(-1) for all analytes except ethyl stearate (LOD 100 ng g(-1) and LOQ 500 ng g(-1)). Calibration curves were linear from the LOQ to 5000 ng g(-1). The validated method was applied to the analysis of 81 meconium samples.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22669306

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PubMed, Hum Brain Mapp. 2012 Jun 5. doi: 10.1002/hbm.22110.

7. PRIMARY VISUAL RESPONSE (M100) DELAYS IN ADOLESCENTS WITH FASD AS MEASURED WITH MEG

Coffman BA, Kodituwakku P, Kodituwakku EL, Romero L, Sharadamma NM, Stone D, Stephen JM.
The Mind Research Network and Lovelace Biomedical and Environmental Research Institute, Albuquerque, New Mexico; Department of Psychology, University of New Mexico, Albuquerque, New Mexico. bcoffman@mrn.org

ABSTRACT

Fetal alcohol spectrum disorders (FASD) are debilitating, with effects of prenatal alcohol exposure persisting into adolescence and adulthood. Complete characterization of FASD is crucial for the development of diagnostic tools and intervention techniques to decrease the high cost to individual families and society of this disorder. In this experiment, we investigated visual system deficits in adolescents (12-21 years) diagnosed with an FASD by measuring the latency of patients' primary visual M100 responses using MEG. We hypothesized that patients with FASD would demonstrate delayed primary visual responses compared to controls. M100 latencies were assessed both for

FASD patients and age-matched healthy controls for stimuli presented at the fovea (central stimulus) and at the periphery (peripheral stimuli; left or right of the central stimulus) in a saccade task requiring participants to direct their attention and gaze to these stimuli.

Source modeling was performed on visual responses to the central and peripheral stimuli and the latency of the first prominent peak (M100) in the occipital source timecourse was identified. The peak latency of the M100 responses were delayed in FASD patients for both stimulus types (central and peripheral), but the difference in latency of primary visual responses to central vs. peripheral stimuli was significant only in FASD patients, indicating that, while FASD patients' visual systems are impaired in general, this impairment is more pronounced in the periphery.

These results suggest that basic sensory deficits in this population may contribute to sensorimotor integration deficits described previously in this disorder.

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http://www.ncbi.nlm.nih.gov/pubmed/22674650

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PubMed, Drug Alcohol Rev. 2012 Jun 5. doi: 10.1111/j.1465-3362.2012.00475.x.

8. LACK OF INTERNATIONAL CONSENSUS IN LOW-RISK DRINKING GUIDELINES

Furtwaengler NA, de Visser RO.
School of Psychology, University of Sussex, Brighton, UK.

ABSTRACT

Introduction and aims: To encourage moderate alcohol consumption, many governments have developed guidelines for alcohol intake, guidelines for alcohol consumption during pregnancy and legislation relating to blood alcohol limits when driving. The aim of this study was to determine the degree of international consensus within such guidelines.

Design and methods: Official definitions of standard drinks and consumption guidelines were searched for on government websites, including all 27 European Union Member States and countries from all global geographic regions.

Results: There was a remarkable lack of agreement about what constitutes harmful or excessive alcohol consumption on a daily basis, a weekly basis and when driving, with no consensus about the ratios of consumption guidelines for men and women.

Discussion and conclusions: International consensus in low-risk drinking guidelines is an important-and achievable-goal. Such agreement would facilitate consistent labelling of packaged products and could help to promote moderate alcohol consumption. However, there are some paradoxes related to alcohol content labelling and people's use of such information: although clearer information could increase people's capacity to monitor and regulate their alcohol consumption, not all drinkers are motivated to drink moderately or sensibly, and drinkers who intend to get drunk may use alcohol content labelling to select more alcoholic products.

Link to the Article,
http://www.ncbi.nlm.nih.gov/pubmed/22672631

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PubMed, PLoS One. 2012;7(5):e38057. Epub 2012 May 29.

9. FOLIC ACID TRANSPORT TO THE HUMAN FETUS IS DECREASED IN PREGNANCIES WITH CHRONIC ALCOHOL EXPOSURE

Hutson JR, Stade B, Lehotay DC, Collier CP, Kapur BM.
Division of Clinical Pharmacology and Toxicology, Hospital for Sick Children, Toronto, Ontario, Canada.

ABSTRACT

Background: During pregnancy, the demand for folic acid increases since the fetus requires this nutrient for its rapid growth and cell proliferation. The placenta concentrates folic acid into the fetal circulation; as a result the fetal levels are 2 to 4 times higher than the maternal level. Animal and in vitro studies have suggested that alcohol may impair transport of folic acid across the placenta by decreasing expression of transport proteins. We aim to determine if folate transfer to the fetus is altered in human pregnancies with chronic alcohol consumption.

Methodology/principal findings: Serum folate was measured in maternal blood and umbilical cord blood at the time of delivery in pregnancies with chronic and heavy alcohol exposure (n=23) and in non-drinking controls (n=24). In the alcohol-exposed pairs, the fetal: maternal serum folate ratio was ≤1.0 in over half (n=14), whereas all but one of the controls were >1.0. Mean folate in cord samples was lower in the alcohol-exposed group than in the controls (33.15±19.89 vs 45.91±20.73, p=0.04).

Conclusions/Significance: Our results demonstrate that chronic and heavy alcohol use in pregnancy impairs folate transport to the fetus. Altered folate concentrations within the placenta and in the fetus may in part contribute to the deficits observed in the fetal alcohol spectrum disorders.

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http://www.ncbi.nlm.nih.gov/pubmed/22666445

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PubMed, Neurotoxicol Teratol. 2012 May 28.

10. MEMORY ABILITY AND HIPPOCAMPAL VOLUME IN ADOLESCENTS WITH PRENATAL DRUG EXPOSURE

Riggins T, Cacic K, Buckingham-Howes S, Scaletti LA, Salmeron BJ, Black MM.
University of Maryland, College Park, United States.

ABSTRACT

The objective of the present study was to examine the influence of prenatal drug exposure (PDE) on memory performance and supporting brain structures (i.e., hippocampus) during adolescence. To achieve this goal, declarative memory ability and hippocampal volume were examined in a well-characterized sample of 138 adolescents (76 with a history of PDE and 62 from a non-exposed comparison group recruited from the same community, mean age=14years). Analyses were adjusted for: age at time of the assessments, gender, IQ, prenatal exposure to alcohol and tobacco, and indices of early childhood environment (i.e., caregiver depression, potential for child abuse, and number of caregiver changes through 7years of age).

Results revealed that adolescents with a history of PDE performed worse on the California Verbal Learning Test-Child Version (CVLT-C), on story recall from the Children's Memory Scale (CMS), and had larger hippocampal volumes, even after covariate adjustment. Hippocampal volume was negatively correlated with memory performance on the CVLT-C, with lower memory scores associated with larger volumes.

These findings provide support for long-term effects of PDE on memory function and point to neural mechanisms that may underlie these outcomes.

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http://www.ncbi.nlm.nih.gov/pubmed/22652523

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PubMed, Dev Neurobiol. 2012 May 24. doi: 10.1002/dneu.22035.

11. NEUROIMMUNE MECHANISMS IN FETAL ALCOHOL SPECTRUM DISORDER

Kane CJ, Phelan KD, Drew PD.
Department of Neurobiology and Developmental Sciences, University of Arkansas for Medical Sciences, Little Rock, AR 72205. kanecynthiaj@uams.edu

ABSTRACT
Fetal alcohol spectrum disorder (FASD) is a major health concern worldwide and results from maternal consumption of alcohol during pregnancy. It produces tremendous individual, social, and economic losses. This review will first summarize the structural, functional and behavior changes seen in FASD. The development of the neuroimmune system will be then be described with particular emphasis on the role of microglial cells in the normal regulation of homeostatic function in the central nervous system (CNS) including synaptic transmission. The impact of alcohol on the neuroimmune system in the developing CNS will be discussed in the context of several key immune molecules and signaling pathways involved in neuroimmune mechanisms that contribute to FASD. This review concludes with a summary of the development of early therapeutic approaches utilizing immunosuppressive drugs to target alcohol-induced pathologies. The significant role played by neuroimmune mechanisms in alcohol addiction and pathology provides a focus for future research aimed at understanding and treating the consequences of FASD.

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http://www.ncbi.nlm.nih.gov/pubmed/22623427

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PubMed, Biol Psychiatry. 2012 May 22.

12. MALE GERMLINE TRANSMITS FETAL ALCOHOL ADVERSE EFFECT ON HYPOTHALAMIC PROOPIOMELANOCORTIN GENE ACROSS GENERATIONS

Govorko D, Bekdash RA, Zhang C, Sarkar DK.
Rutgers Endocrine Program, Rutgers, The State University of New Jersey, New Brunswick, New Jersey.

ABSTRACT
Background: Neurons containing proopiomelanocortin (POMC)-derived peptides, known to control stress axis, metabolic, and immune functions, have a lower function in patients with a family history of alcoholism, raising the possibility that alcohol effects on the POMC system may transmit through generations. Here we describe epigenetic modifications of Pomc gene that transmit through generation via male germline and may be critically involved in alcoholism-inherited diseases.

Methods: Whether an epigenetic mechanism is involved in causing a Pomc expression deficit in fetal alcohol-exposed rats is studied by determining Pomc gene methylation, expression, and functional abnormalities and their normalization following suppression of DNA methylation or histone acetylation. Additionally, transgenerational studies were conducted to evaluate the germline-transmitted effect of alcohol.

Results: Fetal alcohol-exposed male and female rat offspring showed a significant deficit in POMC neuronal functions. Associated with this was an increased methylation status of several CpG dinucleotides in the proximal part of the Pomc promoter region and altered level of histone-modifying proteins and DNA methyltransferases levels in POMC neurons. Suppression of histone deacetylation and DNA methylation normalized Pomc expression and functional abnormalities. Fetal alcohol-induced Pomc gene methylation, expression, and functional defects persisted in the F2 and F3 male but not in female germline.

Additionally, the hypermethylated Pomc gene was detected in sperm of fetal alcohol-exposed F1 offspring that was transmitted through F3 generation via male germline.

Conclusions: Trangenerational epigenetic studies should spur new insight into the biological mechanisms that influence the sex-dependent difference in genetic risk of alcoholism-inherited diseases.

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http://www.ncbi.nlm.nih.gov/pubmed/22622000

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PubMed, Neurochem Int. 2012 May 18.

13. ATTENUATION OF NF-KB MEDIATED APOPTOTIC SIGNALING BY TOCOTRIENOL AMELIORATES COGNITIVE DEFICITS IN RATS POSTNATALLY EXPOSED TO ETHANOL

Tiwari V, Arora V, Chopra K.
Pharmacology Research Laboratory, University Institute of Pharmaceutical Sciences, UGC Center of Advanced Study, Panjab University, Chandigarh 160 014, India.

ABSTRACT

Ethanol-induced damage in the developing brain may result in cognitive impairment including deficits on neuropsychological tests of learning, memory and executive function, yet the underlying mechanisms remain elusive. In the present study we investigated the protective effect of tocotrienol against cognitive deficit, neuroinflammation and neuronal apoptosis in rat pups postnatally exposed to ethanol. Pups were administered ethanol (5g/kg, 12% v/v) by intragastric intubation on postnatal days 7, 8 and 9. Ethanol-exposed pups showed significant memory impairment in Morris water maze task as evident from increase in escape latency and total distance travelled to reach the hidden platform. Time spent in target quadrant, % total distance traversed in target quadrant and frequency of appearance in target quadrant was also significantly decreased in ethanol exposed pups in probe trial. Poor memory retention was exhibited by ethanol-exposed pups in elevated plus maze test also. Impaired cognition was associated with significantly enhanced acetylcholinesterase activity, increased neuroinflammation (oxidative-nitrosative stress, TNF-α, IL-1β and TGF-β1) and neuronal apoptosis (NF-κβ and Caspase-3) in different brain regions of ethanol-exposed pups. Co-administration with tocotrienol significantly ameliorated all the behavioral, biochemical and molecular alterations in the different brain regions of ethanol exposed pups. The current study thus demonstrates the possible involvement of NF-κβ mediated apoptotic signaling in cognitive deficits associated with postnatal ethanol exposure in rats and points to the potential of tocotrienol in the prevention of cognitive deficits in children with fetal alcohol spectrum disorders (FASDs).

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22613133

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PubMed, J Perinatol. 2012 May 17. doi: 10.1038/jp.2012.57.

14. PRENATAL ALCOHOL EXPOSURE, BLOOD ALCOHOL CONCENTRATIONS AND ALCOHOL ELIMINATION RATES FOR THE MOTHER, FETUS AND NEWBORN

Burd L, Blair J, Dropps K.
North Dakota Fetal Alcohol Syndrome Center, Department of Pediatrics, University of North Dakota School of Medicine and Health Sciences, Grand Forks, ND, USA.

ABSTRACT
Fetal alcohol spectrum disorders (FASDs) are a common cause of intellectual impairment and birth defects. More recently, prenatal alcohol exposure (PAE) has been found to be a risk factor for fetal mortality, stillbirth and infant and child mortality. This has led to increased concern about detection and management of PAE. One to 2 h after maternal ingestion, fetal blood alcohol concentrations (BACs) reach levels nearly equivalent to maternal levels. Ethanol elimination by the fetus is impaired because of reduced metabolic capacity. Fetal exposure time is prolonged owing to the reuptake of amniotic-fluid containing ethanol by the fetus. Alcohol elimination from the fetus relies on the mother's metabolic capacity. Metabolic capacity among pregnant women varies eightfold (from 0.0025 to 0.02 g dl(-1) h(-1)), which may help explain how similar amounts of ethanol consumption during pregnancy results in widely varying phenotypic presentations of FASD. At birth physiological changes alter the neonate's metabolic capacity and it rapidly rises to a mean value of 83.5% of the mother's capacity. FASDs are highly recurrent and younger siblings have increased risk. Detection of prenatal alcohol use offers an important opportunity for office-based interventions to decrease exposure for the remainder of pregnancy and identification of women who need substance abuse treatment. Mothers of children with FAS have been found to drink faster, get drunk quicker and to have higher BACs. A modest increase in the prevalence of a polymorphism of alcohol dehydrogenase, which increases susceptibility to adverse outcomes from PAE has been reported. Lastly, detection of alcohol use and appropriate management would decrease risk from PAE for subsequent pregnancies.

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http://www.ncbi.nlm.nih.gov/pubmed/22595965

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Wiley Online Library - Alcoholism: Clinical and Experimental Research
Article first published online: 17 MAY 2012
DOI: 10.1111/j.1530-0277.2012.01819.x

15. PERSISTENT DOSE-DEPENDENT CHANGES IN BRAIN STRUCTURE IN YOUNG ADULTS WITH LOW-TO-MODERATE ALCOHOL EXPOSURE IN UTERO

Kristen L. Eckstrand[1,2], Zhaohua Ding[1,2], Neil C. Dodge[3], Ronald L. Cowan[2,4], Joseph L. Jacobson[3], Sandra W. Jacobson[3], Malcolm J. Avison[1,2]
1 Department of Radiology and Radiological Sciences, Vanderbilt University Medical Center, Nashville, Tennessee
2 Vanderbilt University Institute of Imaging Science, Vanderbilt University Medical Center, Nashville, Tennessee
3 Department of Psychiatry and Behavioral Neurosciences , Wayne State University School of Medicine, Detroit, Michigan;
4 Department of Psychiatry, Vanderbilt University Medical Center, Nashville, Tennessee

ABSTRACT
Background: Many children with heavy exposure to alcohol in utero display characteristic alterations in brain size and structure. However, the long-term effects of low-to-moderate alcohol exposure on these outcomes are unknown.

Methods: Using voxel-based morphometry and region-of-interest analyses, we examined the influence of lower doses of alcohol on gray and white matter composition in a prospectively

recruited, homogeneous, well-characterized cohort of alcohol-exposed (n = 11, age 19.5 ± 0.3 years) and control (n = 9, age 19.6 ± 0.5 years) young adults. A large proportion of the exposed individuals were born to mothers whose alcohol consumption during pregnancy was in the low-to-moderate range.

Results: There were no differences in total brain volume or total gray or white matter volume between the exposed and control groups. However, gray matter volume was reduced in alcohol-exposed individuals in several areas previously reported to be affected by high levels of exposure, including the left cingulate gyrus, bilateral middle frontal gyri, right middle temporal gyrus, and right caudate nucleus. Notably, this gray matter loss was dose dependent, with higher exposure producing more substantial losses.

Conclusions: These results indicate that even at low doses, alcohol exposure during pregnancy impacts brain development and that these effects persist into young adulthood.

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http://onlinelibrary.wiley.com/doi/10.1111/j.1530-0277.2012.01819.x/abstract;jsessionid=8041CA4C86428C304CBFBA62249D539B.d02t03

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PubMed, J Autism Dev Disord. 2012 May 17.

16. TREATMENTS FOR NEURODEVELOPMENTAL DISORDERS: EVIDENCE, ADVOCACY, AND THE INTERNET

Di Pietro NC, Whiteley L, Mizgalewicz A, Illes J.
National Core for Neuroethics, Division of Neurology, Faculty of Medicine, UBC Hospital, University of British Columbia, 2211 Wesbrook Mall, Koerner S124, Vancouver, BC, V6T 2B5, Canada.

ABSTRACT
The Internet is a major source of health-related information for parents of sick children despite concerns surrounding quality. For neurodevelopmental disorders, the websites of advocacy groups are a largely unexamined source of information. We evaluated treatment information posted on nine highly-trafficked advocacy websites for autism, cerebral palsy, and fetal alcohol spectrum disorder. We found that the majority of claims about treatment safety and efficacy were unsubstantiated. Instead, a range of rhetorical strategies were used to imply scientific support. When peer-reviewed publications were cited, 20 % were incorrect or irrelevant. We call for new partnerships between advocacy and experts in developmental disorders to ensure better accuracy and higher transparency about how treatment information is selected and evidenced on advocacy websites.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22592952

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PubMed, Front Genet. 2012;3:77. Epub 2012 May 16.

17. MICRORNAS AND FETAL BRAIN DEVELOPMENT: IMPLICATIONS FOR ETHANOL TERATOLOGY DURING THE SECOND TRIMESTER PERIOD OF NEUROGENESIS

Miranda RC.
Texas A&M Health Science Center Bryan, USA.

ABSTRACT
Maternal ethanol consumption during pregnancy can lead to a stereotypic cluster of fetal craniofacial, cardiovascular, skeletal, and neurological deficits that are collectively termed the fetal

alcohol spectrum disorder (FASD). Fetal ethanol exposure is a leading non-genetic cause of mental retardation. Mechanisms underlying the etiology of ethanol teratology are varied and complex. This review will focus on the developing brain as an important and vulnerable ethanol target. Near the end of the first trimester, and during the second trimester, fetal neural stem cells (NSCs) produce most of the neurons of the adult brain, and ethanol has been shown to influence NSC renewal and maturation. We will discuss the neural developmental and teratological implications of the biogenesis and function of microRNAs (miRNAs), a class of small non-protein-coding RNAs that control the expression of gene networks by translation repression. A small but growing body of research has identified ethanol-sensitive miRNAs at different stages of NSC and brain maturation. While many miRNAs appear to be vulnerable to ethanol at specific developmental stages, a few, like the miR-9 family, appear to exhibit broad vulnerability to ethanol across multiple stages of NSC differentiation. An assessment of the regulation and function of these miRNAs provides important clues about the mechanisms that underlie fetal vulnerability to alterations in the maternal-fetal environment and yields insights into the genesis of FASD.

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http://www.ncbi.nlm.nih.gov/pubmed/22623924

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Wiley Online Library - Alcoholism: Clinical and Experimental Research
Article first published online: 16 MAY 2012
DOI: 10.1111/j.1530-0277.2012.01848.x

18. A STUDY EVALUATING FOR A THRESHOLD EFFECT OF ALCOHOL CONSUMPTION IN PREGNANCY ON INFANT PHYSICAL CHARACTERISTICS IDEALLY HAS A CONTROL GROUP NOT INGESTING ALCOHOL

Eric Roehm

ABSTRACT

No abstract is available for this article.

Read Full Article,
http://onlinelibrary.wiley.com/doi/10.1111/j.1530-0277.2012.01848.x/full

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PubMed, Alcohol Clin Exp Res. 2012 May 15. doi: 10.1111/j.1530-0277.2011.01718.x.

19. EXECUTIVE FUNCTION PREDICTS ADAPTIVE BEHAVIOR IN CHILDREN WITH HISTORIES OF HEAVY PRENATAL ALCOHOL EXPOSURE AND ATTENTION-DEFICIT/HYPERACTIVITY DISORDER.

Ware AL, Crocker N, O'Brien JW, Deweese BN, Roesch SC, Coles CD, Kable JA, May PA, Kalberg WO, Sowell ER, Jones KL, Riley EP, Mattson SN; the CIFASD.
Center for Behavioral Teratology, San Diego State University, San Diego, California.

ABSTRACT

Background: Prenatal exposure to alcohol often results in disruption to discrete cognitive and behavioral domains, including executive function (EF) and adaptive functioning. In the current study, the relation between these 2 domains was examined in children with histories of heavy prenatal alcohol exposure, nonexposed children with a diagnosis of attention-deficit/hyperactivity disorder (ADHD), and typically developing controls.

Methods: As part of a multisite study, 3 groups of children (8 to 18 years, M = 12.10) were tested:

children with histories of heavy prenatal alcohol exposure (ALC, n = 142), nonexposed children with ADHD (ADHD, n = 82), and typically developing controls (CON, n = 133) who did not have ADHD or a history of prenatal alcohol exposure. Children completed subtests of the Delis-Kaplan Executive Function System (D-KEFS), and their primary caregivers completed the Vineland Adaptive Behavior Scales-II. Data were analyzed using regression analyses.

Results: Analyses showed that EF measures were predictive of adaptive abilities, and significant interactions between D-KEFS measures and group were present. For the ADHD group, the relation between adaptive abilities and EF was more general, with 3 of the 4 EF measures showing a significant relation with adaptive score. In contrast, for the ALC group, this relation was specific to the nonverbal EF measures. In the CON group, performance on EF tasks did not predict adaptive scores over the influence of age.

Conclusions: These results support prior research in ADHD, suggesting that EF deficits are predictive of poorer adaptive behavior and extend this finding to include children with heavy prenatal exposure to alcohol. However, the relation between EF and adaptive ability differed by group, suggesting unique patterns of abilities in these children. These results provide enhanced understanding of adaptive deficits in these populations, as well as demonstrate the ecological validity of laboratory measures of EF.

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http://www.ncbi.nlm.nih.gov/pubmed/22587709

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PubMed, Int J Dev Neurosci. 2012 May 14.

20. EARLY ALCOHOL EXPOSURE DISRUPTS VISUAL CORTEX PLASTICITY IN MICE.
Lantz CL, Wang W, Medina AE.
Department of Anatomy and Neurobiology, School of Medicine, Virginia Commonwealth University, Richmond, VA 23298, United States.

ABSTRACT

There is growing evidence that deficits in neuronal plasticity underlie the cognitive problems seen in fetal alcohol spectrum disorders (FASD). However, the mechanisms behind these deficits are not clear. Here we test the effects of early alcohol exposure on ocular dominance plasticity (ODP) in mice and the reversibility of these effects by phosphodiesterase (PDE) inhibitors. Mouse pups were exposed to 5g/kg of 25% ethanol i.p. on postnatal days (P) 5, 7 and 9. This type of alcohol exposure mimics binge drinking during the third trimester equivalent of human gestation. To assess ocular dominance plasticity animals were monocularly deprived at P21 for 10 days, and tested using optical imaging of intrinsic signals. During the period of monocular deprivation animals were treated with vinpocetine (20mg/kg; PDE1 inhibitor), rolipram (1.25mg/kg; PDE4 inhibitor), vardenafil (3mg/kg; PDE5 inhibitor) or vehicle solution. Monocular deprivation resulted in the expected shift in ocular dominance of the binocular zone in saline controls but not in the ethanol group. While vinpocetine successfully restored ODP in the ethanol group, rolipram and vardenafil did not. However, when rolipram and vardenafil were given simultaneously ODP was restored. PDE4 and PDE5 are specific to cAMP and cGMP respectively, while PDE1 acts on both of these nucleotides. Our findings suggest that the combined activation of the cAMP and cGMP cascades may be a good approach to improve neuronal plasticity in FASD models.

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http://www.ncbi.nlm.nih.gov/pubmed/22617459

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PubMed, BJOG. 2012 May 9. doi: 10.1111/j.1471-0528.2012.03333.x.

21. HEAVY PRENATAL ALCOHOL EXPOSURE AND INCREASED RISK OF STILLBIRTH

O'Leary C, Jacoby P, D'Antoine H, Bartu A, Bower C.
Centre for Population Health Research, Curtin Health Information Research Institute, Curtin University, Perth, WA Division of Population Sciences, Telethon Institute for Child Health Research, West Perth, WA Centre for Child Health Research, University of Western Australia, Perth, WA Menzies School of Health Research, Darwin, NT Faculty of Health Sciences, School of Nursing and Midwifery, Curtin University, Perth, WA, Australia.

ABSTRACT

Objective: To investigate the association between heavy prenatal alcohol exposure and stillbirth. Design Data linkage cohort study. Setting Western Australia (WA). Population The exposed cohort included mothers with an alcohol-related diagnosis (International Classification of Diseases, ninth/tenth revisions) recorded in health data sets and all their offspring born in WA (1983-2007). Mothers without an alcohol-related diagnosis and their offspring comprised the comparison cohort.

Methods: Exposed and comparison mothers were identified through the WA Data Linkage System. Odds ratios for stillbirth at 20 + weeks of gestation were estimated by logistic regression, stratified by Aboriginal status. Main outcome measures The proportion of stillbirths at 20 + weeks of gestation is presented per 1000 births, as well as adjusted odds ratios (aOR) and 95% confidence intervals (95% CI), and population-attributable fractions.

Results: Increased odds of stillbirth were observed for mothers with an alcohol-related diagnosis at any stage of their life for both non-Aboriginal (aOR 1.36; 95% CI 1.05-1.76) and Aboriginal (aOR 1.33; 95% CI 1.08-1.64) births. When an alcohol diagnosis was recorded during pregnancy, increased odds were observed for non-Aboriginal births (aOR 2.24; 95% CI 1.09-4.60), with the highest odds of Aboriginal stillbirth occurring when an alcohol diagnosis was recorded within 1 year postpregnancy (aOR 2.88; 95% CI 1.75-4.73). The population-attributable fractions indicate that 0.8% of non-Aboriginal and 7.9% of Aboriginal stillbirths are the result of heavy alcohol consumption.

Conclusions: Prevention of heavy maternal alcohol use has the potential to reduce stillbirths. The lack of an association between exposure during pregnancy and Aboriginal stillbirth in this study needs further investigation.

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http://www.ncbi.nlm.nih.gov/pubmed/22568788

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PubMed, Dev Neurosci. 2012 May 8.

22. MOLECULAR SUBSTRATES OF SOCIAL AVOIDANCE SEEN FOLLOWING PRENATAL ETHANOL EXPOSURE AND ITS REVERSAL BY SOCIAL ENRICHMENT

Middleton FA, Varlinskaya EI, Mooney SM.
Department of Neuroscience and Physiology, SUNY Upstate Medical University, Syracuse, N.Y., USA.

ABSTRACT

Prenatal ethanol exposure is associated with, and is a risk factor for, developmental disorders with abnormal social behaviors, including autism spectrum disorders. We hypothesize that the specific effects of ethanol on social behavior are defined by the timing of the exposure as well as subsequent changes in brain regions such as the amygdala and ventral striatum. We recently reported that in utero ethanol exposure on gestational day 12 alters social behaviors of weanling [postnatal day (P) 28], adolescent (P42), and young adult (P75) rats. Male, but not female,

offspring of the ethanol-exposed dams showed significant decreases in social investigation (sniffing of a social partner), contact behavior (grooming or crawling over/under the partner), and play fighting (following, chasing, nape attacks, or pinning) at all ages tested with maximal effects at P28 and P42.

Furthermore, ethanol-exposed males and females showed evidence of social avoidance at P42 and P75. The present study sought to test whether a form of social enrichment could normalize any of the social deficits and what the molecular mechanisms of such effects might be. We found that housing rats with nonmanipulated control rats normalized the social avoidance phenotype normally seen when they are housed with sex-matched prenatal ethanol-exposed littermates. There was no mitigation of the other ethanol-induced behavioral deficits. Conversely, male control-treated rats housed with nonlittermates showed deficits in play fighting, social investigation and contact behavior. Molecular analyses of the amygdala and ventral striatum of adolescent rats following fetal ethanol exposure indicated several specific neurotransmitter systems and pathways that might underlie the social avoidance phenotype as well as its reversal.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22572756

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PubMed, Matern Child Health J. 2012 May 4.

23. PREVALENCE AND CORRELATES OF DRINKING IN EARLY PREGNANCY AMONG WOMEN WHO STOPPED DRINKING ON PREGNANCY RECOGNITION

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ABSTRACT
Women of child bearing age that regularly drink alcohol are at risk for drinking in early pregnancy. Evidence indicates a majority of women stop alcohol consumption on pregnancy recognition. However, there is a dearth of studies reporting on patterns and correlates of drinking in early pregnancy prior to stopping on pregnancy recognition, which the current study aims to address.

In 2005, a New Zealand nationwide cross-sectional survey was conducted on a random sample of 1,256 women aged 16-40 years. Data were collected via an interviewer-administered questionnaire using a web-assisted telephone interviewing system. Of the 1,256 women who participated, 127 (10 %) were currently pregnant and 425 women (34 %) were previously pregnant. Half of currently pregnant women and 37 % of previously pregnant women reported that they ceased drinking on recognising pregnancy. Women categorised as "risky drinkers" and those aged 16-24 years had higher odds to drink and binge drink in early pregnancy, compared with non-risky drinkers and women of other age categories respectively.

A majority of women stop alcohol consumption on pregnancy recognition but prior to this, drink at levels posing a risk for the developing foetus. Women most at risk for drinking and binge drinking in early pregnancy were younger in age and exhibited risky drinking behaviour prior to pregnancy. A targeted intervention to reduce the risk for an alcohol exposed pregnancy is warranted for sexually active younger women in New Zealand and elsewhere.

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http://www.ncbi.nlm.nih.gov/pubmed/22555945

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PubMed, BMJ Open. 2012 May 3;2(3). pii: e000968. doi: 10.1136/bmjopen-2012-000968. Print 2012.

24. THE LILILWAN PROJECT: STUDY PROTOCOL FOR A POPULATION-BASED ACTIVE CASE ASCERTAINMENT STUDY OF THE PREVALENCE OF FETAL ALCOHOL SPECTRUM DISORDERS (FASD) IN REMOTE AUSTRALIAN ABORIGINAL COMMUNITIES

Fitzpatrick JP, Elliott EJ, Latimer J, Carter M, Oscar J, Ferreira M, Olson HC, Lucas B, Doney R, Salter C, Peadon E, Hawkes G, Hand M.
The George Institute for Global Health, Sydney, Australia.

ABSTRACT
Introduction: Anecdotal reports suggest that high-risk drinking in pregnancy is common in some remote Australian communities. Alcohol is teratogenic and may cause a range of lifelong conditions termed 'fetal alcohol spectrum disorders' (FASD). Australia has few diagnostic services for FASD, and prevalence of these neurodevelopmental disorders remains unknown. In 2009, Aboriginal leaders in the remote Fitzroy Valley in North Western Australia identified FASD as a community priority and initiated the Lililwani Project in partnership with leading research organisations. This project will establish the prevalence of FASD and other health and developmental problems in school-aged children residing in the Fitzroy Valley, providing data to inform FASD prevention and management.

Methods And Analysis: This is a population-based active case ascertainment study of all children born in 2002 and 2003 and residing in the Fitzroy Valley. Participants will be identified from the Fitzroy Valley Population Project and Communicare databases. Parents/carers will be interviewed using a standardised diagnostic questionnaire modified for local language and cultural requirements to determine the demographics, antenatal exposures, birth outcomes, education and psychosocial status of each child. A comprehensive interdisciplinary health and neurodevelopmental assessment will be performed using tests and operational definitions adapted for the local context. Internationally recognised diagnostic criteria will be applied to determine FASD prevalence. Relationships between pregnancy exposures and early life trauma, neurodevelopmental, health and education outcomes will be evaluated using regression analysis. Results will be reported according to STROBE guidelines for observational studies.

Ethics And Dissemination: Ethics approval has been granted by the University of Sydney Human Research Ethics Committee, the Western Australian Aboriginal Health Information and Ethics Committee, the Western Australian Country Health Service Board Research Ethics Committee and the Kimberley Aboriginal Health Planning Forum Research Sub-committee. Results will be disseminated widely through peer-reviewed manuscripts, reports, conference presentations and the media.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22556161

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PubMed, Alcohol Clin Exp Res. 2012 May 2. doi: 10.1111/j.1530-0277.2012.01811.x

25. CAUDATE VOLUME PREDICTS NEUROCOGNITIVE PERFORMANCE IN YOUTH WITH HEAVY PRENATAL ALCOHOL EXPOSURE

Fryer SL, Mattson SN, Jernigan TL, Archibald SL, Jones KL, Riley EP.
Department of Psychiatry, University of California, San Francisco, San Francisco, California.

ABSTRACT
Background: Fetal alcohol spectrum disorders result from heavy prenatal alcohol exposure and are characterized, in some cases, by central nervous system anomalies and cognitive impairment.

Regional patterns of neuroanatomical abnormalities suggest that alcohol exerts selective damage on the developing fetal brain. This study assessed brain-behavior relationships in a sample of youth with histories of heavy prenatal alcohol exposure. The aim was to characterize how structural brain alterations observed in our previous studies relate to cognitive deficits commonly reported in individuals with histories of heavy prenatal alcohol exposure.

Methods: Twenty-one youth (mean age 13 years) with histories of heavy prenatal alcohol exposure and 7 nonexposed healthy comparison subjects underwent structural magnetic resonance imaging and neurobehavioral testing. Regional brain volumes within the alcohol-exposed group were correlated with neuropsychological measures of cognitive control and verbal learning/recall, as these aspects of cognition have previously been shown to be vulnerable to alcohol teratogenesis.

Results: Between-group effect sizes revealed moderate to large cognitive performance and brain volume decrements in alcohol-exposed subjects, compared with typically developing peers. Within the alcohol-exposed group, volume of the caudate nuclei was the most consistent predictor of neuropsychological performance, after controlling for potentially confounding variables including total brain volume, IQ, and age.

Conclusions: These data are consistent with previous research associating gestational alcohol exposure with structural and functional changes of the caudate nucleus. Our findings extend this previous work by demonstrating that volume reductions of the caudate have behavioral relevance for this population, in relation to cognitive control and verbal learning and recall abilities.

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http://www.ncbi.nlm.nih.gov/pubmed/22551091

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PubMed, Behav Brain Res. 2012 Apr 27;233(1):162-168.

26. SENSITIVITY OF MODIFIED BIEL-MAZE TASK, COMPARED WITH Y-MAZE TASK, TO MEASURE SPATIAL LEARNING AND MEMORY DEFICITS OF ETHANOL TERATOGENICITY IN THE GUINEA PIG

Dobson CC, Mongillo DL, Poklewska-Koziell M, Winterborn A, Brien JF, Reynolds JN.
Department of Biomedical and Molecular Sciences, Pharmacology and Toxicology Graduate Program, Queen's University, Kingston, ON K7L 3N6, Canada.

ABSTRACT
Ethanol consumption during pregnancy can produce a variety of teratogenic effects in offspring, termed Fetal Alcohol Spectrum Disorders (FASD). The most debilitating and permanent consequence of chronic prenatal ethanol exposure (CPEE) is neurobehavioral teratogenicity, which often manifests as cognitive and behavioral impairments, including deficits in spatial learning and memory. This study tested the hypothesis that a modified dry-land version of the multi-choice Biel-maze task is more sensitive than the rewarded-alternation Y-maze task for the determination of spatial learning and memory deficits of ethanol teratogenicity. Pregnant guinea pigs received ethanol (4g/kg maternal body weight/day) or isocaloric-sucrose/pair-feeding (control) for 5days/week throughout gestation. CPEE resulted in ethanol neurobehavioral teratogenicity in offspring, as demonstrated by increased spontaneous locomotor activity at postnatal day (PD) 10 and decreased brain weight at euthanasia (PD 150-200). On PD 21, offspring were randomly assigned to one of two tasks to assess spatial learning and memory performance: a dry-land version of the Biel maze or a rewarded-alternation Y-maze. Animals were habituated to the environment of their assigned task and performance of each CPEE or control offspring was measured. In the modified Biel maze, CPEE and control offspring were not different for percent completed trials or time to complete a trial. However, CPEE offspring made more errors (reversals and entering dead ends) in the Biel maze, demonstrating impaired spatial learning and memory. In contrast, CPEE offspring did not have impaired performance of the rewarded-alternation Y-maze

task. Therefore, the modified dry-land version of the Biel-maze task, which measures cognitive performance using a complex multi-choice design, is more sensitive in demonstrating CPEE-induced spatial learning and memory deficits compared with a simple, rewarded-alternation Y-maze task.

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http://www.ncbi.nlm.nih.gov/pubmed/22562040

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PubMed, Acta Paediatr. 2012 Apr 26. doi: 10.1111/j.1651-2227.2012.02700.x.

27. MOTION PERCEPTION IN CHILDREN WITH FETAL ALCOHOL SYNDROME

Gummel K, Ygge J, Benassi M, Bolzani R.
Dept. of Clinical Neuroscience, Karolinska Institutet, Stockholm, Sweden Pediatric Medical Academy, St-Petersburg, Russia Dept. of Psychology, University of Bologna, Italy.

ABSTRACT
Aim: Evaluation of visual magnocellular pathway by a coherent motion perception test in children with Fetal alcohol syndrome (FAS).

Methods: 89 children (49 with verified FAS and 40 without FAS) aged from 10 to16 years were included into the study. Both the study and the control group were children living in orphanages. A coherent motion perception test was used. The test consisted of 150 white moving dots on a black background presented in different signal-to-noise ratio conditions. The task was direction detection of the coherently moving dots whose percentage decreased at each step.

Results: A significant difference between the two groups was found ($p = 0.018$). Children with FAS had lower coherent motion perception ability in all the signal-to-noise ratio conditions. A significant difference between difficulty levels ($p < 0.001$) was found for all subjects in both groups - decreasing the stimulus signal-to-noise level decreased the motion perception score. In both groups the motion perception score differed for vertical and horizontal stimuli ($p = 0.003$) with better performance with vertical stimuli.

Conclusion: Impaired motion perception in FAS children could be indicative of a dorsal stream developmental dysfunction resulting from alcohol brain damage.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22536909

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PubMed, Evid Based Nurs. 2012 Apr 25.

28. THE ASSOCIATION BETWEEN PRENATAL ALCOHOL EXPOSURE, FETAL GROWTH AND PRETERM BIRTH: EVIDENCE FROM A SYSTEMATIC REVIEW AND META-ANALYSES

O'Leary CM.
Centre for Population Health Research, Curtin Health Innovation Research Institute, Curtin University, Perth, Australia.

No abstract available

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http://www.ncbi.nlm.nih.gov/pubmed/22535581

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PubMed, Physiol Genomics. 2012 Apr 24.

29. MULTIPLEXED DIGITAL QUANTIFICATION OF BINGE-LIKE ALCOHOL-MEDIATED ALTERATIONS IN MATERNAL UTERINE ANGIOGENIC MRNA TRANSCRIPTOME

Ramadoss J, Magness RR.
University of Texas Medical Branch.

ABSTRACT

Genomic studies on Fetal Alcohol Spectrum Disorders (FASD) have either utilized genome-wide microarrays/bioinformatics or targeted real time PCR (RT-PCR). We utilized herein for the first time a novel digital approach with high throughput as well as the capability to focus on one physiologic system. The aim of the present study was to investigate alcohol-induced alterations in uterine angiogenesis-related mRNA abundance using digital mRNA technology. Four biological and three technical replicates of uterine arterial endothelial cells from third trimester ewes were Fluorescence Activated Cell sorted, validated, and treated without or with binge-like alcohol. A capture probe covalently bound to an oligonucleotide containing biotin and a color coded reporter probe were designed for 85 angiogenesis-related genes and analyzed using the Nanostring nCounter system. 20 genes were down-regulated (↓) and two up-regulated (↑), including angiogenic growth factors/receptors (↓placental growth factor), adhesion molecules (↓angiopoietin-like-3; ↓collagen-18A1; ↓endoglin), proteases/matrix proteins/inhibitors (↓alanyl aminopeptidase; ↓collagen-4A3; ↓heperenase; ↓plasminogen, ↑plasminogen activator urokinase; ↓platelet factor-4; ↓plexin domain containing-1; ↓Tissue Inhibitor of Metalloproteinases-3), transcription/signaling molecules (↓Heart and Neural Crest Derivatives-2; ↓DNA-binding protein inhibitor; ↓NOTCH-4; ↓ribosomal protein-L13a1; ↓ribosomal protein large-P1), cytokines/chemokines (↓interleukin-1B), and miscellaneous growth factors (↓leptin; ↓platelet-derived growth factor-α; ↓Transforming Growth Factor (TGF-α; ↑TGF-βreceptor-1). These novel data show significant detrimental alcohol effects on genes controlling angiogenesis supporting a mechanistic role for abnormal uteroplacental vascular development in FASD. The tripartite digital gene expression system is therefore a valuable tool to answer many additional questions about FASD from both mechanistic as well as ameliorative perspectives.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22535877

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PubMed, Alcohol Clin Exp Res. 2012 Apr 24. doi: 10.1111/j.1530-0277.2012.01757.x.

30. GENE EXPRESSION CHANGES IN C57BL/6J AND DBA/2J MICE FOLLOWING PRENATAL ALCOHOL EXPOSURE

Downing C, Flink S, Florez-McClure ML, Johnson TE, Tabakoff B, Kechris KJ.
Department of Biomedical and Pharmaceutical Sciences , College of Pharmacy, Idaho State University, Pocatello, Idaho.

ABSTRACT

Background: Prenatal alcohol exposure can result in fetal alcohol spectrum disorders (FASD). Not all women who consume alcohol during pregnancy have children with FASD and studies have shown that genetic factors can play a role in ethanol teratogenesis. We examined gene expression in embryos and placentae from C57BL/6J (B6) and DBA/2J (D2) mice following prenatal alcohol exposure. B6 fetuses are susceptible to morphological malformations following prenatal alcohol exposure while D2 are relatively resistant.

Methods: Male and female B6 and D2 mice were mated for 2 hours in the morning, producing 4 embryonic genotypes: true-bred B6B6 and D2D2, and reciprocal B6D2 and D2B6. On gestational day 9, dams were intubated with 5.8 g/kg ethanol, an isocaloric amount of maltose dextrin, or

nothing. Four hours later, dams were sacrificed and embryos and placentae were harvested. RNA was extracted, labeled and hybridized to Affymetrix Mouse Genome 430 v2 microarray chips. Data were normalized, subjected to analysis of variance and tested for enrichment of gene ontology molecular function and biological process using the Database for Annotation, Visualization and Integrated Discovery (DAVID).

Results: Several gene classes were differentially expressed in B6 and D2 regardless of treatment, including genes involved in polysaccharide binding and mitosis. Prenatal alcohol exposure altered expression of a subset of genes, including genes involved in methylation, chromatin remodeling, protein synthesis, and mRNA splicing. Very few genes were differentially expressed between maltose-exposed tissues and tissues that received nothing, so we combined these groups for comparisons with ethanol. While we observed many expression changes specific to B6 following prenatal alcohol exposure, none were specific for D2. Gene classes up- or down-regulated in B6 following prenatal alcohol exposure included genes involved in mRNA splicing, transcription, and translation.

Conclusions: Our study identified several classes of genes with altered expression following prenatal alcohol exposure, including many specific for B6, a strain susceptible to ethanol teratogenesis. Lack of strain specific effects in D2 suggests there are few gene expression changes that confer resistance. Future studies will begin to analyze functional significance of the expression changes.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22530671

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PubMed, Alcohol Clin Exp Res. 2012 Apr 23. doi: 10.1111/j.1530-0277.2012.01736.x.

31. ENFORCED PAX6 EXPRESSION RESCUES ALCOHOL-INDUCED DEFECTS OF NEURONAL DIFFERENTIATION IN CULTURES OF HUMAN CORTICAL PROGENITOR CELLS

Mo Z, Milivojevic V, Zecevic N.
Department of Neuroscience, University of Connecticut Health Center, Farmington, Connecticut; Department of Pathology, Conemaugh Memorial Medical Center, Johnstown, Pennsylvania.

ABSTRACT

Background: Alcohol is the most widely consumed substance of abuse, and its use during pregnancy can lead to serious disorders of brain development. The precise molecular action of alcohol on human brain development, however, is still unknown. We previously enriched multipotent progenitor cells, radial glia (RG) cells, from human fetal forebrain and demonstrated that they express transcription factor Pax6 that is necessary for their neurogenic fate.

Methods: Enriched human fetal RG cells were maintained in vitro as either control or Pax6-expressing retrovirus infected cells. Cultures were treated with increasing doses of alcohol to evaluate Pax6 expression, proliferation, and differentiation of RG cells by immunocytochemistry, Western blot, and RT-PCR methods.

Results: In vitro treatment with alcohol reduced the expression of transcription factor Pax6 and proliferation of RG cells, which decreased neurogenesis. Consistent with this finding, the overexpression of Pax6 in RG cells under alcohol treatment rescued cell proliferation and restored the generation of neurons. In contrast to this effect on neurogenesis, the overexpression of Pax6 inhibits the generation of astroglia regardless of alcohol treatment, implying lineage-specific effects.

Conclusions: These findings suggest that the effect of alcohol on human neurogenesis is partially due to the reduced expression of transcription factor Pax6 in RG cells.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22524987

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PubMed, J Matern Fetal Neonatal Med. 2012 Apr 23.

32. FETAL AND NEONATAL OUTCOMES IN WOMEN REPORTING INGESTION OF LOW OR VERY LOW ALCOHOL INTAKE DURING PREGNANCY

Han JY, Choi JS, Ahn HK, Kim MH, Chung JH, Ryu HM, Kim MY, Yang JH, Nava-Ocampo AA.
Korean Motherisk Program, Department of Obstetrics and Gynaecology, Cheil Hospital and Women's Healthcare Centre, Kwandong University School of Medicine, Seoul, South Korea.

ABSTRACT
Abstract Objective: This study aimed to assess the pregnancy outcomes of women who reported social intake of low or very low alcohol levels during pregnancy.

Methods: Obstetric and foetal outcomes were assessed in a prospective cohort of 1,667 pregnant women who reported low or very low alcohol consumption during pregnancy (cases) and 1,840 alcohol abstainer women (controls).

Results: Among cases, alcohol consumption occurred during the first 4.4 (median) weeks of pregnancy, with a median ingestion of 1.0 (0.01-6.0) drinks/week, equivalent to 7.6 (0.09-47.5) g/week. Cigarette smoking was reported approximately 4 times more often in the exposed group than in the controls (P <0.001). Pregnancy outcomes were similar between groups. There were 37 (2.4%) babies born with malformations in the exposed group and 41 (2.4%) in the control group (P= 0.9).

Conclusions: low-to-very low levels of alcohol ingestion during pregnancy do not appear to be associated with adverse maternal or foetal outcomes.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22524896

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PubMed, J Popul Ther Clin Pharmacol. 2012;19(1):e99-e110. Epub 2012 Apr 19.

33. SOCIAL PROBLEM SOLVING IN CHILDREN WITH FETAL ALCOHOL SPECTRUM DISORDERS

Stevens SA, Majors D, Rovet J, Koren G, Fantus E, Nulman I, Desrocher M.

ABSTRACT
Background: Children with fetal alcohol spectrum disorders (FASD) show impairments in social functioning. However, the factors underlying these impairments are poorly understood. Recent evidence has shown that social problem solving is a critical component of effective social functioning

Objectives: The present study sought to examine social information processing as one potential factor contributing to social skills and behavior impairments observed in children with FASD.

Methods: Forty-three children, 20 with FASD (mean age 12.6 years) and 23 typically developing controls (TDC; mean age 12.5 years) were studied. Social information processing was investigated using the Children's Interpersonal Problem Solving task (ChIPS; Shure and Spivack, 1985), which

assesses problem solving in response to social dilemmas.ResultsChildren with FASD produced fewer relevant responses than TDC and their responses belonged to a fewer number of categories.

Conclusion: Children with FASD show reduced ability in generating solutions for social dilemmas. By understanding this weakness, which may partially explain the social skill deficiencies in FASD, targeted therapies may be designed to improve social functioning following prenatal alcohol exposure.

Link to the Article,
http://www.ncbi.nlm.nih.gov/pubmed/22535836

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PubMed, Brain Res. 2012 Jun 6;1458:18-33. Epub 2012 Apr 17.

34. LONG-TERM ALTERATIONS TO THE BRAIN TRANSCRIPTOME IN A MATERNAL VOLUNTARY CONSUMPTION MODEL OF FETAL ALCOHOL SPECTRUM DISORDERS

Kleiber ML, Laufer BI, Wright E, Diehl EJ, Singh SM.
Molecular Genetics Unit, Department of Biology, University of Western Ontario, London, Ontario, Canada N6A 5B7.

ABSTRACT

Many women continue to consume low to moderate quantities of alcohol during pregnancy, which can result in the variable neurobehavioural effects in the absence of physiological abnormalities that characterize fetal alcohol spectrum disorders (FASD). Previously, we reported that a mouse model for FASD based on voluntary maternal ethanol consumption throughout gestation resulted in offspring that showed mild developmental delay, anxiety-related traits, and deficits in spatial learning. Here, we extend this model by evaluating the gene expression changes that occur in the adult brain of C57BL/6J mice prenatally exposed to ethanol via maternal preference drinking. The results of two independent expression array experiments indicate that ethanol induces subtle but consistent changes to global gene expression. Gene enrichment analysis showed over-represented gene ontology classifications of cellular, embryonic, and nervous system development. Molecular network analysis supported these classifications, with significant networks related to cellular and tissue development, free radical scavenging, and small molecule metabolism. Further, a number of genes identified have previously been implicated in FASD-relevant neurobehavioural phenotypes such as cognitive function (Ache, Bcl2, Cul4b, Dkc1, Ebp, Lcat, Nsdh1, Sstr3), anxiety (Bcl2), attention deficit hyperactivity disorder (Nsdh1), and mood disorders (Bcl2, Otx2, Sstr3). The results suggest a complex residual "footprint" of neurodevelopmental ethanol exposure that may provide a new perspective for identifying mechanisms that underlie the life-long persistence of FASD-related cognitive and behavioural alterations, including potential targets for treatment.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22560501

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PubMed, Alcohol Clin Exp Res. 2012 Apr 17. doi: 10.1111/j.1530-0277.2012.01784.x.

35. CHOLINE SUPPLEMENTATION AND DNA METHYLATION IN THE HIPPOCAMPUS AND PREFRONTAL CORTEX OF RATS EXPOSED TO ALCOHOL DURING DEVELOPMENT

Otero NK, Thomas JD, Saski CA, Xia X, Kelly SJ.
Department of Psychology, University of South Carolina, Columbia, South Carolina.

ABSTRACT

Background: Some of the most frequent deficits seen in children with fetal alcohol spectrum

disorders (FASD) and in animal models of FASD are spatial memory impairments and impaired executive functioning, which are likely related to alcohol-induced alterations of the hippocampus and prefrontal cortex (PFC), respectively. Choline, a nutrient supplement, has been shown in a rat model to ameliorate some of alcohol's teratogenic effects, and this effect may be mediated through choline's effects on DNA methylation.

Methods: Alcohol was given by intragastric intubation to rat pups during the neonatal period (postnatal days 2 to 10) (ET group), which is equivalent to the third trimester in humans and a period of heightened vulnerability of the brain to alcohol exposure. Control groups included an intubated control group given the intubation procedure without alcohol (IC) and a nontreated control group (NC). Choline or saline was administered subcutaneously to each subject from postnatal days 2 to 20. On postnatal day 21, the brains of the subjects were removed and assayed for global DNA methylation patterning as measured by chemiluminescence using the cpGlobal assay in both the hippocampal region and PFC.

Results: Alcohol exposure caused hypermethylation in the hippocampus and PFC, which was significantly reduced after choline supplementation. In contrast, control animals showed increases in DNA methylation in both regions after choline supplementation, suggesting that choline supplementation has different effects depending upon the initial state of the brain.

Conclusions: This study is the first to show changes in global DNA methylation of the hippocampal region and PFC after neonatal alcohol exposure. Choline supplementation impacts global DNA methylation in these 2 brain regions in alcohol-exposed and control animals in a differential manner. The current findings suggest that both alcohol and choline have substantial impact on the epigenome in the PFC and hippocampus, and future studies will be needed to describe which gene families are impacted in such a way that function of the nervous system is changed.

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http://www.ncbi.nlm.nih.gov/pubmed/22509990

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Wiley Online Library - Alcoholism: Clinical and Experimental Research
Article first published online: 6 APR 2012
DOI: 10.1111/j.1530-0277.2012.01783.x

36. ALCOHOL CONSUMPTION AMONG PREGNANT WOMEN IN A SWEDISH SAMPLE AND ITS EFFECTS ON THE NEWBORN OUTCOMES

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1 Department of Neuroscience, Unit of Pharmacology, Uppsala University, Uppsala, Sweden
2 Center for Clinical Research Västerås, Västerås Central Hospital, Uppsala University, Uppsala, Sweden
3 Department of Women's and Children's Health, Unit of Obstetrics and Gynecology, Uppsala University, Uppsala, Sweden
4 Department of Laboratory Medicine, Karolinska Institutet and Karolinska University Laboratory, Stockholm, Sweden
5 Department of Neuroscience, Unit of Child and Adolescent Psychiatry, Uppsala University, Uppsala, Sweden

ABSTRACT

Background: Little is known about the effects of low levels of maternal alcohol intake on the neuropsychological development of the child. This study is part of an ongoing investigation on maternal drinking and presents data on demographic variables, maternal alcohol use, and birth

outcomes from that study.

Methods: The sample comprised 2,264 women from a Swedish antenatal clinic. Retrospective self-report data were collected on alcohol consumption before and during pregnancy, using the Alcohol Use Disorders Identification Test (AUDIT), and on nicotine use. Specific alcohol biomarkers for excessive drinking, carbohydrate-deficient transferrin (CDT) in serum and phosphatidylethanol (PEth) in whole blood, were determined during mid-pregnancy in a subsample of the women. Data on labor and early characteristics of the child were also assessed.

Results: Before pregnancy, 89% of the women regularly consumed alcohol and 49% reported occasional or frequent binge drinking. Nicotine was used by 15% before and by 5% during pregnancy. During pregnancy, 12% continued using alcohol and 5% also admitted binge drinking. However, all alcohol biomarker values were below the reporting limits (CDT ≤ 1.7% disialotransferrin; total PEth < 0.1 μmol/L). Self-reported drinking during pregnancy was associated with a higher AUDIT score before pregnancy, nicotine use at the time of the first prenatal visit, older age, and previous legal abortions.

Conclusions: The AUDIT questionnaire and 2 specific alcohol biomarkers were used in routine maternity care to collect information about drinking during pregnancy and thereby to identify children at risk for alcohol-related complications. While the AUDIT results suggested that a significant number of women continued using alcohol during pregnancy, implying a risk for fetal disorders, the biomarkers showed negative test values thus indicating only modest drinking levels.

Read Full Article,
http://onlinelibrary.wiley.com/doi/10.1111/j.1530-0277.2012.01783.x/abstract

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PubMed, Am J Addict. 2012 May;21(3):199-201.
doi: 10.1111/j.1521-0391.2012.00229.x.
Epub 2012 Apr 6.

37. ACCEPTANCE OF NALTREXONE BY PREGNANT WOMEN ENROLLED IN COMPREHENSIVE DRUG ADDICTION TREATMENT: AN INITIAL SURVEY

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RTI International, Research Triangle Park, North Carolina The Departments of Psychiatry and Behavioral Sciences and Obstetrics and Gynecology, The Johns Hopkins University School of Medicine, Baltimore, Maryland.

ABSTRACT

This paper reports the results of an initial survey regarding the potential interest in naltrexone treatment by pregnant women enrolled in comprehensive treatment for substance use disorders. Pregnant women (N = 112) were asked about their interest in taking either an oral or long-acting injectable medication that would stop heroin and/or alcohol use, leave them "clear-headed," and without neonatal withdrawal.
Results indicate strong interest among pregnant women in antagonist treatment, in either form, with clear interest in learning more about naltrexone. Findings lend support for patient's acceptance of a clinical trial of antagonist treatment in this population.

Link to the Article,
http://www.ncbi.nlm.nih.gov/pubmed/22494221

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Wiley Online Library - Acta Paediatrica
Article first published online: 4 APR 2012
DOI: 10.1111/j.1651-2227.2012.02670.x

38. ALCOHOL AND SUBSTANCE ABUSE IDENTIFIED DURING PREGNANCY: MATERNAL MORBIDITY, CHILD MORBIDITY AND WELFARE INTERVENTIONS

Taisto Sarkola[1], Mika Gissler[2], Hanna Kahila[3], Ilona Autti-Rämö[4], Erja Halmesmäki[3]
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2 .THL National Institute for Health and Welfare, Helsinki, Finland and Nordic School of Public Health, Gothenburg, Sweden
3 .Department of Obstetrics and Gynecology, Helsinki University Central Hospital, Helsinki, Finland
4 .The Social Insurance Institution (SII), Research Department, Helsinki, Finland

ABSTRACT

Aim: To study the relations between postnatal maternal morbidity, child morbidity and welfare interventions in families with prenatal alcohol or substance abuse.

Methods: A register-based longitudinal retrospective cohort study. The exposed cohort included 638 children born to 524 women followed-up during pregnancy for alcohol or substance abuse 1992–2001. Non-exposed children (n = 1914) born to control women were matched for maternal age, parity, number of foetuses, month of birth and delivery hospital of the index child. Perinatal and follow-up data of both cohorts were collected from national registers until 2007.

Results: Postnatal maternal abuse-related healthcare utilization and use of medication were associated with child out-of-home care. Significant differences were in particular observed in the categories of maternal mental and behavioural disorders caused by psychoactive substance use as well as injury and poisoning. Maternal inpatient care for mental and behavioural disorders peaked at the time of child out-of-home care. Maternal abuse-related healthcare utilization was associated with early child healthcare utilization and use of medication for mental and behavioural disorders. These associations were largely explained by the association with child out-of-home care.

Conclusions: Postnatal maternal abuse-related morbidity is associated with significant early child morbidity, use of medication and timing of out-of-home care.

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http://onlinelibrary.wiley.com/doi/10.1111/j.1651-2227.2012.02670.x/abstract

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Wiley Online Library, Aust J Rural Health. 2012 Apr;20(2):99. doi: 10.1111/j.1440-1584.2012.01255.x.

39. MORE DATA NEEDED ON FOETAL ALCOHOL SPECTRUM DISORDER

ABSTRACT

No Abstract available

Read Full Article,
http://onlinelibrary.wiley.com/doi/10.1111/j.1440-1584.2012.01255.x/abstract

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Oxford Journals - Alcohol and Alcoholism (2012), doi: 10.1093/alcalc/ags051
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40. EVALUATION OF THE IMPACT OF THE IMAGE USED IN A COMMUNICATION CAMPAIGN TO RAISE AWARENESS ABOUT THE EFFECTS OF ALCOHOL USE DURING PREGNANCY

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3 Addiction Department, Local Health Authority of Treviso, Treviso,Veneto Region, Italy
4 Department of Innovation, Development and Planning, Local Health Authority of Treviso, Treviso,Veneto Region, Italy
5 University of Udine, Udine, Italy
6 University of Trieste, Trieste, Italy

ABSTRACT
Aims: To assess the impact of the advertising image used in the health communication campaign 'Mummy Drinks Baby Drinks', aimed to raise awareness about the effects of drinking alcohol during pregnancy in the childbearing-aged population of the Local Health Authority of Treviso (Italy). The image depicted a foetus inside a glass of a local alcoholic drink.

Methods: A survey using a semi-structured self-reported questionnaire was carried out. The questionnaire was administered to a consecutive series of 690 parents or caregivers who accompanied children aged 0–2 years in the vaccination clinics of the Local Health Unit, during a 30-day period 1 year after the start of the campaign. The questionnaire measured the level of exposure to the image, emotional reactions and awareness of the health messages conveyed by the image.

Results: Overall, 84% of the respondents said that they remembered the image. Almost all (93%) recalled the warning message and 53% recalled the health behaviours suggested by the campaign. The image generally seemed to arouse a high emotive impact: 38% indicated distress and 40% liking as a general opinion, while ~50% expressed distress emotions and 13% were pleasantly affected when reflecting on the feelings evoked. We did not find unequivocal relationships between the level and kind of emotional reactions and the recalling of the health behaviours.

Conclusion: The image obtained a high level of visibility. It was effective in spreading the health message conveyed by the campaign, regardless of the level and kind of emotive impact evoked.

Read Full Article,
http://alcalc.oxfordjournals.org/content/early/2012/05/02/alcalc.ags051.full

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PubMed, Dev Neurosci. 2012 Mar 29. [Epub ahead of print]

41. FRONTOSTRIATAL CONNECTIVITY IN CHILDREN DURING WORKING MEMORY AND THE EFFECTS OF PRENATAL METHAMPHETAMINE, ALCOHOL, AND POLYDRUG EXPOSURE

Roussotte FF, Rudie JD, Smith L, O'Connor MJ, Bookheimer SY, Narr KL, Sowell ER.
Developmental Cognitive Neuroimaging Laboratory, Children's Hospital Los Angeles and Department of Pediatrics, University of Southern California, Los Angeles, Calif., USA.

ABSTRACT
Various abnormalities in frontal and striatal regions have been reported in children with prenatal

alcohol and/or methamphetamine exposure. In a recent fMRI study, we observed a correlation between accuracy on a working-memory task and functional activation in the putamen in children with prenatal methamphetamine and polydrug exposure. Because the putamen is part of the corticostriatal motor loop whereas the caudate is involved in the executive loop, we hypothesized that a loss of segregation between distinct corticostriatal networks may occur in these participants. The current study was designed to test this hypothesis using functional connectivity MRI. We examined 50 children ranging in age from 7 to 15, including 19 with prenatal methamphetamine exposure (15 of whom had concomitant prenatal alcohol exposure), 13 with prenatal exposure to alcohol but not methamphetamine, and 18 unexposed controls. We measured the coupling between blood oxygenation level dependent (BOLD) fluctuations during a working-memory task in four striatal seed regions and those in the rest of the brain. We found that the putamen seeds showed increased connectivity with frontal brain regions involved in executive functions while the caudate seeds showed decreased connectivity with some of these regions in both groups of exposed subjects compared to controls.

These findings suggest that localized brain abnormalities resulting from prenatal exposure to alcohol and/or methamphetamine lead to a partial rewiring of corticostriatal networks. These results represent important progress in the field, and could have substantial clinical significance in helping devise more targeted treatments and remediation strategies designed to better serve the needs of this population.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22472800

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Unbound Medline, Alcohol Clin Exp Res 2012 Mar 28.

42. DELAYS IN AUDITORY PROCESSING IDENTIFIED IN PRESCHOOL CHILDREN WITH FASD

Stephen JM, Kodituwakku PW, Kodituwakku EL, Romero L, Peters AM, Sharadamma NM, Caprihan A, Coffman BA

ABSTRACT
Background: Both sensory and cognitive deficits have been associated with prenatal exposure to alcohol; however, very few studies have focused on sensory deficits in preschool-aged children. As sensory skills develop early, characterization of sensory deficits using novel imaging methods may reveal important neural markers of prenatal alcohol exposure.

Methods: Participants in this study were 10 children with a fetal alcohol spectrum disorder (FASD) and 15 healthy control (HC) children aged 3 to 6 years. All participants had normal hearing as determined by clinical screens. We measured their neurophysiological responses to auditory stimuli (1,000 Hz, 72 dB tone) using magnetoencephalography (MEG). We used a multidipole spatio-temporal modeling technique to identify the location and timecourse of cortical activity in response to the auditory tones. The timing and amplitude of the left and right superior temporal gyrus sources associated with activation of left and right primary/secondary auditory cortices were compared across groups.

Results: There was a significant delay in M100 and M200 latencies for the FASD children relative to the HC children ($p = 0.01$), when including age as a covariate. The within-subjects effect of hemisphere was not significant. A comparable delay in M100 and M200 latencies was observed in children across the FASD subtypes.

Conclusions: Auditory delay revealed by MEG in children with FASDs may prove to be a useful neural marker of information processing difficulties in young children with prenatal alcohol exposure. The fact that delayed auditory responses were observed across the FASD spectrum

suggests that it may be a sensitive measure of alcohol-induced brain damage. Therefore, this measure in conjunction with other clinical tools may prove useful for early identification of alcohol affected children, particularly those without dysmorphia.

Read Full Article,
http://www.unboundmedicine.com/medline/ebm/record/22458372/abstract/Delays_in_Auditory_Processing_Identified_in_Preschool_Children_with_FASD_

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Wiley Online Library - Alcoholism: Clinical and Experimental Research
Article first published online: 28 MAR 2012
DOI: 10.1111/j.1530-0277.2012.01793.x

43. OPPOSING ACTIONS OF ETHANOL AND NICOTINE ON MICRORNAS ARE MEDIATED BY NICOTINIC ACETYLCHOLINE RECEPTORS IN FETAL CEREBRAL CORTICAL–DERIVED NEURAL PROGENITOR CELLS

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Department of Neuroscience and Experimental Therapeutics , College of Medicine, Texas A&M Health Science Centre, Bryan, Texas.

ABSTRACT

Background: Ethanol (EtOH) and nicotine are often co-abused. However, their combined effects on fetal neural development, particularly on fetal neural stem cells (NSCs), which generate most neurons of the adult brain during the second trimester of pregnancy, are poorly understood. We previously showed that EtOH influenced NSC maturation in part, by suppressing the expression of specific microRNAs (miRNAs). Here, we tested in fetal NSCs the extent to which EtOH and nicotine coregulated known EtOH-sensitive (miR-9, miR-21, miR-153, and miR-335), a nicotine-sensitive miRNA (miR-140-3p), and mRNAs for nicotinic acetylcholine receptor (nAChR) subunits. Additionally, we tested the extent to which these effects were nAChR dependent.

Methods: Gestational day 12.5 mouse fetal murine cerebral cortical–derived neurosphere cultures were exposed to EtOH, nicotine, and mecamylamine, a noncompetitive nAChR antagonist, individually or in combination, for short (24 hour) and long (5 day) periods, to mimic exposure during the in vivo period of neurogenesis. Levels of miRNAs, miRNA-regulated transcripts, and nAChR subunit mRNAs were assessed by quantitative reverse transcription polymerase chain reaction.

Results: EtOH suppressed the expression of known EtOH-sensitive miRNAs and miR-140-3p, while nicotine at concentrations attained by cigarette smokers induced a dose-related increase in these miRNAs. Nicotine's effect was blocked by EtOH and by mecamylamine. Finally, EtOH decreased the expression of nAChR subunit mRNAs and, like mecamylamine, prevented the nicotine-associated increase in α4 and β2 nAChR transcripts.

Conclusions: EtOH and nicotine exert mutually antagonistic, nAChR-mediated effects on teratogen-sensitive miRNAs in fetal NSCs. These data suggest that concurrent exposure to EtOH and nicotine disrupts miRNA regulatory networks that are important for NSC maturation.

Read Full Article,
http://onlinelibrary.wiley.com/doi/10.1111/j.1530-0277.2012.01793.x/abstract

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Wiley Online Library - Human Brain Mapping
Article first published online: 25 MAR 2012
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44. DIFFERENCES IN CORTICO-STRIATAL-CEREBELLAR ACTIVATION DURING WORKING MEMORY IN SYNDROMAL AND NONSYNDROMAL CHILDREN WITH PRENATAL ALCOHOL EXPOSURE

Vaibhav A. Diwadkar[1],*, Ernesta M. Meintjes[2],[3], Dhruman Goradia[1], Neil C. Dodge[1], Christopher Warton[3], Christopher D. Molteno[4], Sandra W. Jacobson[1],[3],[4], Joseph L. Jacobson[1],[3],[4],*

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ABSTRACT
Although children with heavy prenatal alcohol exposure may exhibit the distinctive facial dysmorphology seen in full or partial fetal alcohol syndrome (FAS/PFAS), many lack that dysmorphology. This study examined the functional organization of working memory in the brain in three groups of children—those meeting diagnostic criteria for FAS or PFAS, heavily exposed (HE) nonsyndromal children, and healthy controls. A verbal n-back task (1-back and 0-back) was administered to 47 children (17 with FAS/PFAS, 13 HE, and 17 controls) during fMRI. Intra-group one-sample t-tests were used to identify activity regions of interest central to verbal working memory including the dorsal prefrontal cortex (dPFC), inferior frontal gyrus, caudate/putamen, parietal cortex, and cerebellar Crus I/lobule VI and lobule VIIB-IX. Whereas groups did not differ in task sensitivity, fMRI analyses suggested different patterns of sub-network recruitment across groups. Controls primarily recruited left inferior frontal gyrus (Broca's area). By contrast, HE primarily recruited an extensive set of fronto-striatal regions, including left dPFC and left caudate, and the FAS/PFAS group relied primarily on two cerebellar subregions and parietal cortex. This study is, to our knowledge, the first to demonstrate differential recruitment of critical brain regions that subserve basic function in children with different fetal alcohol spectrum disorders compared to controls. The distinct activation patterns seen in the two exposed groups may be related to substantial differences in alcohol dose/occasion to which these groups were exposed in utero.

Read Full Article,
http://onlinelibrary.wiley.com/doi/10.1002/hbm.22042/abstract

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PubMed, Fetal Pediatr Pathol. 2012 Mar 23

45. FETAL CENTRAL NERVOUS SYSTEM DEVELOPMENT AND ALCOHOL - THE EVIDENCE SO FAR

Ahmed-Landeryou MJ.
London South Bank university, Occupational Therapy, Allied Health Sciences , Southwark, London, UK.

ABSTRACT
Currently in the UK, there is no absolute guidance about alcohol consumption in pregnancy. The guidance for drinking during pregnancy is one or two units of alcohol one or two times weekly, but conservative advice is to abstain as a cautionary measure [1]. Despite the lack of consensus about

the safe levels of alcohol consumption in pregnancy, there is increasing evidence of the impact of alcohol on the developing central nervous system. This article explores the evidence regarding alcohol consumption and its effects on the developing fetal central nervous system.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22443150

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Alcohol. 2012 May;46(3):277-83. Epub 2012 Mar 22.

46. EFFECTS OF BINGE DRINKING ON INFANT GROWTH AND DEVELOPMENT IN AN INUIT SAMPLE

Fraser SL, Muckle G, Abdous BB, Jacobson JL, Jacobson SW.
Centre de Recherche du Centre Hospitalier, Université Laval, Sainte-Foy, QC, Canada G1K 7P4.

ABSTRACT
Prenatal exposure to an average of 0.5 oz absolute alcohol per day (the equivalent of 7 standard drinks per week) during pregnancy has been found to be associated with numerous adverse effects on pre- and postnatal development. In the animal model, concentrated alcohol exposure has been found to lead to more adverse effects than exposure to the same total quantity of alcohol ingested in smaller doses over a longer period of time. The primary aim of this study is to determine whether, in a population where binge drinking is common but total alcohol consumption across pregnancy is low, prenatal exposure to alcohol is associated with effects on prenatal growth, visual acuity and cognitive development during infancy. The second aim is to determine which of several indicators of alcohol consumption best predicts pre- and postnatal outcomes. Data were collected from 216 Inuit women and their infants living in Nunavik, the northern region of Québec. Maternal interviews were conducted during mid-pregnancy and at 1 and 6 months postpartum. Birth weight, length, and head circumference were assessed at delivery. Visual acuity and cognitive development were assessed at 6 months of age. In this population in which infrequent heavy episodic drinking is common, even occasional binge exposure was associated with reduced prenatal growth and poorer visual acuity at 6 months of age. A simple dichotomous measure of binge drinking during pregnancy provided the best predictor of fetal growth and 6-month acuity. The population studied here is unusual in terms of its pattern of binge alcohol consumption. To our knowledge, this is the first study to observe effects of binge drinking during pregnancy on infant growth and development in a sample where the average daily alcohol intake is low (<0.5 ounces).

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22444956

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Alcohol, Received 26 February 2011; received in revised form 14 November 2011; accepted 28 November 2011. published online 22 March 2012

47. RECOGNIZED SPONTANEOUS ABORTION IN MID-PREGNANCY AND PATTERNS OF PREGNANCY ALCOHOL USE

Lisa M. Chiodo, Beth A. Bailey, Robert J. Sokol, James Janisse, Virginia Delaney-Black, John H. Hannigan

ABSTRACT
Alcohol consumption during pregnancy is one potential risk factor for spontaneous abortion (SAb). Prior research suggested that heavy drinking during pregnancy was associated with significantly increased rates of SAb, but results for lower levels of drinking have been inconsistent. We

examined the association between different levels and patterns of prenatal alcohol consumption and SAb in a high-risk inner-city sample. We hypothesized that higher levels, binge patterns, and more frequent drinking would be associated with increased rates of SAb. The quantity and frequency of self-reported peri-conceptional and repeated in-pregnancy maternal drinking volumes per beverage type were assessed with semi-structured interviews in a prospective subsample of 302 African-American mothers. Relations between various measures of prenatal alcohol exposure and SAb were assessed using logistic regression. After controlling for various potential confounders, there was a significant positive relation between average absolute alcohol use per day across pregnancy and SAb. Greater frequency of drinking episodes also predicted SAb: an average of even one day of drinking per week across pregnancy was associated with an increase in the incidence of SAb. However, contrary to our hypothesis, neither the amount of alcohol drunk per drinking day nor a measure of binge drinking was significantly related to SAb after controlling for confounders. Differences in when women who drank at risk levels initiated antenatal care may have under-estimated the impact of alcohol on SAb in this low-SES urban African-American sample. Some drinking measures averaged across pregnancy may have under-estimated consumption and overestimated risk of SAb, but other risk drinking measures that avoid this limitation show similar relations to SAb. Identifying fetal risk drinking in pregnant women is critical to increasing the effectiveness of interventions that reduce risk level alcohol consumption and protect from pregnancy loss.

Read Full Article,
http://www.alcoholjournal.org/article/S0741-8329(12)00031-6/abstract

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Alcohol, Received 19 April 2011; received in revised form 12 September 2011; accepted 23 September 2011. published online 22 March 2012.

48. NEONATAL SCREENING FOR PRENATAL ALCOHOL EXPOSURE: ASSESSMENT OF VOLUNTARY MATERNAL PARTICIPATION IN AN OPEN MECONIUM SCREENING PROGRAM

Irene Zelner, Sarit Shor, Hazel Lynn, Henry Roukema, Lisa Lum, Kirsten Eisinga, Gideon Koren

ABSTRACT
Meconium fatty acid ethyl esters (FAEEs) are validated biomarkers of fetal alcohol exposure. Meconium FAEE testing can potentially be used as a screen by health-care professionals to identify neonates at-risk for Fetal Alcohol Spectrum Disorder, thereby permitting diagnostic follow-up of these children and early intervention in those who develop disabilities. The purpose of this study was to assess whether women would willingly partake in a screening program of this nature. This was determined by launching a pilot screening program for prenatal alcohol exposure in a high-risk obstetric unit previously shown to have a high prevalence of FAEE-positive meconium via anonymous meconium testing. The program involved voluntary testing of meconium for FAEEs and long-term developmental follow-up of positive cases through an existing public health program. The participation rate in the screening program was significantly lower than when testing was conducted anonymously (78% vs. 95%, respectively; $p < 0.05$), and the positivity rate was 3% in contrast to 30% observed under anonymous conditions ($p < 0.001$). These low rates suggest that the majority of mothers who consumed alcohol in pregnancy refused to participate. We conclude that despite the potential benefits of such screening programs, maternal unwillingness to consent, likely due to fear, embarrassment, and guilt, may limit the effectiveness of meconium testing for population-based open screening, highlighting the need for public education and social marketing efforts for such programs to be of benefit.

Read Full Article,
http://www.alcoholjournal.org/article/S0741-8329(12)00021-3/abstract

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Wiley Online Library - Acta Obstetricia et Gynecologica Scandinavica, 21st March 2012
DOI: 10.1111/j.1600-0412.2012.01402.x

49. ALCOHOL PREVENTION IN SWEDISH ANTENATAL CARE: EFFECTIVENESS AND PERCEPTIONS OF THE RISK DRINKING PROJECT COUNSELING MODEL

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ABSTRACT

Objectives: To compare an earlier Swedish antenatal care counseling routine concerning alcohol consumption with an expanded model in terms of effectiveness in achieving abstinence in pregnancy. A further objective was to assess the women's perceptions of the alcohol counseling.

Design: Cohort study. Setting. Antenatal care center in a provincial Swedish university town. Population. Women who received alcohol counseling; 1533 in cohort 1 (routine counseling) and 1476 in cohort 2 (expanded model). Approximately 93% of all pregnant women in Linköping are registered at this center. Methods. Data were collected by means of an anonymous questionnaire. Thirteen questions in the questionnaire were analyzed for this study. Main outcome measures. Replies from three questions concerning pre-pregnancy drinking and three questions on drinking during pregnancy.

Results: The response rate was 60% for cohort 1 and 64% for cohort 2. Perceptions of the advice from the antenatal care center were generally favorable. Similar proportions of women, approximately 6%, in both cohorts drank at least once during the pregnancy (after pregnancy recognition). There were four predictors for drinking during pregnancy: older age, having previously given birth to a child, frequency of pre-pregnancy drinking, and perceiving the message from antenatal care as “small amounts of alcohol during pregnancy don't matter”.

Conclusions: An expanded counseling model implemented in Swedish antenatal care did not reduce the proportion of women who continued drinking during pregnancy in comparison with a previous counseling model although the advice provided in the new model was perceived more favorably.

Read Full Article,
http://onlinelibrary.wiley.com/doi/10.1111/j.1600-0412.2012.01402.x/abstract

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PubMed, J Vis Exp. 2012 Mar 20;(61). pii: 3704. doi: 10.3791/3704.

50. ASSESSING TERATOGENIC CHANGES IN A ZEBRAFISH MODEL OF FETAL ALCOHOL EXPOSURE

Loucks E, Ahlgren S.
Program in Developmental Biology, Children's Memorial Research Center.

ABSTRACT

Fetal alcohol syndrome (FAS) is a severe manifestation of embryonic exposure to ethanol. It presents with characteristic defects to the face and organs, including mental retardation due to disordered and damaged brain development. Fetal alcohol spectrum disorder (FASD) is a term used to cover a continuum of birth defects that occur due to maternal alcohol consumption, and occurs in approximately 4% of children born in the United States. With 50% of child-bearing age women reporting consumption of alcohol, and half of all pregnancies being unplanned, unintentional exposure is a continuing issue(2). In order to best understand the damage produced by ethanol, plus produce a model with which to test potential interventions, we developed a model

of developmental ethanol exposure using the zebrafish embryo. Zebrafish are ideal for this kind of teratogen study(3-8). Each pair lays hundreds of eggs, which can then be collected without harming the adult fish. The zebrafish embryo is transparent and can be readily imaged with any number of stains. Analysis of these embryos after exposure to ethanol at different doses and times of duration and application shows that the gross developmental defects produced by ethanol are consistent with the human birth defect. Described here are the basic techniques used to study and manipulate the zebrafish FAS model.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22453686

Watch Video Article at,
http://www.jove.com/video/3704/assessing-teratogenic-changes-in-a-zebrafish-model-of-fetal-alcohol-exposure

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PubMed, Alcohol Clin Exp Res. 2012 Mar 20. doi: 10.1111/j.1530-0277.2012.01763.x.

51. FALSE-POSITIVE MECONIUM TEST RESULTS FOR FATTY ACID ETHYL ESTERS SECONDARY TO DELAYED SAMPLE COLLECTION

Zelner I, Hutson JR, Kapur BM, Feig DS, Koren G.
Division of Clinical Pharmacology & Toxicology, The Hospital for Sick Children, Toronto, Ontario, Canada; Department of Pharmacology & Toxicology, University of Toronto, Toronto, Ontario, Canada.

ABSTRACT
Background: Meconium analysis for fatty acid ethyl esters (FAEEs) is a validated method for identifying heavy prenatal ethanol (EtOH) exposure. This study investigated whether delayed sample collection can result in false-positive test results for FAEEs because of collection of samples potentially contaminated with postnatally produced stool.

Methods: Serial excretions were prospectively collected from neonates born to nondrinking mothers to capture the transition from meconium to postnatal stool. These were analyzed for FAEEs using headspace-solid phase microextraction and gas chromatography-mass spectrometry. Experiments involving incubation of samples with glucose or EtOH were performed to explore a potential mechanism of FAEE elevation.

Results: A total of 136 samples were collected from 30 neonates during their first few days of life (median of 4 samples/baby over a mean period of 68.5 hours postpartum). Although the first-collected meconium sample tested negative for FAEEs in all babies, later samples tested above the 2 nmol/g positive cutoff in 19 of 30 babies. Median time to appearance of FAEE-positive samples was 59.2 hours postpartum. In vitro experiments demonstrated that FAEE levels can be further increased in late samples (likely containing postnatal stool) after incubation with glucose, and that FAEEs are readily formed in meconium in the presence of EtOH.

Conclusions: Collection of samples excreted later in the postpartum period can lead to false-positive test results for FAEEs, which could be because of contamination with dietary components of postnatally produced stool and EtOH-producing microorganisms. Clinically, it is critical to collect the earliest possible excretion for determination of FAEEs to ensure that the FAEE content is representative of in utero EtOH exposure.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22432438

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PubMed, Hippocampus. 2012 Mar 19. doi: 10.1002/hipo.22009.

52. THE EFFECTS OF PERINATAL CHOLINE SUPPLEMENTATION ON HIPPOCAMPAL CHOLINERGIC DEVELOPMENT IN RATS EXPOSED TO ALCOHOL DURING THE BRAIN GROWTH SPURT

Monk BR, Leslie FM, Thomas JD.
Department of Psychology, Center for Behavioral Teratology, San Diego State University, San Diego, California.

ABSTRACT

Prenatal alcohol exposure leads to long-lasting cognitive and attention deficits, as well as hyperactivity. Using a rat model, we have previously shown that perinatal supplementation with the essential nutrient, choline, can reduce the severity of some fetal alcohol effects, including hyperactivity and deficits in learning and memory. In fact, choline can mitigate alcohol-related learning deficits even when administered after developmental alcohol exposure, during the postnatal period. However, it is not yet known how choline is able to mitigate alcohol-related behavioral alterations. Choline may act by altering cholinergic signaling in the hippocampus. This study examined the effects of developmental alcohol exposure and perinatal choline supplementation on hippocampal M(1) and M(2/4) muscarinic receptors. Sprague-Dawley rat pups were orally intubated with ethanol (5.25 mg/kg/day) from postnatal days (PD) 4-9, a period of brain development equivalent to the human third trimester; control subjects received sham intubations. From PD 4-30, subjects were injected s.c. with choline chloride (100 mg/kg/day) or saline vehicle. Open field activity was assessed from PD 30 through 33, and brain tissue was collected on PD 35 for autoradiographic analysis. Ethanol-exposed subjects were more active compared to controls during the first 2 days of testing, an effect attenuated with choline supplementation. Developmental alcohol exposure significantly decreased the density of muscarinic M(1) receptors in the dorsal hippocampus, an effect that was not altered by choline supplementation. In contrast, developmental alcohol exposure significantly increased M(2/4) receptor density, an effect mitigated by choline supplementation. In fact, M(2/4) receptor density of subjects exposed to alcohol and treated with choline did not differ significantly from that of controls. These data suggest that developmental alcohol exposure can cause long-lasting changes in the hippocampal cholinergic system and that perinatal choline supplementation may attenuate alcohol-related behavioral changes by influencing cholinergic systems.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22431326

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PubMed, Basic Clin Pharmacol Toxicol. 2012 Mar 19. doi: 10.1111/j.1742-7843.2012.00879.x.

53. ACUTE EFFECTS OF ETHANOL ON GLUTAMATE RECEPTORS

Möykkynen T, Korpi ER.
Institute of Biomedicine, Pharmacology, University of Helsinki, Helsinki, Finland; Department of Biosciences, Division of Biochemistry and Biotechnology, University of Helsinki, Helsinki, Finland.

ABSTRACT

Several studies have revealed that acute ethanol inhibits the function of glutamate receptors. Glutamate receptor-mediated synaptic plasticity, such as N-methyl-d-aspartate-dependent long-term potentiation, is also inhibited by ethanol. However, the inhibition seems to be restricted to certain brain areas such as the hippocampus, amygdala and striatum. Ethanol inhibition of glutamate receptors generally requires relatively high concentrations and may therefore explain consequences of severe ethanol intoxication such as impairment of motor performance and memory. Effects of ethanol on glutamate system of developing nervous system may have a role in causing foetal alcohol syndrome. Newly found regulatory proteins of α-amino-3-hydroxy-5-methyl-

4-isoxazolepropionic acid AMPA receptors seem to affect ethanol inhibition thus opening new lines of research.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22429661

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Pediatrics – Offical Journal of the American Academy of Pediatrics
Published online March 19, 2012
PEDIATRICS Vol. 129 No. 4 April 1, 2012 , pp. 681 -688 (doi: 10.1542/peds.2011-2209)

54. PRENATAL METHAMPHETAMINE EXPOSURE AND CHILDHOOD BEHAVIOR PROBLEMS AT 3 AND 5 YEARS OF AGE

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[d] Department of Psychology, The University of Tulsa, Tulsa, Oklahoma;
[e] Blank Children's Hospital Regional Child Protection Center, Iowa Health, Des Moines, Iowa;
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[g] Intramural Research Program, National Institute on Drug Abuse, National Institutes of Health, Baltimore, Maryland

ABSTRACT

Objective: We evaluated behavior problems in children who were prenatally exposed to methamphetamine (MA) at ages 3 and 5 years.

Methods: The Infant Development, Environment, and Lifestyle study, a prospective, longitudinal study of prenatal MA exposure and child outcome, enrolled subjects postpartum in Los Angeles, California; Honolulu, Hawaii; Des Moines, Iowa; and Tulsa, Oklahoma. Prenatal exposure was determined by maternal self-report and/or meconium results. Exposed and comparison groups were matched on race, birth weight, public health insurance, and education. Mothers in the comparison group denied use and had a negative meconium screen for amphetamines. Prenatal exposures to tobacco, alcohol, or marijuana occurred in both groups. At ages 3 and 5 years, 330 children (166 exposed and 164 comparison) were assessed for behavior problems by using the caregiver report on the Child Behavior Checklist.

General linear mixed models were used to determine the effects of prenatal MA exposure, including heavy exposure (≥3 days per week), age, and the interaction of exposure and age on behavior problems with adjustment for other drugs of abuse and environmental risk factors.

Results: MA exposure was associated with increased emotional reactivity and anxious/depressed problems at both ages and externalizing and attention-deficit/hyperactivity disorder problems by age 5 years. Heavy exposure was related to attention problems and withdrawn behavior at both ages. There were no effects of MA on the internalizing or total behavior problems scales.

Conclusions: This first report of behavior problems in patients as young as 3 years associated with MA exposure identifies an important public health problem. Continued follow-up can inform

the development of preventive intervention programs.

Read Full Article,
http://pediatrics.aappublications.org/content/129/4/681.abstract?sid=1d07ab33-55b3-4584-8f04-5543cf988bfe

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Wiley Online Library - Journal of Paediatrics and Child Health, Volume 48, Issue 3, pages 190–192, March 2012
Article first published online: 14 MAR 2012, DOI: 10.1111/j.1440-1754.2012.02422.x

55. THERE'S HOPE IN THE VALLEY

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ABSTRACT
Aboriginal women in the remote Fitzroy Valley region in Western Australia's Kimberley were concerned about high rates of alcohol use in pregnancy and its possible impact on child development. They successfully lobbied for restricted access to alcohol in 2007. In 2009 they developed a strategy for the diagnosis and prevention of Fetal Alcohol Spectrum Disorders (FASD) and the support of parents and carers of affected children. Aboriginal organisations then partnered with research and clinical groups from Sydney to conduct a FASD prevalence study. This commenced in 2010 following extensive community consultation and receipt of community consent. Data from this study are still being collected and will be used by the community to advocate for improved services and new models of health care. Prevention of FASD is important to optimise health and development for future generations of Aboriginal children and to ensure the transfer of culture and language from one generation to the next.

Read Full Article,
http://onlinelibrary.wiley.com/doi/10.1111/j.1440-1754.2012.02422.x/abstract

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Wiley Online Library - Alcoholism: Clinical and Experimental Research
Article first published online: 13 MAR 2012
DOI: 10.1111/j.1530-0277.2012.01754.x

56. QUANTITATIVE TRAIT LOCUS MAPPING FOR ETHANOL TERATOGENESIS IN BXD RECOMBINANT INBRED MICE

Chris Downing[1,*], Christina Balderrama-Durbin[1], Alexi Kimball[1], Jami Biers[1], Hali Wright[1], David Gilliam[2], Thomas E. Johnson[1,3]
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2 School of Psychological Sciences, University of Northern Colorado, Greeley, Colorado
3 Department of Integrative Physiology, University of Colorado, Boulder, Colorado

ABSTRACT
Background: Individual differences in susceptibility to the detrimental effects of prenatal ethanol

(EtOH) exposure have been demonstrated. Many factors, including genetics, play a role in susceptibility and resistance. We have previously shown that C57BL/6J (B6) mice display a number of morphological malformations following an acute dose of EtOH in utero, while DBA/2J (D2) mice are relatively resistant. Here, we present the results of quantitative trait locus (QTL) mapping for EtOH teratogenesis in recombinant inbred strains derived from a cross between B6 and D2 (BXD RIs).

Methods: Pregnant dams were intubated with either maltose-dextrin or 5.8 g/kg EtOH on day 9 of gestation (GD9). On GD 18, dams were sacrificed and fetuses and placentae were removed. Placentae and fetuses were weighed; fetuses were sexed and examined for gross morphological malformations. Fetuses were then either placed in Bouin's fixative for subsequent soft-tissue analyses or eviscerated and placed in EtOH for subsequent skeletal examinations. QTL mapping for maternal weight gain (MWG), prenatal mortality, fetal weight (FW) at c-section, placental weight (PW), and several morphological malformations was performed using WebQTL.

Results: Heritability for our traits ranged from 0.06 for PW to 0.39 for MWG. We found suggestive QTLs mediating all phenotypes and significant QTLs for FW and digit and rib malformations. While most QTL regions are large, several intriguing candidate genes emerged based on polymorphisms between B6 and D2 and gene function.

Conclusions: In this first mapping study for EtOH teratogenesis, several QTLs were identified. Future studies will further characterize these regions. Identification of genes and epigenetic modifications mediating susceptibility to the teratogenic effects of alcohol in mice will provide targets to examine in human populations.

Read Full Article,
http://onlinelibrary.wiley.com/doi/10.1111/j.1530-0277.2012.01754.x/abstract

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PubMed, Alcohol Alcohol. 2012 Mar 13.

57. THE EFFECTS OF PRENATAL ALCOHOL EXPOSURE ON THE DEVELOPMENTAL RETINA OF MICE

Deng JX, Liu X, Zang JF, Huang HE, Xi Y, Zheng H, Yao HL, Yu DM, Deng JB.
Institute of Neurobiology of Nursing College, Henan University, Kaifeng 475004, People's Republic of China.

ABSTRACT
Aims: Our aim is to investigate the effects of prenatal alcohol exposure (PAE) on the development of retinal bipolar and horizontal cells.

Methods: The alterations of the retinal bipolar and horizontal cells in P7, P14 and P30 mice were observed after PAE, with immunofluorescent labeling and DiI diolistic assay.

Results: The retinal development of filial pups was affected by PAE in a dose-dependent and long-term manner. The number of bipolar cells of alcohol groups was significantly lower than that of the control, and the dendritic receptive field of horizontal cells was also significantly smaller than those of the control groups ($P < 0.01$). Conclusion: PAE was able to cause retarded development of pup retinal neural cells.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22414921

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Wiley Online Library - Developmental Medicine & Child Neurology
Article first published online: 12 MAR 2012
DOI: 10.1111/j.1469-8749.2012.04254.x

58. CHILDREN WITH FETAL ALCOHOL SPECTRUM DISORDER SHOW AN AMBLYOPIA-LIKE PATTERN OF VISION DEFICIT

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ABSTRACT

Aim: The aim of the study was to assess and characterize visual functioning in children with fetal alcohol spectrum disorder (FASD) using a broader and more inclusive range of measures than has been reported previously.

Method: Standard tests of visual functioning were used to assess 21 children (11 females, 10 males) with FASD and 21 sex- and age-matched comparison children without FASD. The age of the children ranged from 6 years 9 months to 11 years 11 months (mean 9y 6mo). Children were tested individually under standardized conditions for visual acuity, stereoacuity, contrast sensitivity, ocular alignment/motility, color vision, and refractive error.

Results: Compared with non-affected children, children with FASD showed deficits in visual acuity, contrast sensitivity, and stereoacuity. Ocular alignment/motility, refractive error, and color vision measures were normal. Among children with FASD, 62% met the criteria for referral to an eye specialist, compared with 20% of children without FASD.

Interpretation: Children with FASD showed an amblyopia-like pattern of vision deficit in the absence of the optical and oculomotor disruptions of early experience that usually precede this condition. Evidence from animal models suggests that the deficits in spatial vision may be due to alterations in the functional architecture of the neocortex that occurs following prenatal alcohol exposure.

Read Full Article,
http://onlinelibrary.wiley.com/doi/10.1111/j.1469-8749.2012.04254.x/abstract

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PubMed, Klin Padiatr. 2012 Mar;224(2):66-71. Epub 2012 Mar 9.

59. CHILDREN AND ADOLESCENTS WITH FETAL ALCOHOL SYNDROME (FAS): BETTER SOCIAL AND EMOTIONAL INTEGRATION AFTER EARLY DIAGNOSIS

Alex K, Feldmann R.
University Hospital, Pediatrics, Münster, Germany.

ABSTRACT

Alcoholism during pregnancy is one of the most common factors in western societies causing persisting congenital and multiple physical as well as neurological impairments. Finding the diagnosis at first sight puts medical professionals into a demanding situation. Therefore the objective of this study was to detect patients' developmental characteristics with the main focus on the necessity of the diagnosis itself.125 young patients, whose diagnosis of fetal alcohol syndrome (FAS) was made at the Muenster University Hospital, were followed up.Biographic details such as

living conditions, health, developmental problems and educational career were gathered using a structured interview.

The diagnosis itself and the impact of this on the patients were also explored.Patients displayed characteristics of a less mature trait of character. The majority were looked after by foster parents. High rates of social and developmental problems could be found.

The diagnosis was identified as a protective factor, with significantly better outcomes for patients being diagnosed in early childhood. A diagnosis established later in life was particularly helpful for the families and caregivers. Feelings of failure and self-blame could be diminished.The early detection of affected children has to be improved as receiving the correct diagnosis, despite the persistent impairments, is of major benefit for both patients and their families.

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http://www.ncbi.nlm.nih.gov/pubmed/22407470

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PubMed, Qual Life Res. 2012 Mar 9.

60. THE FATIGUE ASSOCIATED WITH DEPRESSION QUESTIONNAIRE (FASD): RESPONSIVENESS AND RESPONDER DEFINITION

Matza LS, Wyrwich KW, Phillips GA, Murray LT, Malley KG, Revicki DA.
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ABSTRACT

Purpose: The Fatigue Associated with Depression Questionnaire (FAsD) was developed to assess fatigue and its impact among patients with depression. The purpose of this study was to examine the questionnaire's responsiveness to change and identify a responder definition for interpretation of treatment-related changes.

Methods: Data were collected at baseline and at 6 weeks from patients with depression starting treatment with a new antidepressant.

Results: Of the 96 participants, 55.2% were women, with a mean age of 43.4 years. The total score and both subscales demonstrated statistically significant change with moderate to large effect sizes (absolute values ≥0.76). FAsD change scores were significantly correlated with change on the Brief Fatigue Inventory ($r \geq 0.73$; $p < 0.001$).

FASD mean change scores discriminated among patient subgroups differing by degree of improvement in patient- and clinician-reported fatigue and depression. Responder definition for the two subscales and total score (0.67, 0.57, 0.62) was estimated primarily based on mean change among patients who reported a small but important improvement in fatigue.

Discussion: The FAsD was responsive to change, and the responder definition may be used when interpreting treatment-related change. Results add to previous findings suggesting the FAsD is a useful measure of fatigue among patients with depression.

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http://www.ncbi.nlm.nih.gov/pubmed/22403040

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Wiley Online Library - Alcoholism: Clinical and Experimental Research
Article first published online: 8 MAR 2012, DOI: 10.1111/j.1530-0277.2012.01750.x

61. RELATION OVER TIME BETWEEN FACIAL MEASUREMENTS AND COGNITIVE OUTCOMES IN FETAL ALCOHOL-EXPOSED CHILDREN

Tatiana Foroud[1],*, Leah Wetherill[1], Sophia Vinci-Booher[1], Elizabeth S. Moore[2], Richard E. Ward[2], H. Eugene Hoyme[3], Luther K. Robinson[4], Jeffrey Rogers[5], Ernesta M. Meintjes[6], Christopher D. Molteno[7], Joseph L. Jacobson[8,9], Sandra W. Jacobson[8,9]

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8 Department of Psychiatry and Behavioral Neurosciences, Faculty of Health Sciences, University of Cape Town, Cape Town, South Africa
9 Department of Psychiatry and Behavioral Neurosciences, Wayne State University School of Medicine, Detroit, Michigan

ABSTRACT

Background: The identification of individuals exposed prenatally to alcohol can be challenging, with only those having the characteristic pattern of facial features, central nervous system abnormality, and growth retardation receiving a clinical diagnosis of fetal alcohol syndrome (FAS).

Methods: Seventeen anthropometric measurements were obtained at 5 and 9 years from 125 Cape Town, South African children, studied since birth. The children were divided into 3 groups: FAS or partial FAS (PFAS), heavily exposed nonsyndromal (HE), and non-alcohol-exposed controls (C). Anthropometric measurements were evaluated for mean group differences. Logistic regression models were used to identify the subset of anthropometric measures that best predicted group membership. Anthropometric measurements were examined at the 2 ages in relation to prenatal alcohol exposure obtained prospectively from the mothers during pregnancy. Correlation of these facial measurements with key neurobehavioral outcomes including Wechsler Intelligence Scales for Children-IV IQ and eyeblink conditioning was used to assess their utility as indicators of alcohol-related central nervous system impairment.

Results: Significant group differences were found for the majority of the anthropometric measures, with means of these measures smaller in the FAS/PFAS compared with HE or C. Upper facial widths, ear length, lower facial depth, and eye widths were consistent predictors distinguishing those exposed to alcohol from those who were not. Using longitudinal data, unique measures were identified that predicted facial anomalies at one age but not the other, suggesting the face changes as the individual matures. And 41% of the FAS/PFAS group met criteria for microtia at both ages. Three of the predictive anthropometric measures were negatively related to measures of prenatal alcohol consumption, and all were positively related to at least 1 neurobehavioral outcome.

Conclusions: The analysis of longitudinal data identified a common set of predictors, as well as some that are unique at each age. Prenatal alcohol exposure appears to have its primary effect on brain growth, reflected by smaller forehead widths, and may suppress neural crest migration to the branchial arches, reflected by deficits in ear length and mandibular dimensions. These results may

improve diagnostic resolution and enhance our understanding of the relation between the face and the neuropsychological deficits that occur.

Read Full Article,
http://onlinelibrary.wiley.com/doi/10.1111/j.1530-0277.2012.01750.x/abstract

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Wiley Online Library - Alcoholism: Clinical and Experimental Research
Article first published online: 8 MAR 2012
DOI: 10.1111/j.1530-0277.2012.01743.x

62. ACUTE AND LONG-TERM PURKINJE CELL LOSS FOLLOWING A SINGLE ETHANOL BINGE DURING THE EARLY THIRD TRIMESTER EQUIVALENT IN THE RAT

Nirelia M. Idrus, Ruth M. A. Napper
Department of Anatomy and Structural Biology, University of Otago, Dunedin, New Zealand

ABSTRACT
Background: In the rat, binge-like ethanol (EtOH) exposure during the early neonatal period (a developmental period equivalent to the human third trimester) can result in a permanent deficit of cerebellar Purkinje cells (Pcells). However, the consequences of a moderate binge alcohol exposure on a single day during this postnatal period have not been established. This is an issue of importance as many pregnant women binge drink periodically at social drinking levels. This study aimed to identify both the acute and long-term effects of exposure to a single alcohol binge that achieved a mean peak blood EtOH concentration of approximately 250 mg/dl during early postnatal life using a rat model of fetal alcohol spectrum disorders.

Methods: Acute apoptotic Pcell death 10 hours after a moderate dose binge EtOH exposure from postnatal days (PDs) 0 to 10 was assessed using active caspase-3 immunolabeling. Acute Pcell apoptosis was quantified in cerebellar vermal lobules I–X using the physical disector method. Long-term effects were assessed at PD 60 using stereological methods to determine total Pcell numbers in the vermis, lobule III, and lobule IX, following a moderate dose binge EtOH exposure at PDs 0, 2, or 4.

Results: Acute apoptosis was induced by EtOH on PDs 1 to 8 in a time and lobular-dependent manner. For EtOH exposure on PD 2, significant long-term Pcell loss occurred in lobule III. EtOH exposure on PD 4 resulted in significant long-term Pcell loss throughout the entire vermis.

Conclusions: These results indicate that a single, early EtOH episode of moderate dose can create significant and permanent Pcell loss in the developing cerebellum.

Read Full Article,
http://onlinelibrary.wiley.com/doi/10.1111/j.1530-0277.2012.01743.x/abstract

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PubMed, J Pediatr Psychol. 2012 Mar 7.

63. SOURCE MONITORING IN CHILDREN WITH AND WITHOUT FETAL ALCOHOL SPECTRUM DISORDERS

Kully-Martens K, Pei J, Job J, Rasmussen C.
Department of Pediatrics, University of Alberta, and Department of Educational Psychology, University of Alberta.

ABSTRACT
Objectives: Deficits in memory are well-documented in children with fetal alcohol spectrum

disorders (FASD); however, one aspect of memory not yet studied in children with FASD is source monitoring. This study examined overall source monitoring ability and performance profiles of children with FASD compared to controls.

Methods: Participants included 19 children with FASD and 38 typically developing children (aged 6-12 years). Children were presented with auditory word lists and were required to recall the source of words for reality, external, and internal source monitoring tasks.

Results: Children with FASD showed poorer performance than controls across all three conditions in both recognition memory and memory for source. However, both groups exhibited a comparable pattern of performance across conditions. Specifically, performance was lowest on the internal task and highest on the reality task.

Conclusions: Information about source monitoring deficits further delineates the intricacies of memory deficits in FASD, and has implications for both assessment and intervention.

Link to the Article,
http://www.ncbi.nlm.nih.gov/pubmed/22381645

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PubMed, Trends Neurosci.
2012 Mar 6.

64. DOES MODERATE DRINKING HARM THE FETAL BRAIN? INSIGHTS FROM ANIMAL MODELS

Valenzuela CF, Morton RA, Diaz MR, Topper L.

Department of Neurosciences, School of Medicine, University of New Mexico Health Sciences Center, Albuquerque, NM 87131, USA.

ABSTRACT

Although public health campaigns advise pregnant women to abstain from ethanol, drinking during pregnancy is pervasive. Here, we highlight recent studies that have clearly demonstrated long-lasting neurobehavioral deficits in the offspring of laboratory animals exposed to moderate levels of ethanol during development. Alterations in learning, memory, motor coordination, social behavior, and stress responses were identified in these animals. Increased vulnerability to substance abuse was also demonstrated.

These behavioral alterations have been associated with impairments in neurotransmitter systems, neuromodulators, and/or synaptic plasticity in several brain regions. With this review we hope to contribute to a better appreciation of the potential effects of developmental exposure to moderate ethanol levels, leading to better interventions aimed at relieving fetal alcohol spectrum disorders.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22402065

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Translational Psychiatry (2012) 2, e84; doi:10.1038/tp.2012.12
Published online 6 March 2012
Received 25 May 2011; Revised 23 January 2012; Accepted 25 January 2012

65. PRENATAL EXPOSURE TO CIGARETTE SMOKE OR ALCOHOL AND CEREBELLUM VOLUME IN ATTENTION-DEFICIT/HYPERACTIVITY DISORDER AND TYPICAL DEVELOPMENT

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ABSTRACT
Prenatal exposure to teratogenic substances, such as nicotine or alcohol, increases the risk of developing attention-deficit/hyperactivity disorder (ADHD). To date, studies examining this relationship have used symptom scales as outcome measures to assess the effect of prenatal exposure, and have not investigated the neurobiological pathways involved. This study explores the effect of prenatal exposure to cigarettes or alcohol on brain volume in children with ADHD and typically developing controls. Children with ADHD who had been exposed prenatally to either substance were individually matched to children with and without ADHD who had not been. Controls who had been exposed prenatally were also individually matched to controls who had not been. For prenatal exposure to both smoking and alcohol, we found a pattern where subjects with ADHD who had been exposed had the smallest brain volumes and unexposed controls had the largest, with intermediate volumes for unexposed subjects with ADHD. This effect was most pronounced for cerebellum. A similar reduction fell short of significance for controls who had been exposed to cigarettes, but not alcohol. Our results are consistent with an additive effect of prenatal exposure and ADHD on brain volume, with the effects most pronounced for cerebellum.

Read Full Article,
http://www.nature.com/tp/journal/v2/n3/full/tp201212a.html

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Wiley Online Library - Drug and Alcohol Review
Special Issue: Low Risk Drinking Guidelines. Guest Editors: Tim Stockwell and Robin Room
Volume 31, Issue 2, pages 121–125, March 2012
Article first published online: 5 MAR 2012
DOI: 10.1111/j.1465-3362.2011.00416.x

66. CONSTRUCTING AND RESPONDING TO LOW-RISK DRINKING GUIDELINES: CONCEPTUALISATION, EVIDENCE AND RECEPTION

Tim Stockwell[1], Robin Room[2]
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2 Centre for Alcohol Policy Research, Turning Point Alcohol & Drug Centre, Fitzroy, Australia, School of Population Health, University of Melbourne, Melbourne, Australia, Centre for Social Research on Alcohol & Drugs, Stockholm University, Stockholm, Sweden

ABSTRACT
Despite significant scientific and communication challenges, government and non-government agencies in numerous countries continue to issue advice to the populace on how to reduce health and safety risks when alcohol is consumed. Often referred to as 'low-risk drinking guidelines' (LRDG), the advice typically recommends both average and upper daily levels for consumption for

men and women, as well as when to abstain completely. The scientific and conceptual bases for these guidelines have only rarely been precisely spelt out and subjected to independent scrutiny. Having both been engaged in the development and revision of LRDGs in Australia and Canada at different times over the past 15 years and having come to different conclusions [l,2]—we hope the various perspectives and empirical analyses presented here will contribute to informed debate, clearer thinking and perhaps eventually an improved conceptual and empirical basis for future guidelines.

The challenges in developing simple upper limits to consumption on a particular occasion and in terms of volume over time are immense despite or maybe because of the voluminous literature linking levels of alcohol consumption with risks of various types of social and health harm. In the first place, there is enormous variation in the riskiness of a particular dose or volume of alcohol. Individual responses to a standard dose of alcohol vary substantially. Whether or not food has been taken beforehand can affect obtained blood alcohol levels by two or threefold [3]; body weight varies across individuals to an even greater extent; both metabolic and functional tolerance to the effects of alcohol vary substantially across individual drinkers, but also over time for one person as a function of their current pattern of drinking and age [4]; risk of injury from a given dose is influenced to a large degree by drinking context [5]; for longer-term or chronic risks of serious illnesses, a whole range of other lifestyle, genetic and other risk factors interact with the effects of alcohol [6]. In the second place, there are substantial problems in measuring and communicating the dosage of alcohol. It is extremely difficult outside of a controlled experimental environment to determine the typical doses of alcohol people administer themselves in their daily lives [7].

In most countries which permit the sale of alcohol, there are typically thousands, even tens of thousands, of different types of alcoholic products varying in terms of taste, price, alcohol content and volume. Beers can vary in strength from 0.5% to 25% or more as just one example—and of course serve sizes can go from a sip to a barrel. Epidemiological studies of patterns of alcohol use by necessity simplify this variation using such devices as the concept of a ‘standard drink’, even though in practice there is wide divergence in actual serve sizes and understandings of this term [7,8]. In the third place, most epidemiological studies of alcohol, health and safety use self-report measures of consumption which typically underestimate actual consumption by large margins [7,9]. Even when one thinks one is on firm ground by using ‘abstainers’ as a comparison group, a close examination of this assumption reveals it also to be ephemeral: estimates of risk can shift substantially depending on whether former drinkers (whether moderate or heavy) are included in this category [10].

If there are difficulties with the quantification off exposure to risk from alcohol, assessment of the harms can be equally fraught. There are at least 60 major categories of serious health consequences and over 200 specific diagnostic codes in the International Classification of Diseases-10 (ICD-10) to which drinking contributes causally, with a wide range of different risk functions of the relation between level of consumption and the disease—the shape can be linear, U-shaped, J-shaped, exponential or other [1 1].

Against such odds, is it scientifically defensible to offer simple ‘ballpark’ upper limit advice to the general public? Two commentators in this issue present opposing views: Nick Heather suggests there is a moral imperative to provide consumers with advice to protect their health [12] ; Sally Casswell argues there is wide scope for their misunderstanding, weak evidence for the effectiveness of alcohol education and the worrying possibility of diverting attention from more effective public policies [13]

Read Full Article,
http://onlinelibrary.wiley.com/doi/10.1111/j.1465-3362.2011.00416.x/abstract

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Unbound Medline, Child Neuropsychol 2012 Mar 5.

67. NEUROPSYCHOLOGICAL IMPAIRMENTS ON THE NEPSY-II AMONG CHILDREN WITH FASD

Rasmussen C, Tamana S, Baugh L, Andrew G, Tough S, Zwaigenbaum L

ABSTRACT

Background: We examined the pattern of neuropsychological impairments of children with FASD (compared to controls) on NEPSY-II measures of attention and executive functioning, language, memory, visuospatial processing, and social perception.

Methods: Participants included 32 children with FASD and 30 typically developing control children, ranging in age from 6 to 16 years. Children were tested on the following subtests of the NEPSY-II: Attention and Executive Functioning (animal sorting, auditory attention/response set, and inhibition), Language (comprehension of instructions and speeded naming), Memory (memory for names/delayed memory for names), Visual-Spatial Processing (arrows), and Social Perception (theory of mind). Groups were compared using MANOVA.

Results: Children with FASD were impaired relative to controls on the following subtests: animal sorting, response set, inhibition (naming and switching conditions), comprehension of instructions, speeded naming, and memory for names total and delayed, but group differences were not significant on auditory attention, inhibition (inhibition condition), arrows, and theory of mind. Among the FASD group, IQ scores were not correlated with performance on the NEPSY-II subtests, and there were no significant differences between those with and without comorbid ADHD.

Conclusions: The NEPSY-II is an effective and useful tool for measuring a variety of neuropsychological impairments among children with FASD. Children with FASD displayed a pattern of results with impairments (relative to controls) on measures of executive functioning (set shifting, concept formation, and inhibition), language, and memory, and relative strengths on measures of basic attention, visual spatial processing, and social perception.

Read Full Article,
http://www.unboundmedicine.com/medline/ebm/record/22384972/abstract/Neuropsychological_impairments_on_the_NEPSY_II_among_children_with_FASD

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PubMed, Prenat Diagn. 2012 Mar;32(3):277-83. doi: 10.1002/pd.3824.

68. ISOLATED CORPUS CALLOSUM AGENESIS: A TEN-YEAR FOLLOW-UP AFTER PRENATAL DIAGNOSIS (HOW ARE THE CHILDREN WITHOUT CORPUS CALLOSUM AT 10 YEARS OF AGE?)

Moutard ML, Kieffer V, Feingold J, Lewin F, Baron JM, Adamsbaum C, Gélot A, Isapof A, Kieffer F, de Villemeur TB.
Service de Neuropédiatrie, Pathologie du développement, Hôpital Trousseau, Paris, France. marielaure.moutard@trs.aphp.fr

ABSTRACT

Background: Corpus callosum agenesis (CCA) is generally diagnosed in utero. Outcome appears to be better if the malformation is isolated. The aim of this study, which is the first one with a long (10 years) and standardized follow up, was to report cognitive abilities of children with isolated CCA diagnosed prenatally.

Methods: We prospectively evaluated 17 children. Clinical examinations, neuropsychological tests were performed each year. School achievement and personal and familial data were collected.

Results: Twelve children completed the entire follow up. One child was finally considered to have associated CCA, because signs of fetal alcohol syndrome had become obvious. Of the 11 other children, three (27%) had borderline intelligence whereas the intelligence levels of eight (73%) were in the normal range, although half of these children experienced some difficulties in scholastic achievement. Neither epilepsy nor intellectual deficiency was noted and intellectual quotient scores correlated strongly with the mother's education level.

Conclusion: Although prenatal diagnosis of isolated CCA is reliable, false postnatal diagnoses remain possible (10-20%) even with complete prenatal screening. Outcome is mostly favorable because intelligence is within the normal range for nearly 3/4 of the children. However, they frequently have mild learning difficulties.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22430728

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Wiley Online Library - Alcoholism: Clinical and Experimental Research
Article first published online: 29 FEB 2012
DOI: 10.1111/j.1530-0277.2012.01745.x

69. DETECTION OF ALCOHOL USE IN THE SECOND TRIMESTER AMONG LOW-INCOME PREGNANT WOMEN IN THE PRENATAL CARE SETTINGS IN JEFFERSON COUNTY, ALABAMA

Qing Li[1],*, Janet Hankin[2], Sharon C. Wilsnack[3], Ernest L. Abel[4], Russell S. Kirby[5], Louis G. Keith[6], Sarah G. Obican[7]
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2 Department of Sociology, Wayne State University, Detroit, Michigan
3 Department of Clinical Neuroscience, School of Medicine & Health Sciences, University of North Dakota, Grand Forks, North Dakota
4 Department of Obstetrics & Gynecology, Wayne State University, Detroit, Michigan
5 Department of Community and Family Health, College of Public Health, University of South Florida, Tampa, Florida
6 Department of Obstetrics & Gynecology, Feinberg School of Medicine, Northwestern University, Chicago, Illinois
7 Department of Obstetrics & Gynecology, George Washington University, Washington, District of Columbia

ABSTRACT
Background: Prenatal alcohol use, a leading preventable cause of birth defects and developmental disabilities, remains a prevalent public health concern in the United States. This study aims to detect the proportion and correlates of prenatal alcohol use in the prenatal care settings in Alabama. Prenatal care settings were chosen because of their potential as stable locations to screen for and to reduce prenatal alcohol use within a community.

Methods: We conducted a cross-sectional study of 3,046 women in the 22 and 23 weeks of gestation who sought prenatal care in 8 community-based public clinics and participated in the Perinatal Emphasis Research Center project in Jefferson County, Alabama, from 1997 to 2001. Frequency and quantity of alcohol use in the past 3 months were assessed by research nurses during face-to-face interviews. We conducted logistic regression analyses to calculate adjusted odds ratios (ORs) and 95% confidence intervals (CIs) of correlates of prenatal alcohol use.

Results: Participants were predominantly young, African American, and unmarried, 86.5% on Medicaid. The proportion of alcohol use in the second trimester of pregnancy was 5.1%; 0.3% of women reported 4 or more drinks on a drinking day to research nurses. Older maternal age (OR =

1.11; 95% CI = 1.08 to 1.15), use of welfare (OR = 1.43; 95% CI = 1.02 to 2.02), and male partner–perpetrated violence (OR = 2.96; 95% CI = 1.92 to 4.56) were positively associated with elevated risk of prenatal alcohol use. Protective factors included higher levels of self-esteem (OR = 0.94; 95% CI = 0.89 to 0.98) and more years of education (OR = 0.88; 95% CI = 0.78 to 0.98).

Conclusions: Prenatal alcohol use remains a public health issue among low-income pregnant women in Jefferson County, Alabama. Research nurses detected it in the second trimester. Future studies need to encourage screening for prenatal alcohol use in the prenatal care settings by obstetrician-gynecologists, family physicians, nurses, and midwives. Combined interventions to educate and empower women and strengthen families are needed.

Read Full Article,
http://onlinelibrary.wiley.com/doi/10.1111/j.1530-0277.2012.01745.x/abstract

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PubMed, Exp Biol Med (Maywood). 2012 Mar 1;237(3):236-40. Epub 2012 Feb 29.

70. FETAL ETHANOL EXPOSURE ATTENUATES AVERSIVE ORAL EFFECTS OF TRPV1, BUT NOT TRPA1 AGONISTS IN RATS

Glendinning JI, Simons YM, Youngentob L, Youngentob SL.
Department of Biology, Barnard College, Columbia University, New York, NY 10027.

ABSTRACT
In humans, fetal ethanol exposure is highly predictive of adolescent ethanol use and abuse. Prior work in our labs indicated that fetal ethanol exposure results in stimulus-induced chemosensory plasticity in the taste and olfactory systems of adolescent rats. In particular, we found that increased ethanol acceptability could be attributed, in part, to an attenuated aversion to ethanol's aversive odor and quinine-like bitter taste quality. Here, we asked whether fetal ethanol exposure also alters the oral trigeminal response of adolescent rats to ethanol. We focused on two excitatory ligand-gated ion channels, TrpV1 and TrpA1, which are expressed in oral trigeminal neurons and mediate the aversive orosensory response to many chemical irritants.

To target TrpV1, we used capsaicin, and to target TrpA1, we used allyl isothiocyanate (or mustard oil). We assessed the aversive oral effects of ethanol, together with capsaicin and mustard oil, by measuring short-term licking responses to a range of concentrations of each chemical. Experimental rats were exposed in utero by administering ethanol to dams through a liquid diet. Control rats had ad libitum access to an iso-caloric iso-nutritive liquid diet. We found that fetal ethanol exposure attenuated the oral aversiveness of ethanol and capsaicin, but not mustard oil, in adolescent rats. Moreover, the increased acceptability of ethanol was directly related to the reduced aversiveness of the TrpV1-mediated orosensory input. We propose that fetal ethanol exposure increases ethanol avidity not only by making ethanol smell and taste better, but also by attenuating ethanol's capsaicin-like burning sensations.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22378825

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PubMed, J Child Neurol. 2012 Feb 28.

71. BRAIN SINGLE-PHOTON EMISSION COMPUTED TOMOGRAPHY IN FETAL ALCOHOL SYNDROME: A CASE REPORT AND STUDY IMPLICATIONS

Codreanu I, Yang J, Zhuang H.

ABSTRACT
The indications of brain single-photon emission computed tomography (SPECT) in fetal alcohol

syndrome are not clearly defined, even though the condition is recognized as one of the most common causes of mental retardation. This article reports a case of a 9-year-old adopted girl with developmental delay, mildly dysmorphic facial features, and behavioral and cognitive abnormalities. Extensive investigations including genetic studies and brain magnetic resonance imaging (MRI) revealed no abnormalities, and a diagnosis of fetal alcohol syndrome was considered since official diagnostic criteria were met. A brain SPECT was requested and showed severely decreased tracer activity in the thalami, basal ganglia, and temporal lobes on both sides, the overall findings being consistent with the established diagnosis of fetal alcohol syndrome. With increasing availability of functional brain imaging, the study indications and possible ethical implications in suspected prenatal alcohol exposure or even before adoption need further consideration. In this patient, SPECT was the only test to yield positive results.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22378656

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J Popul Ther Clin Pharmacol Vol 19(1):e51-e65;
February 25, 2012

72. A MODEL FOR ESTIMATING THE ECONOMIC IMPACT OF FETAL ALCOHOL SPECTRUM DISORDER

Svetlana Popova, Brenda Stade, Shannon Lange, Jürgen Rehm

ABSTRACT

Background: Fetal alcohol spectrum disorder (FASD) is a group of disorders with lifelong disabilities that require a large amount of support from various services including health, community, remedial education, and many others. Thus, FASD has a huge economic and societal impact.

Objectives: To develop a sound methodology for calculating a comprehensive, evidence-based picture of the economic impact of FASD for Canada.

Methods: The economic model was developed within the framework of the revised International Guidelines for Estimating the Costs of Substance Abuse. In addition, the Guidelines generated during the first national Roundtable held by the Public Health Agency of Canada were employed. The methodologies of the few existing studies on the economic cost of FASD from Canada and USA were also considered.

Results: A new and comprehensive methodology for estimating of the economic impact of FASD is presented. The model includes the direct and indirect costs associated with the experiences of those affected by FASD, as well as those of their families/caregivers during multiple life/developmental stages. Preliminary cost estimates for the main cost drivers for Canada are presented.

Conclusion: The developed methodology is appropriate for use in Canada, and has the potential to be used by other countries. The challenges associated with implementing the economic model and estimating the economic costs of FASD are discussed, as are the levels of analysis.

Read Full Article,
http://www.cjcp.ca/pubmed.php?articleId=357

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PubMed, Eur Child Adolesc Psychiatry. 2012 Apr;21(4):221-31. Epub 2012 Feb 23.

73. ADAPTIVE BEHAVIOUR IN CHILDREN AND ADOLESCENTS WITH FOETAL ALCOHOL SPECTRUM DISORDERS: A COMPARISON WITH SPECIFIC LEARNING DISABILITY AND TYPICAL DEVELOPMENT

Ase F, Ilona AR, Mirjam K, Pekka S, Eugene HH, Sarah MN, Marit K.
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ABSTRACT

Foetal alcohol spectrum disorders (FASD) is a leading cause of intellectual disability in the western world. Children and adolescents with FASD are often exposed to a double burden in life, as their neurological sequelae are accompanied by adverse living surroundings exposing them to further environmental risk.

In the present study, the adaptive abilities of a group of children and adolescents with FASD were examined using the Vineland Adaptive Behaviour Scales (VABS) and compared to those of a group of IQ-matched children with specific learning disorder (SLD) as well as with typically developing controls (CON). The results showed significantly different adaptive abilities among the groups: Children with FASD performed worse than IQ-matched children with SLD, who in turn performed worse than typically developing children on all domains (communication, daily living skills and socialization) on the VABS. Compared to the other groups, social skills declined with age in the FASD group. These results support previous studies of adaptive behaviour deficits in children with FASD and provide further evidence of the specificity of these deficits. On a societal level, more efforts and resources should be focused on recognizing and diagnosing FASD and supporting communication skills, daily living skills and most of all social skills across diagnostic groups within FASD. Without adequate intervention, adolescents and young adults with FASD run a great risk of marginalization and social maladjustment, costly not only to society but also to the lives of the many young people with FASD.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22358422

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PubMed, Front Genet. 2012;3:10.
Epub 2012 Feb 22.

74. THE EFFECT OF PRECONCEPTION PATERNAL ALCOHOL EXPOSURE ON EPIGENETIC REMODELING OF THE H19 AND RASGRF1 IMPRINTING CONTROL REGIONS IN MOUSE OFFSPRING

Knezovich JG, Ramsay M.
Molecular Genetics Laboratory, Division of Human Genetics, University of the Witwatersrand Johannesburg, South Africa.

ABSTRACT

Imprinted loci play a critical role in fetal development. Their expression is often regulated by CCCTC-binding factor (CTCF) protein binding at imprinting control regions (ICRs). Prenatal alcohol exposure has been shown to reduce global DNA methylation in the developing mouse fetus. This study explored the effect of preconception paternal alcohol exposure on DNA methylation at two paternally methylated ICRs (H19 and Rasgrf1) in the sperm of exposed males and somatic DNA of sired offspring. Significant reductions at the H19 CTCF 1 ($p = 0.0027$) and CTCF 2 ($p = 0.0009$) binding sites were observed in the offspring of ethanol-treated sires, which was significantly correlated with reduced weight at postnatal days 35-42 ($p < 0.05$). As birth weight was unaffected

and growth was only delayed during the postnatal weaning period, with subsequent re-convergence, we hypothesize that this may be the result of a mental deficit causing delayed establishment of independent feeding following weaning and would explain why this effect is transient. No difference in DNA methylation was observed in the sperm of alcohol-exposed males, indicating that the transmission of the epigenetic signal at conception is not due to altered methylation, but may be the result of an RNA-mediated mechanism or altered chromatin remodeling.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22371710

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Taylor & Francis Online, Journal of Addictive Diseases
Volume 31, Issue 1, 2012, Pages 19-28
DOI:10.1080/10550887.2011.642765
Available online: 22 Feb 2012

75. SUBSTANCE USE DURING PREGNANCY AND POSTNATAL OUTCOMES

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ABSTRACT

Substance exposure in utero has been associated with physical birth defects and increased risk of regulatory and neuropsychological difficulties. The aims of this study were to describe women who use substances and are in treatment with respect to the type and number of substances used during pregnancy, as well as their background, and to examine the effect substance use has on gestational age, birth weight, and the development of neonatal abstinence syndrome at birth. A sample of 161 pregnant women and their 163 newborn children were included.

The results indicate that the children whose mothers continued to use substances throughout their pregnancies were born at a lower gestational age (Chi-Square = 15.1(2), P < .01); children exposed to poly-substances in utero were more affected than those exposed to only alcohol and those with no substance exposure. The same children were more vulnerable to the development of neonatal abstinence syndrome at birth (Chi-Square = 51.7(2), P < .001). Newborns who were exposed primarily to alcohol in utero were at a significant risk of being born with low birth weight (Chi-Square = 8.8(2), P < .05) compared with those exposed to other types of substances. More than 50% of the mothers ceased using any substances (with the exception of tobacco) by birth, indicating that the treatment program did have an interventional effect on the mothers. The mothers' ability to either cease or decrease the use of substances during pregnancy appears to have direct positive effect on their newborns.

Read Full Article,
http://www.tandfonline.com/doi/abs/10.1080/10550887.2011.642765?url_ver=Z39.88-2003&rfr_id=ori:rid:crossref.org&rfr_dat=cr_pub%3dpubmed

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PubMed, Matern Child Health J. 2012 Feb 19.

76. A PROSPECTIVE STUDY OF PREVALENCE AND PREDICTORS OF CONCURRENT ALCOHOL AND TOBACCO USE DURING PREGNANCY

Powers JR, McDermott LJ, Loxton DJ, Chojenta CL.
Priority Research Centre for Gender, Health and Ageing, University of Newcastle, Callaghan, NSW, 2308, Australia, jenny.powers@newcastle.edu.au

ABSTRACT
Concurrent drinking and smoking during pregnancy is a major public health concern. Changes in these behaviours are under-researched, although essential if effective interventions are to be implemented. Hence this paper investigated characteristics of women who decreased concurrent drinking and smoking during pregnancy. 1,591 women were identified as pregnant at one of three surveys from 2000 to 2006 of the Australian Longitudinal Study on Women's Health and not pregnant at the previous survey. Relative risks (RRs) were calculated for concurrent drinkers and smokers before pregnancy of (1) decreasing drinking, (2) decreasing smoking and (3) decreasing drinking and smoking during pregnancy. Three hundred and fifty-four women (22%) were concurrent drinkers and smokers before pregnancy; of these women, 73% decreased drinking, 72% decreased smoking and 53% decreased drinking and smoking during pregnancy. Decreased concurrent drinking and smoking was significantly higher among women who had at least 12 years education (RRs: 1.5-1.6), who drank at least 1-2 days/week (RRs: 1.5-1.6) and who had 3 or more drinks per occasion (RRs: 1.6-1.8), and significantly lower among heavy smokers, mothers of other children (RRs: 0.8) and disadvantaged women: those stressed about money, with poor mental health, low social support and experience of partner violence (RRs: 0.6-0.7). Clearly programs are needed to tackle concurrent drinking and smoking during pregnancy. Given many pregnancies are unplanned, these programs should target drinking and smoking before and during pregnancy, as well as disadvantaged women, to reduce the deleterious effects of concurrent substance use on their babies and themselves.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22350683

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Wiley Online Library - Alcoholism: Clinical and Experimental Research
Article first published online: 10 FEB 2012
DOI: 10.1111/j.1530-0277.2011.01732.x

77. METABOLIC BIOMARKERS OF PRENATAL ALCOHOL EXPOSURE IN HUMAN EMBRYONIC STEM CELL–DERIVED NEURAL LINEAGES

Jessica A. Palmer1,2,†, Ashley M. Poenitzsch1,†, Susan M. Smith3,*, Kevin R. Conard2, Paul R. West2, Gabriela G. Cezar1,2,*
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† These authors contributed equally to this manuscript.

ABSTRACT
Background: Fetal alcohol spectrum disorders (FASD) are a leading cause of neurodevelopmental disability. The mechanisms underlying FASD are incompletely understood, and biomarkers to identify those at risk are lacking. Here, we perform metabolomic analysis of embryoid bodies and neural lineages derived from human embryonic stem (hES) cells to identify the neural secretome produced in response to ethanol (EtOH) exposure.

Methods: WA01 and WA09 hES cells were differentiated into embryoid bodies, neural progenitors, or neurons. Cells along this progression were cultured for 4 days with 0, 0.1, or 0.3% EtOH. Supernatants were subjected to C18 chromatography followed by ESI-QTOF-MS. Features were

annotated using public databases, and the identities of 4 putative biomarkers were confirmed with purified standards and comparative MS/MS.

Results: EtOH treatment induced statistically significant changes to metabolite abundance in human embryoid bodies (180 features), neural progenitors (76 features), and neurons (42 features). There were no shared significant features between different cell types. Fifteen features showed a dose–response to EtOH. Four chemical identities were confirmed: l-thyroxine, 5′-methylthioadenosine, and the tryptophan metabolites, l-kynurenine and indoleacetaldehyde. One feature with a putative annotation of succinyladenosine was significantly increased in both EtOH treatments. Additional features were selective to EtOH treatment but were not annotated in public databases.

Conclusions: EtOH exposure induces statistically significant changes to the metabolome profile of human embryoid bodies, neural progenitors, and neurons. Several of these metabolites are normally present in human serum, suggesting their usefulness as potential serum FASD biomarkers. These findings suggest the biochemical pathways that are affected by EtOH in the developing nervous system and delineate mechanisms of alcohol injury during human development.

Read Full Article,
http://onlinelibrary.wiley.com/doi/10.1111/j.1530-0277.2011.01732.x/abstract

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Wiley Online Library – Alcoholism: Clinical and Experimental Research
Article first published online: 10 FEB 2012
DOI: 10.1111/j.1530-0277.2011.01726.x

78. HOUSING IN ENVIRONMENTAL COMPLEXITY FOLLOWING WHEEL RUNNING AUGMENTS SURVIVAL OF NEWLY GENERATED HIPPOCAMPAL NEURONS IN A RAT MODEL OF BINGE ALCOHOL EXPOSURE DURING THE THIRD TRIMESTER EQUIVALENT

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2 Department of Psychology, Indiana University-Purdue University Indianapolis, Indianapolis, Indiana
3 Psychology Department, Beckman Institute, University of Illinois, Urbana, Illinois

ABSTRACT
Background: Binge-like alcohol exposure in neonatal rats during the brain growth spurt causes deficits in adult neurogenesis in the hippocampal dentate gyrus (DG). Previous data from our laboratory demonstrated that 12 days of voluntary wheel running (WR) beginning on postnatal day (PD) 30 significantly increased the number of newly generated cells evident in the DG on PD42 in both alcohol-exposed (AE) and control rats, but 30 days later a sustained beneficial effect of WR was evident only in control rats. This study tested the hypothesis that housing rats in environmental complexity (EC) following WR would promote the survival of the newly generated cells stimulated by WR, particularly in AE rats.

Methods: On PD4 to 9, pups were intubated with alcohol in a binge-like manner (5.25 g/kg/d), sham-intubated (SI), or reared normally. In Experiment 1, animals were either assigned to WR during PD30 to 42 or socially housed (SH). On PD42, animals were injected with bromodeoxyuridine (BrdU; 200 mg/kg) and perfused 2 hours later to confirm the WR-induced stimulation of proliferation. In Experiment 2, all animals received WR on PD30 to 42 and were injected with BrdU on the last full day of WR. On PD42, animals were randomly assigned either to

EC (WR/EC) or to SH (WR/SH) for 30 days and subsequently perfused and brains were processed for immunohistochemical staining to identify BrdU+-, Ki67+-, and BrdU+/NeuN+-labeled cells in DG.

Results: In Experiment 1, WR exposure significantly increased the number of proliferating cells in all 3 postnatal conditions. In Experiment 2, the AE rats given WR/SH had significantly fewer BrdU+ cells compared with control rats given WR/SH. However, WR/EC experience significantly increased the number of surviving BrdU+ cells in both the AE and SI groups compared with WR/SH rats of the same neonatal treatment. Approximately 80% of the surviving BrdU+ cells in the DG across the conditions were colabeled with NeuN.

Conclusions: WR followed by EC could provide a behavioral model for developing interventions in humans to ameliorate hippocampal-dependent impairments associated with fetal alcohol spectrum disorders.

Read Full Article,
http://onlinelibrary.wiley.com/doi/10.1111/j.1530-0277.2011.01726.x/abstract

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J Popul Ther Clin Pharmacol Vol 19(1):e41-e50; February 5, 2012

79. AN EXPLORATION OF THE EXPERIENCES AND PERSPECTIVES OF NEW ZEALANDERS WITH FETAL ALCOHOL SPECTRUM DISORDER

Jenny V Salmon, Stephen A Buetow

ABSTRACT

Background: The experiences and perspectives of New Zealanders with fetal alcohol spectrum disorder (FASD) need to be heard since no research to date has been performed. FASD, a neuro-developmental disability with life-lasting effects, is irreversible. The condition is caused by prenatal exposure to alcohol.

Objectives: This study aimed to explore and understand the daily challenges of New Zealand individuals with FASD.

Methods: Our sequential mixed methods design used two discrete but compatible qualitative methodologies – transcendental phenomenology in Phase One and classic grounded theory in Phase Two – framed by the meta-theory of pragmatism, which allows the use of 'what works' in research. One methodology alone would not have answered our research question. Using the same sample of 14 individuals, 14 to 37 years, two separate data sets were produced sequentially using face-to-face unstructured interviews. Participants had been diagnosed with either fetal alcohol syndrome (FAS) or fetal alcohol effects (FAE). Data credibility was checked using triangulation.

Results: Six themes common to the participants' experiences emerged: daily challenges in the classroom; daily challenges in the workplace; coping with mental health issues; memory problems; socialization difficulties and involvement with the law and authority. Phase Two's emerging theory revealed that because the participants perceived they had been under-supported by the social/health systems, many engaged in illegal behaviours (secondary disabilities) and experienced employment and social problems. Many disavowed having the disability, but with maturity and knowing the signs and symptoms, accepted it. They suggested ways in which their concerns could be resolved.

Conclusion: In order that progress in this field can take place, health and social agencies, educational and criminal justice systems and policy-makers need to have increased awareness of

the disability and the complex problems that individuals with the disability and their families face.

Read Full Article,
http://www.cjcp.ca/pubmed.php?articleId=356

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PubMed, J Urban Health. 2012 Feb 4.

80. PREGNANCY AND DRINKING AMONG WOMEN OFFENDERS UNDER COMMUNITY SUPERVISION IN THE UNITED STATES: 2004-2008

Sung HE.
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ABSTRACT
Drinking during pregnancy raises risks of pregnancy, labor, and delivery complications in mothers and lasting neurological or behavioral consequences in babies. This public health issue has recently attracted the attention of criminal justice (CJ) researchers, as the prevalence of Fetal Alcohol Spectrum Disorders (FASDs) appears to be unusually high among offender populations. Nevertheless, in addition to becoming a main caretaker of individuals with FASDs, the CJ system already may have under its care some of the women at the highest risk of drinking during pregnancy. This study sets out to determine the prevalence, patterns, and correlates of alcohol consumption among women offenders on probation or parole in the United States. Analysis of data collected from seven waves of the National Survey on Drug Use and Health (2004-2008) were performed on women who were under community supervision during the year prior to the survey interview. Results revealed that 1.9% of women of child-bearing ages of 12-44 years in the general population were pregnant, as compared to 4.7% of comparable women under community supervision. Pregnant offenders were more likely to come from minority groups and be socially disadvantaged than their non-CJ-involved counterparts. Alarmingly, they were nearly three times as likely to have engaged in problem drinking (e.g., two drinks a day for a month) than non-CJ-involved women. Negative behavioral consequences resulting from alcohol consumption and concurrent use of other substances were also significantly more pervasive among drinkers under community supervision. Effective prevention and control of the problem requires rethinking the role of corrections systems in health promotion. Concrete recommendations are discussed.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22311615

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PubMed, Matern Child Health J. 2012 Feb 4.

81. OLDER NOT WISER: RISK OF PRENATAL ALCOHOL USE BY MATERNAL AGE

Meschke LL, Holl J, Messelt S.
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ABSTRACT

High levels of alcohol use among pregnant women have been associated with a spectrum of birth defects. Greater maternal age has been related to an increased risk of drinking during pregnancy. Although the context, process, and outcomes of pregnancy and alcohol use vary by maternal age, no studies have examined predictors of prenatal drinking by age. This study addresses this gap by examining potential risk factors associated with prenatal alcohol use (any versus none) by

maternal age (<20, 20-25, 26-34, and 35 years or older). Descriptive and logistic regression analyses were completed on survey data from 9,004 pregnant women from the north central U.S.

Descriptive statistics revealed teens in general had a higher level or greater occurrence of risk factors previously identified with prenatal drinking compared to older women, yet women of advanced maternal age (35 years or older) were most likely to drink alcohol during pregnancy.
Based on the regression by age, 20-25 year old women had the greatest number of significant risk factors associated with prenatal drinking including being employed, white, unmarried, first birth, smoking prenatally, greater levels of depressed mood, and more experiences related to alcohol abuse.

The number and patterns of significant predictors of drinking alcohol while pregnant by age encourage greater investigation of other social, contextual factors that might contribute to the risk of prenatal drinking. This is especially salient for women of advanced maternal age, for whom very few significant predictors emerged.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22307728

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PubMed, Arch Pharm Res. 2012 Jan;35(1):171-8.
Epub 2012 Feb 2.

82. PROTECTIVE EFFECT OF [6]-GINGEROL ON THE ETHANOL-INDUCED TERATOGENESIS OF CULTURED MOUSE EMBRYOS

Yon JM, Baek IJ, Lee SR, Kim MR, Hong JT, Yong H, Lee BJ, Yun YW, Nam SY.
College of Veterinary Medicine and Research Institute of Veterinary Medicine, Chungbuk National University, Cheongju 361-763, Korea.

ABSTRACT

Excessive ethanol consumption during pregnancy causes fetal alcohol syndrome. We investigated the effect of [6]-gingerol on ethanol-induced embryotoxicity using a whole embryo culture system. The morphological changes of embryos and the gene expression patterns of the antioxidant enzymes cytosolic glutathione peroxidase (cGPx), cytoplasmic Cu/Zn superoxide dismutase (SOD1), and Mn-SOD (SOD2), and SOD activity were examined in the cultured mouse embryos exposed to ethanol (5 μL/3 mL) and/or [6]-gingerol (1×10(-8) or 1×10(-7) μg/mL) for 2 days. In ethanol-exposed embryos, the standard morphological score of embryos was significantly decreased compared with those of the control (vehicle) group.

However, cotreatment of embryos with [6]-gingerol and ethanol significantly improved all of the developmental parameters except crownrump length and head length, compared with those of the ethanol alone group. The mRNA expression levels of cGPx and SOD2, not SOD1, were decreased consistently, SOD activity were significantly decreased compared with the control group.

However, the decreases in mRNA levels of antioxidant enzymes and SOD activity were significantly restored to the control levels by [6]-gingerol supplement. These results indicate that [6]-gingerol has a protective effect against ethanol-induced teratogenicity during mouse embryogenesis.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22297756

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Oxford Journals, Toxicological Sciences
Toxicol. Sci. (2012) 127 (1): 18-28.
doi: 10.1093/toxsci/kfs068
First published online: February 1, 2012, Received October 13, 2011. Accepted January 20, 2012.

83. ETHANOL EXPOSURE INDUCES UPREGULATION OF SPECIFIC MICRORNAS IN ZEBRAFISH EMBRYOS

Ana Raquel Soares, Patrícia M. Pereira, Violeta Ferreira, Marisa Reverendo, João Simões, Ana Rita Bezerra, Gabriela R. Moura and Manuel A. S. Santos[1]
RNA Biology Laboratory, Department of Biology, CESAM, University of Aveiro, 3810-193 Aveiro, Portugal
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ABSTRACT
Prenatal exposure to ethanol leads to a myriad of developmental disorders known as fetal alcohol spectrum disorder, often characterized by growth and mental retardation, central nervous system damage, and specific craniofacial dysmorphic features. The mechanisms of ethanol toxicity are not fully understood, but exposure during development affects the expression of several genes involved in cell cycle control, apoptosis, and transcriptional regulation. MicroRNAs (miRNAs) are implicated in some of these processes, however, it is not yet clear if they are involved in ethanol-induced toxicity. In order to clarify this question, we have exposed zebrafish embryos to ethanol and evaluated whether a miRNA deregulation signature could be obtained. Zebrafish embryos were exposed to 1 and 1.5% of ethanol from 4 h postfertilization (hpf) to 24 hpf. The miRNA expression profiles obtained reveal significant miRNA deregulation and show that both ethanol concentrations upregulate miR-153a, miR-725, miR-30d, let-7k, miR-100, miR-738, and miR-732. Putative gene targets of deregulated miRNAs are involved in cell cycle control, apoptosis, and transcription, which are the main processes affected by ethanol toxicity. The conservation of affected mechanisms among vertebrates leads us to postulate that similar miRNA deregulation occurs in humans, highlighting a relevant role of miRNAs in ethanol toxicology.

Read Full Article,
http://toxsci.oxfordjournals.org/content/127/1/18.abstract

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Can J Occup Ther. 2012 Feb;79(1):60-3.

84. SENSORY PROCESSING AND ADHD IN CHILDREN WITH FETAL ALCOHOL SPECTRUM DISORDER

Abele-Webster LA, Magill-Evans JE, Pei JR.
University of Alberta, Glenrose Rehabilitation Hospital, 10230 - 111 Avenue, Edmonton, AB, T5G 0B7. polasta@shaw.ca

ABSTRACT
Background: Sensory processing problems are prevalent in children who have fetal alcohol spectrum disorder. It is unclear to what degree these problems are distinct from attention deficits as measured during the diagnostic process in these children.

Purpose: To understand sensory processing in these children, which may assist with early identification and intervention.

Method: The relationship between attention and sensory processing was studied in a retrospective sample of 26 Canadian children diagnosed with fetal alcohol spectrum disorder.

Findings: A very low correlation (r = .02) between Short Sensory Profile scores and the attention

deficit hyperactivity index of the Conners' Parent Rating Scales was found for the five- to ten-year-old children. Sensory processing problems were found in 81% of the children, similar to other studies of children with fetal alcohol spectrum disorder.

Implications: These findings can guide modifications of the environments, tasks, and approaches to children with fetal alcohol spectrum disorder.

Link to the Article,
http://www.ncbi.nlm.nih.gov/pubmed/22439293

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PubMed, Prescrire Int. 2012 Feb;21(124):49.

85. ALCOHOL DURING PREGNANCY: INFORM WOMEN, WITHOUT OVERDRAMATISING OR INDUCING FEELINGS OF GUILT
[No authors listed]

No Abstract

Link to the Article,
http://www.ncbi.nlm.nih.gov/pubmed/22413726

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PubMed, Prescrire Int. 2012 Feb;21(124):48.

86. ANTI-ALCOHOL MEDICATION AND PREGNANCY: WEIGHING THE RISKS
[No authors listed]

ABSTRACT
When there is a major risk of alcohol withdrawal symptoms in a pregnant woman, a 7-day course of benzodiazepine therapy has a positive harm-benefit balance. Medications designed to help maintain abstinence are not sufficiently effective to warrant initiating treatment during pregnancy.

Link to the Article,
http://www.ncbi.nlm.nih.gov/pubmed/22413725

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PubMed, Prescrire Int. 2012 Feb;21(124):46.

87. PREGNANCY: ANOTHER OPPORTUNITY TO RAISE THE ISSUE OF ALCOHOL
[No authors listed]

ABSTRACT
A good doctor-patient relationship allows the issue of alcohol consumption to be raised. Simple questionnaires can also help.

Link to the Article,
http://www.ncbi.nlm.nih.gov/pubmed/22413724

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PubMed, Prescrire Int. 2012 Feb;21(124):44-50.

88. PREGNANCY AND ALCOHOL: OCCASIONAL, LIGHT DRINKING MAY BE SAFE
[No authors listed]

ABSTRACT
Many pregnant women drink varying quantities of alcohol, although several guidelines recommend total abstinence. What is known of the dangers of alcohol for the outcome of pregnancy and for the unborn child? To answer this question, we conducted a review of the literature using the standard Prescrire methodology. Fetal alcohol syndrome, which combines facial dysmorphism, growth retardation and intellectual disability, occurs in about 5% of children who are regularly exposed to at least five standard units per day (about 50 g of alcohol per day). Four studies have explored the link between heavy maternal alcohol use over a short period and the risk of cognitive impairment in the child. The results were inconclusive, however, and the authors failed to take concomitant chronic alcohol consumption into account. A methodologically sound study showed an increase in neurological abnormalities (seizures and epilepsy) when the mother drank heavily during short periods between the 11th and 16th weeks of pregnancy. There is a risk of cognitive and behavioural problems in children whose mothers regularly drank more than 2 standard units per day. Studies involving a total of about 150 000 pregnancies sought a link between low-level alcohol consumption and abnormal pregnancy outcomes. Very few showed a statistically significant link, and the results are undermined by the failure to take other risk factors into account. Weekly consumption of 5 standard units or more during pregnancy has been linked to an increased risk of cryptorchidism. Studies in a total of 57 000 pregnancies showed no effect of minimal alcohol consumption on the risk of malformations. A study of 1000 pregnancies showed a statistically significant risk of major malformations, but there were several apparent biases. A link between infant mortality and alcohol consumption during pregnancy was examined in large cohort studies. Consumption of at least 4 standard units per week increased the risk of early neonatal death. Smoking further increased the risk. Daily alcohol consumption should be avoided during pregnancy. A face-to-face interview remains the best way of detecting at-risk drinking during pregnancy. Specific questionnaires (T-ACE and Tweak) can also be helpful. Women often spontaneously cut down on their drinking in early pregnancy. A clinical trial showed that women with at-risk drinking were more likely to reduce their consumption if they were informed of the risks for their pregnancy and their unborn child on several occasions than if they were simply given an information leaflet. In practice, women must be informed of the risks of alcohol consumption during pregnancy, but this must be done tactfully. The risks of minimal alcohol consumption should not be overstated.

Link to the Article,
http://www.ncbi.nlm.nih.gov/pubmed/22413723

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PubMed, J Child Neurol. 2012 Feb;27(2):258-63.

89. FETAL ALCOHOL SPECTRUM DISORDERS-- IMPLICATIONS FOR CHILD NEUROLOGY, PART 1: PRENATAL EXPOSURE AND DOSIMETRY
Paintner A, Williams AD, Burd L.

North Dakota Fetal Alcohol Syndrome Center, Department of Pediatrics, University of North Dakota School of Medicine and Health Sciences, Grand Forks, ND, USA.

ABSTRACT
In the United States, approximately 80 000 women consume ethanol through all 3 trimesters of pregnancy each year. In this article, we review prevalence rates of prenatal alcohol exposure in the United States and discuss the mechanisms of prenatal alcohol exposure and placental-umbilical effects. Cigarette smoking and delayed prenatal care are often associated with prenatal alcohol

exposure. In addition, increased risk for postnatal adversity is common, including maternal depression, foster care placement, and developmental delay. In part 2, we review prevalence rates and the diagnostic criteria for fetal alcohol spectrum disorder and the implications for child neurologists. We discuss management strategies and the importance of a long-term management plan and anticipatory management to prevent the development of secondary disabilities in fetal alcohol spectrum disorders. Child neurologists play a key role in diagnosis and the development of appropriate intervention programs for affected children and their families.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22351188

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PubMed, Behav Pharmacol. 2012 Feb;23(1):105-12.

90. MATERNAL ETHANOL CONSUMPTION BY PREGNANT GUINEA PIGS CAUSES NEUROBEHAVIORAL DEFICITS AND INCREASES ETHANOL PREFERENCE IN OFFSPRING

Shea KM, Hewitt AJ, Olmstead MC, Brien JF, Reynolds JN.
Division of Pharmacology and Toxicology, Department of Biomedical and Molecular Sciences, Queen's University, Kingston, Ontario, Canada.

ABSTRACT
The objective of this study was to test the hypothesis that prenatal exposure to ethanol, through maternal consumption of an aqueous ethanol solution, induces neurobehavioral deficits and increases ethanol preference in offspring. Pregnant Dunkin-Hartley-strain guinea pigs were given 24-h access to an aqueous ethanol solution (5%, v/v) sweetened with sucralose (1 g/l), or water sweetened with sucralose (1 g/l), throughout gestation. Spontaneous locomotor activity was measured in the offspring on postnatal day (PD) 10. The offspring underwent either ethanol preference testing using a two-bottle-choice paradigm beginning on PD 40 or Morris water maze testing using a hidden moving platform design beginning on PD 60. Maternal consumption of a 5% (v/v) ethanol solution (average daily dose of 2.3±0.1 g of ethanol/kg maternal body weight; range: 1.8-2.8 g/kg) decreased offspring birth weight, increased spontaneous locomotor activity, and increased preference for an aqueous ethanol solution. In the Morris water maze test, sucralose-exposed offspring decreased escape latency on the second day of testing, whereas the ethanol-exposed offspring showed no improvement. These data demonstrate that moderate maternal consumption of ethanol produces hyperactivity, enhances ethanol preference, and impairs learning and memory in guinea pig offspring.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22157142

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PubMed, Ross Fiziol Zh Im I M Sechenova. 2012 Feb;98(2):202-11.

91. THE EFFECT OF ETHANOL EXPOSURE IN PREGNANCY ON MATURATION OF MONOAMINERGIC SYSTEMS IN THE DEVELOPING RAT BRAIN

[No authors listed]

ABSTRACT
Simultaneous study of the main neurotransmitter of monoaminergic system of the brain, its metabolites, activity of catechol-O-methyltransferase (COMT) and the state of different subtypes of dopamine (DA) receptors in the developing brain of offspring from mothers alcoholized in gestation and feeding periods revealed a decrease in activity of all monoaminergic systems studied with reduction of noradrenaline and DA level in alcoholized fetus as well as of mPNA of COMT, an

enzyme of catecholamine metabolism, in the structures of the forebrain on the 17th day but not on 13th day of prenatal development. In parallel experiments, an increase of the contents of both long and short splice variants of D2 DA receptor was registered. In postnatal period (days 4, 10, 17), further decrease of the DA system activity was observed, particularly a reduction of DOPAC level and DOPAC/DA ratio in rat litter, mothers of whom took alcohol in the gestation period with withdrawal it after birth of offspring. The serotonin system activity was also reduced in alcoholized litter in the postnatal period and was registered in the early stages (on the 4th day of life). Therefore, the serotonin system activity is changing at early stages of development (the 4th day), whereas inhibition of the DA system activity is registered at later stages (the 10th day of life).

Link to the Article,
http://www.ncbi.nlm.nih.gov/pubmed/22650063

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PubMed, Am J Occup Ther. 2012 Jan-Feb;66(1):24-34.

92. NEUROCOGNITIVE HABILITATION THERAPY FOR CHILDREN WITH FETAL ALCOHOL SPECTRUM DISORDERS: AN ADAPTATION OF THE ALERT PROGRAM

Wells AM, Chasnoff IJ, Schmidt CA, Telford E, Schwartz LD.
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ABSTRACT
Objective: This study evaluated the effectiveness of neurocognitive habilitation, a group therapy intervention for foster and adoptive caregivers and their children who were prenatally exposed to alcohol.

Method: Participants were recruited from clients seeking evaluation for fetal alcohol syndrome (FAS) and alcohol-related neurodevelopmental disorder (ARND) and were randomly assigned to treatment and no-treatment control groups. Forty children participated in the treatment program and were compared with 38 control participants using the Behavior Rating Inventory of Executive Function (BRIEF) and the Roberts Apperception Test for Children (RATC).

Results: Significant differences between the treatment and control groups were demonstrated on the BRIEF and on the RATC, suggesting that the intervention improved executive functioning and emotional problem-solving skills.

Conclusion: These findings yield promising evidence of the effectiveness of the neurocognitive habilitation intervention in improving executive functioning and emotional problem solving in children with FAS or ARND.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22251828

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Medical Hypotheses, Volume 78, Issue 4 , Pages 489-493, April 2012
Received 18 July 2011; accepted 9 January 2012. published online 30 January 2012.

93. VITAMIN A, FOLATE, AND CHOLINE AS A POSSIBLE PREVENTIVE INTERVENTION TO FETAL ALCOHOL SYNDROME

Mark S. Ballard, Muxin Sun, Jenny Ko

ABSTRACT
It is recognized that alcohol consumption during pregnancy is associated with fetal alcohol

syndrome (FAS). Alcohol can trigger a pattern of neurodegeneration in rat brains similar to other known gamma-aminobutyric acid (GABA) specific agonists. However this does not seem to explain FAS entirely, as impoverished care-giving environments have been shown to increase the risk of FAS. Individuals living under the poverty level are at risk for micronutrient deficiencies due to insufficient intake. In particular, three nutrients commonly found to be deficient are folate, choline and vitamin A. There is evidence to suggest that ethanol alone may not explain the entire spectrum of anomalies seen in individuals with FAS. It is hypothesized that FAS may be caused more by the nutritional deficiencies that are exacerbated by alcohol than by direct alcoholic neurotoxicity.

It is known that ethanol inhibits folate, choline, and vitamin A/retinoic acid metabolism at multiple steps. Additionally, mice exposed to ethanol demonstrated epigenetic changes, or variations in the methylation of DNA to control gene expression. Folate is important in the production of methyl groups, which are subsequently used to create and methylate DNA. Choline (which is metabolized to acetylcholine) is important in neurotransmission and neurodevelopment. It is also involved in an alternative pathway in the production of methyl groups.

In fact a study by Thomas et al. in 2009 found that nutritional supplementation with choline in rats exposed to ethanol in utero almost completely mitigated the degenerative effects of ethanol on development and behaviour. Lastly, vitamin A and retinoic acid metabolism is associated with the regulation of one sixth of the entire proteome. Thus supplementation of folate, choline and vitamin A to mothers may mitigate the effects of the alcohol and reduce the severity or prevalence of FAS.

Read Full Article,
http://www.medical-hypotheses.com/article/S0306-9877(12)00025-4/abstract

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The Journal of Neuroscience, 25 January 2012, 32(4): 1377-1382; doi: 10.1523/JNEUROSCI.5520-11.2012
Received November 2, 2011.
Revision received November 29, 2011., Accepted December 9, 2011.

94. EARLY EXPOSURE TO ALCOHOL LEADS TO PERMANENT IMPAIRMENT OF DENDRITIC EXCITABILITY IN NEOCORTICAL PYRAMIDAL NEURONS

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Author contributions: A.G. and M.E.L. designed research; A.G., L.M.P., A.D.G., D.T., and M.E.L. performed research; A.G., A.D.G., D.T., and M.E.L. analyzed data; A.G., L.M.P., and M.E.L. wrote the paper.

ABSTRACT

Exposure to alcohol in utero is a well known cause of mental retardation in humans. Using experimental models of fetal alcohol spectrum disorder, it has been demonstrated that cortical pyramidal neurons and their projections are profoundly and permanently impaired. Yet, how the functional features of these cells are modified and how such modifications impact cognitive processes is still unknown. To address this, we studied the intrinsic electrophysiological properties of pyramidal neurons in young adult rats (P30–P60) exposed to ethanol inhalation during the first week of postnatal life (P2–P6). Dual whole-cell recordings from the soma and distal apical dendrites were performed and, following the injection of depolarizing current into the dendrites, layer 5 neurons from ethanol-treated (Et) animals displayed a lower number and a shorter duration of dendritic spikes, attributable to a downregulation of calcium electrogenesis. As a consequence, the mean number of action potentials recorded at the soma after dendritic current injection was also lower in Et animals. No significant differences between Et and controls were observed in the

firing pattern elicited in layer 5 neurons by steps of depolarizing somatic current, even though the firing rate was significantly lower in Et animals. The firing pattern and the firing rate of layer 2/3 neurons were not affected by alcohol exposure.

Read Full Article,
http://www.jneurosci.org/content/32/4/1377.abstract

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Wiley Online Library - Developmental Medicine & Child Neurology
Volume 54, Issue 3, pages 224–230, March 2012
Article first published online: 23 JAN 2012
DOI: 10.1111/j.1469-8749.2011.04201.x

95. HEAVY MATERNAL ALCOHOL CONSUMPTION AND CEREBRAL PALSY IN THE OFFSPRING

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ABSTRACT
Aim: The aim of this study was to investigate the association between heavy maternal alcohol consumption and pre- peri- and postneonatally acquired cerebral palsy (CP).
Method: The records of all mothers with an International Classification of Diseases, revision 9 or 10 (ICD-9/-10) alcohol-related diagnostic code, indicating heavy alcohol consumption, recorded on population-based health, mental health, and drug and alcohol data sets from 1983 to 2007, and their children were identified through the Western Australian Data-linkage System. This 'exposed' cohort was frequency matched with mothers without an alcohol-related diagnosis and their offspring (comparison group). Cases of CP were identified through linkage with the Western Australia CP Register. Analyses were undertaken using multivariate logistic regression.

Results: There were 23 573 live births in the exposed group (58.6% non-Aboriginal; 41.4% Aboriginal) and 292 cases of CP. The odds of pre/perinatally acquired CP were elevated for children of non-Aboriginal mothers with an alcohol-related diagnosis recorded during pregnancy (adjusted odds ratio 3.32; 95% confidence interval [CI] 1.30–8.48) and for Aboriginal children when an alcohol-related diagnosis was recorded up to 12 months before the mother's pregnancy (adjusted odds ratio 2.49; 95% CI 0.99–6.25). Increased odds of postneonatally acquired CP following any alcohol-related diagnosis were found for non-Aboriginal children (adjusted odds ratio 7.92; 95% CI 2.23–28.14).

Interpretation: These results suggest that heavy maternal alcohol consumption is a direct cause of pre/perinatally acquired CP, and an indirect cause of postneonatally acquired CP, in non-Aboriginal children. The lack of an association for Aboriginal children requires further investigation but may be due to under ascertainment of alcohol use disorders during pregnancy and other aetiological pathways.

Read Full Article,
http://onlinelibrary.wiley.com/doi/10.1111/j.1469-8749.2011.04201.x/abstract

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Wiley Online Library - Infant Mental Health Journal
Volume 33, Issue 1, pages 70–81, January/February 2012
Article first published online: 23 JAN 2012
DOI: 10.1002/imhj.20342

96. SUBSTANCE-ABUSING MOTHERS IN RESIDENTIAL TREATMENT WITH THEIR BABIES: IMPORTANCE OF PRE- AND POSTNATAL MATERNAL REFLECTIVE FUNCTIONING†

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† This study was supported by grants from International Psychoanalytic Association (IPA), the National Institute of Drug Abuse (NIDA, NIH), and the Finnish Medical Foundation to the corresponding author. There are no disclosures of interests.

ABSTRACT

A residential treatment program has been developed specifically for substance-abusing pregnant and parenting women in Finland, focusing on simultaneously supporting maternal abstinence from substances and the mother–baby relationship.

The aims of the study are to explore maternal pre- and postnatal reflective functioning and its association with background factors, maternal exposure to trauma, and psychiatric symptoms, postnatal interaction, child development, and later child foster care placement.
Participants were 34 mother–baby pairs living in three residential program units during the pre- to postnatal period. We employed self-report questionnaires on background, trauma history, and psychiatric symptoms (Brief Symptom Inventory: L.R. Derogatis, 1993; Edinburgh Postnatal Depression Scale: J.L. Cox, J.M. Holden, & R. Sagovsky, 1987; Traumatic Antecedents Questionnaire: B. Van der Kolk, 2003), videotaped mother–child interactions coded for sensitivity, control, and unresponsiveness (Care Index for Infants and Toddlers: P. Crittenden, 2003); a standardized test of child development (Bayley Scales of Infant Development-II: N. Bayley, 1993); and semistructured interviews for maternal reflective functioning (Pregnancy Interview: A. Slade, E. Bernbach, J. Grienenberger, D.W. Levy, & A. Locker, 2002; Parent Development Interview: A. Slade et al., 2005).

Pre- and postnatal maternal reflective functioning (RF) was on average low, but varied considerably across participants. Average RF increased significantly during the intervention. Increase in RF level was found to be associated with type of abused substance and maternal trauma history. Mothers who showed lower postnatal RF levels relapsed to substance use more often after completing a residential treatment period, and their children were more likely to be placed in foster care.

The intensive focus on maternal RF is an important direction in the development of efficacious treatment for this very high risk population.

Read Full Article,
http://onlinelibrary.wiley.com/doi/10.1002/imhj.20342/abstract

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Wiley Online Library - Developmental Medicine & Child Neurology
Volume 54, Issue 3, page 200, March 2012, Article first published online: 23 JAN 2012
DOI: 10.1111/j.1469-8749.2011.04207.x

97. ALCOHOL CONSUMPTION DURING PREGNANCY: THE GROWING EVIDENCE

Steven M Day
Mortality Research and Consulting, Newport Beach, CA, USA.

No abstract is available for this article.

Read Full Article,
http://onlinelibrary.wiley.com/doi/10.1111/j.1469-8749.2011.04207.x/abstract

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BMC Neuroscience 2012, 13:11 doi:10.1186/1471-2202-13-11
Received: 11 August 2011, Accepted: 19 January 2012, Published: 19 January 2012

98. NEUROPROTECTION WITH METFORMIN AND THYMOQUINONE AGAINST ETHANOL-INDUCED APOPTOTIC NEURODEGENERATION IN PRENATAL RAT CORTICAL NEURONS

Ikram Ullah[1], Najeeb Ullah[1,2], Muhammad Imran Naseer[1,3], Hae Young Lee[1] and Myeong OK Kim[1]*
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2 Institute of Basic Medical Sciences, Khyber Medical University, Peshawar, Khyber Pakhtoonkhwa, Pakistan
3 Center of Excellence in Genomic Medicine (CEGMR), King Abdulaziz University, Jeddah, Saudi Arabia

ABSTRACT
Background: Exposure to ethanol during early development triggers severe neuronal death by activating multiple stress pathways and causes neurological disorders, such as fetal alcohol effects or fetal alcohol syndrome. This study investigated the effect of ethanol on intracellular events that predispose developing neurons for apoptosis via calcium-mediated signaling. Although the underlying molecular mechanisms of ethanol neurotoxicity are not completely determined, mitochondrial dysfunction, altered calcium homeostasis and apoptosis-related proteins have been implicated in ethanol neurotoxicity. The present study was designed to evaluate the neuroprotective mechanisms of metformin (Met) and thymoquinone (TQ) during ethanol toxicity in rat prenatal cortical neurons at gestational day (GD) 17.5.

Results: We found that Met and TQ, separately and synergistically, increased cell viability after ethanol (100 mM) exposure for 12 hours and attenuated the elevation of cytosolic free calcium [Ca2+]c. Furthermore, Met and TQ maintained normal physiological mitochondrial transmembrane potential (ΔψM), which is typically lowered by ethanol exposure. Increased cytosolic free [Ca2+]c and lowered mitochondrial transmembrane potential after ethanol exposure significantly decreased the expression of a key anti-apoptotic protein (Bcl-2), increased expression of Bax, and stimulated the release of cytochrome-c from mitochondria. Met and TQ treatment inhibited the apoptotic cascade by increasing Bcl-2 expression. These compounds also repressed the activation of caspase-9 and caspase-3 and reduced the cleavage of PARP-1. Morphological conformation of cell death was assessed by TUNEL, Fluoro-Jade-B, and PI staining. These staining methods demonstrated more cell death after ethanol treatment, while Met, TQ or Met plus TQ prevented ethanol-induced apoptotic cell death.

Conclusion: These findings suggested that Met and TQ are strong protective agents against ethanol-induced neuronal apoptosis in primary rat cortical neurons. The collective data

demonstrated that Met and TQ have the potential to ameliorate ethanol neurotoxicity and revealed a possible protective target mechanism for the damaging effects of ethanol during early brain development.

Read Full Article,
http://www.biomedcentral.com/1471-2202/13/11

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PubMed, Eur J Paediatr Neurol. 2012 Jan 18

99. NEUROPHYSIOLOGY OF CIRCADIAN RHYTHM SLEEP DISORDERS OF CHILDREN WITH NEURODEVELOPMENTAL DISABILITIES

Jan JE, Bax MC, Owens JA, Ipsiroglu OS, Wasdell MB.
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ABSTRACT
This article reviews circadian rhythm sleep disorders (CRSDs) of children with neurodevelopmental disabilities. These sleep disturbances frequently occur in this population but they are misunderstood and under diagnosed. The causes and features of CRSD in children with brain disorders differ in many ways from those seen in typically developing children. It is the brain, including the eyes, which regulates sleep and circadian rhythmicity by modulating pineal melatonin production/secretion and when there is significant brain damage, the sleep/wake patterns may be modified. In most instances CRSD are not disorders of the suprachiasmatic nuclei because these small hypothalamic structures only adjust their functions to the changing photic and non-photic modulatory influences. Each form of CRSD is accompanied by characteristic changes in serum melatonin levels and clinical features. When nocturnal melatonin production/secretion is inappropriately timed or impaired in relation to the environment, timed melatonin replacement therapy will often be beneficial. In this review an attempt is made to clarify the neurophysiological mechanisms underlying the various forms of CRSD because without understanding the photic and non-photic influences on sleep, these sleep disorders can not be fully characterized, defined or even appropriately treated. In the future, the existing definitions for the different forms of CRSD should be modified by experts in pediatric sleep medicine in order to include children with neurodevelopmental disabilities.

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http://www.ncbi.nlm.nih.gov/pubmed/22264650

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Wiley Online Library - Alcoholism: Clinical and Experimental Research
Volume 36, Issue 4, pages 670–676, April 2012, Article first published online: 17 JAN 2012
DOI: 10.1111/j.1530-0277.2011.01664.x

100. PRENATAL ALCOHOL EXPOSURE PATTERNS AND ALCOHOL-RELATED BIRTH DEFECTS AND GROWTH DEFICIENCIES: A PROSPECTIVE STUDY

Haruna Sawada Feldman, Kenneth Lyons Jones, Suzanne Lindsay, Donald Slymen, Hillary Klonoff-Cohen, Kelly Kao, Smriti Rao, Christina Chambers
From the Department of Pediatrics (HSF, KLJ, KK, SR, CC), University of California, San Diego, La Jolla, California; Graduate School of Public Health (SL, DS), San Diego State University, San Diego, La Jolla, California; and Department of Family and Preventative Medicine (HK-C, CC), University of California, San Diego, La Jolla, California.

ABSTRACT
Background: The physical features of fetal alcohol syndrome include smooth philtrum, thin

vermillion border, short palpebral fissures, microcephaly, and growth deficiencies on weight and height. However, little is known about the specific quantities of alcohol exposure, pattern of drinking, timing of exposure, and magnitude of risk for each of these features.

Methods: Using data on 992 subjects collected prospectively in California between 1978 and 2005, we examined the patterns and timing of alcohol exposure in relation to these features. Structural features were assessed by a dysmorphologist who performed a blinded physical examination of all infants. Patterns of drinking were evaluated by drinks per day, number of binge episodes, and maximum number of drinks. Timing of exposure was evaluated 0 to 6 weeks postconception, 6 to 12 weeks postconception, first trimester, second trimester, and third trimester.

Results: Higher prenatal alcohol exposure in every pattern was significantly associated with the incidence of smooth philtrum but not with short palpebral fissures. The strongest associations were with timing of exposure in the second half of the first trimester (RR 1.25, 95% CI 1.14 to 1.36 for average number of drinks per day; RR 1.17, 95% CI 1.09 to 1.26 for maximum number of drinks in 1 episode). Similarly, thin vermillion border was most strongly associated with exposure in the second half of the first trimester. Findings with respect to timing of exposure were similar for microcephaly and reduced birth weight. However, reduced birth length was increased with exposure in any trimester. These associations were linear, and there was no evidence of a threshold.

Conclusions: Reduced birth length and weight, microcephaly, smooth philtrum, and thin vermillion border are associated with specific gestational timing of prenatal alcohol exposure and are dose-related without evidence of a threshold. Women should continue to be advised to abstain from alcohol consumption from conception throughout pregnancy.

Read Full Article,
http://onlinelibrary.wiley.com/doi/10.1111/j.1530-0277.2011.01664.x/abstract

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American Journal of Obstetrics & Gynecology
Volume 206, Issue 4 , Pages 358.e19-358.e22, April 2012,
Received 27 September 2011; received in revised form 8 December 2011; accepted 6 January 2012. published online 16 January 2012.

101. THE EXPRESSION OF ANTIOXIDANT ENZYMES IN A MOUSE MODEL OF FETAL ALCOHOL SYNDROME

Presented orally at the SMFM 2011 annual meeting, San Francisco, CA, Feb. 11, 2011.
Nathan Drever, MD, Huaizhi Yin, MS, Talar Kechichian, BS, MS, Maged Costantine, MD, Monica Longo, MD, PhD, George R. Saade, MD, Egle Bytautiene, MD, PhD
Department of Obstetrics and Gynecology, Division of Maternal-Fetal Medicine, University of Texas Medical Branch, Galveston, TX

ABSTRACT
Objective: Superoxide dismutase, glutathione peroxidase, and catalase prevent cellular damage produced by free radicals. Our objective was to evaluate if prenatal alcohol exposure decreased the expression of antioxidant enzymes in the brain, liver, or placenta of fetal mice.

Study Design: Timed, pregnant C57BL6/J mice were treated on gestational day 8 with intraperitoneal injection of alcohol (0.03 mL/g) or saline (control). Fetuses were harvested on gestational day 18. Fetal brain, liver, and placenta were analyzed for mRNA expression of superoxide dismutase, glutathione peroxidase, and catalase by real-time polymerase chain reaction, with 18S RNA used as reference.

Results: Superoxide dismutase, glutathione peroxidase, and catalase expression was lower in fetal brains exposed to alcohol with no differences detected in the liver or placenta between the 2

groups.

Conclusion: Maternal alcohol consumption causes a decrease in superoxide dismutase, glutathione peroxidase, and catalase expression in the fetal brain. This may explain the long-term neurologic findings in fetal alcohol syndrome.

Read Full Article,

http://www.ajog.org/article/S0002-9378(12)00029-4/abstract

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PubMed, J Pharm Biomed Anal. 2012 Jan 16.

102. DETERMINATION OF MATERNAL-FETAL BIOMARKERS OF PRENATAL EXPOSURE TO ETHANOL: A REVIEW

Joya X, Friguls B, Ortigosa S, Papaseit E, Martínez SE, Manich A, Garcia-Algar O, Pacifici R, Vall O, Pichini S.

Unitat de Recerca Infància i Entorn (URIE), IMIM - Parc de Salut Mar, Barcelona, Spain; Red SAMID, RETIC Instituto Carlos III, Madrid, Spain; Departament de Bioquímica i Biologia Molecular, Universitat Autònoma Barcelona, Bellaterra, Spain.

ABSTRACT

The deleterious effects exerted by prenatal ethanol exposure include physical, mental, behavioural and/or learning disabilities that are included in the term fetal alcohol spectrum disorder (FASD). Objective assessment of exposure to ethanol at both prenatal and postnatal stages is essential for early prevention and intervention. Since pregnant women tend to underreport alcohol drinking by questionnaires, a number of biological markers have been proposed and evaluated for their capability to highlight gestational drinking behaviour.

These biomarkers include classical biomarkers (albeit indirect) of alcohol-induced pathology (mean corpuscular volume (MCV), gamma glutamyltransferase (GGT), aspartate aminotransferase (AST) and alanine aminotransferase (ALT)) acetaldehyde-derived conjugates, and finally derivatives of non-oxidative ethanol metabolism (fatty acid ethyl esters (FAEEs), ethyl glucuronide (EtG), ethyl sulphate (EtS) and phosphaditylethanol (PEth)). Since ethanol itself and acetaldehyde are only measured few hours after ethanol intake in conventional matrices such as blood, urine and sweat, they are only useful to detect recent ethanol exposure.

In the past few years, the non-oxidative ethanol metabolites have received increasing attention because of their specificity and in some case wide time-window of detection in non-conventional matrices from the pregnant mother (oral fluid and hair) and fetus-newborn (neonatal hair, meconium, placenta and umbilical cord). This article reviews bioanalytical procedures for the determination of these markers of ethanol consumption during pregnancy and related prenatal exposure.

In addition, clinical toxicological applications of these procedures are presented and discussed.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22300909

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Wiley Online Library - Drug and Alcohol Review
Special Issue: Low Risk Drinking Guidelines. Guest Editors: Tim Stockwell and Robin Room
Volume 31, Issue 2, pages 162–169, March 2012, Article first published online: 12 JAN 2012
DOI: 10.1111/j.1465-3362.2011.00413.x

103. RESPONSES TO RISK: PUBLIC SUBMISSIONS ON AUSTRALIAN ALCOHOL GUIDELINES FOR LOW-RISK DRINKING

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ABSTRACT
Introduction and Aims: In 2007 the National Health and Medical Research Council issued the draft of the revised Australian alcohol guidelines. The document presented guidelines explicitly in terms of risk. This paper seeks to explore the public response to this document by analysing the submissions received during the 60 day period for public feedback.

Methods: One-hundred and three submissions were reviewed. Considerations of what interests were reflected in submissions and how interest groups responded to the framing of risk were examined.

Results: Submissions were received from individuals and organisations. Analysis revealed a range of views and rhetoric. Temperance interests wanted the guidelines' thresholds to be lower; the industry critiqued the evidence base as flawed and also argued that the public was unlikely to listen to low-risk drinking messages; submissions from public health groups and government wanted a greater rationale for the guidelines and were also concerned with the dropping of a second differentiation of a higher-risk level; personal testimonies supported the risk assessments based on personal experiences; and those working in clinical service provision expressed concern about the reception of the guidelines among client groups.

Discussion and Conclusions:The diversity of views expressed seems to have had little effect in the revision of the guidelines. Disseminating the low-risk drinking guidelines message poses many challenges. [Wilkinson C. Responses to risk: Public submissions on Australian alcohol guidelines for low-risk drinking.

Read Full Article,
http://onlinelibrary.wiley.com/doi/10.1111/j.1465-3362.2011.00413.x/abstract

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PubMed, J Child Neurol. 2012 Mar;27(3):355-62. Epub 2012 Jan 12.

104. FETAL ALCOHOL SPECTRUM DISORDERS--IMPLICATIONS FOR CHILD NEUROLOGY, PART 2: DIAGNOSIS AND MANAGEMENT

Paintner A, Williams AD, Burd L.
North Dakota Fetal Alcohol Syndrome Center, Department of Pediatrics, University of North Dakota School of Medicine and Health Sciences, Grand Forks, ND 58203-9037, USA.

ABSTRACT
In part 1, we discussed the mechanism of alcohol exposure, dosimetry, and the teratogenic

pathways of damage to the fetus. In part 2, we review the diagnosis of fetal alcohol spectrum disorders and the developmental implications of prenatal alcohol exposure. Fetal alcohol spectrum disorders are associated with increased rates of mental retardation, seizure disorders, brain malformations, and premature mortality. The risk of comorbid disorders is increased among this population, which enhances phenotype severity and complexity of management. Recurrence rates are high and younger siblings tend to be more severely affected. Detection of prenatal alcohol use warrants substance abuse intervention, which can avoid exposure in subsequent pregnancies. Fetal alcohol spectrum disorders are common developmental disorders with a phenotype that is influenced by both age and development and require long-term management. Child neurologists are essential in the diagnosis and management of fetal alcohol spectrum disorders.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22241713

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Oxford Journals - Alcohol and Alcoholism (March/April 2012) 47 (2): 109-117.
doi: 10.1093/alcalc/agr166
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105. PLACENTAL HYPOXIA AND FOETAL DEVELOPMENT VERSUS ALCOHOL EXPOSURE IN PREGNANCY

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ABSTRACT

Aims: To examine the causes of variability in the effect of maternal drinking on the foetus, with particular reference to the pattern, frequency and duration of the period of drinking, differences in maternal, foetal and placental metabolism of ethanol/acetaldehyde, and genetic factors.

Methods: Narrative review of published studies of the pathogenesis of foetal alcohol syndrome (FAS) with emphasis in the development of the central nervous system.

Results: Animal models suggest that acetaldehyde, the primary hepatic oxidative metabolite of ethanol, reaches the foetus either by placental production or by placental transference, which in turn could affect foetal growth and development. The most likely hypothesis regarding the decrease of foetal growth is via hypoxia and increased oxidative/nitrative stress, which interfere with cellular processes that require oxygen in order to function adequately, such as placental transport.

Conclusion: There seems to be an association between the teratogenic effect, hypoxia and oxidative stress, the molecular mechanism involved (e.g. apoptosis) and the range of effects. The review sums ups the evidence that could explain some of the abnormalities in the brain development that could be related to behavioural problems observed in individuals with FAS/foetal alcohol spectrum disorder. This suggests that alcohol consumption produces failures in the normal migration of radial cells, from which the rest of the brain cells would eventually develop.

Read Full Article,
http://alcalc.oxfordjournals.org/content/47/2/109.abstract

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J Popul Ther Clin Pharmacol Vol 19 (1):e26-e31; January 10, 2012

106. CLINICAL USE OF MECONIUM FATTY ACID ETHYL ESTERS FOR IDENTIFYING CHILDREN AT RISK FOR ALCOHOL-RELATED DISABILITIES: THE FIRST REPORTED CASE

Irene Zelner, Sarit Shor, Hazel Lynn, Henry Roukema, Lisa Lum, Kirsten Eisinga, Gideon Koren

ABSTRACT

Fatty acid ethyl esters (FAEEs) in meconium are validated biomarkers of heavy fetal alcohol exposure that may potentially be used clinically for identifying children at risk for alcohol-related disabilities. However, until now, FAEEs have been largely used anonymously in epidemiological studies, and by child protection authorities in need for verification of heavy alcohol use in pregnancy. Here we describe the first case of a neonate identified as part of a research study on a pilot neonatal screening program for prenatal alcohol exposure. The neonate's meconium tested high for FAEEs (52 nmol/g; positive cut-off ≥2 nmol/g), which prompted active follow-up of the infant's development, identifying early neurocognitive problems and allowing initiation of a remedial program.

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http://www.cjcp.ca/pubmed.php?articleId=354

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J Popul Ther Clin Pharmacol Vol 19(1):e32-e40; January 10, 2012

107. ALCOHOL -INDUCED BEHAVIOURAL PROBLEMS IN FETAL ALCOHOL SPECTRUM DISORDER VERSUS CONFOUNDING BEHAVIOURAL PROBLEMS

Marion Malone, Gideon Koren

ABSTRACT

Prenatal alcohol exposure is strongly associated with disruptive behaviour in childhood and antisocial behaviour later in life. There are numerous confounding risk factors in the lives of alcohol-abusing mothers that may contribute to the behaviour problems seen in their children, rather than direct brain injury by alcohol. In fact, many of these additional environmental and genetic risk factors for childhood behaviour problems co-occur with prenatal alcohol exposure and affect the same child, creating a confluence of risk. As a result, one cannot with any certainty attribute behaviour problems in an individual child to prenatal alcohol exposure. This has important clinical and legal implications.

Read Full Article,
http://www.cjcp.ca/pubmed.php?articleId=355

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PubMed, Int J Epidemiol. 2012 Apr;41(2):405-413. Epub 2012 Jan 9.

108. MODERATE ALCOHOL INTAKE DURING PREGNANCY AND RISK OF FETAL DEATH.

Andersen AM, Andersen PK, Olsen J, Grønbæk M, Strandberg-Larsen K.
Section of Social Medicine, Department of Public Health, University of Copenhagen, Oster Farimagsgade 5, DK-1014 Copenhagen K, Denmark, Section of Biostatistics, Department of Public Health, University of Copenhagen, Oster Farimagsgade 5, DK-1014 Copenhagen K, Denmark, Unit of Epidemiology, Institute of Public Health, University of Aarhus, Denmark and Centre for Alcohol Research, National Institute of Public Health, University of Southern Denmark, Øster Farimagsgade 5A, DK-1399 Copenhagen K, Denmark.

ABSTRACT

Background: Controversies still exist regarding the existence of a 'safe' level of alcohol intake

during pregnancy. The aim of this study was to assess the risk of fetal death (spontaneous abortion and stillbirth) according to maternal alcohol consumption in a large Danish pregnancy cohort.

Methods: A cohort study carried out within the framework of the Danish National Birth Cohort. A total of the 92 719 participants enrolled in the Danish National Birth Cohort who provided information about lifestyle during first trimester of pregnancy were included in the study. Information about average weekly consumption of alcohol during pregnancy, smoking, coffee drinking, occupational status and reproductive history were obtained by means of computer-assisted telephone interviews. Pregnancy outcomes (spontaneous abortion, stillbirth, live birth and other pregnancy outcome) and gestational age at end of pregnancy were obtained through register linkage with the Civil Registration System and the National Discharge Registry.

Data were analysed using Cox regression models, taking the varying gestational age at recruitment and time-dependent co-variables into account.

Results: Fifty-five per cent of the participants abstained from alcohol drinking during pregnancy and only 2.2% reported four or more drinks per week. The adjusted hazard ratios for fetal death in first trimester were 1.66 [95% confidence interval (CI) 1.43-1.92] and 2.82 (95% CI 2.27-3.49) for women who reported 2-3½ drinks per week and 4 or more drinks per week, respectively, and 1.57 (95% CI 1.30-1.90) and 1.73 (95% CI 1.24-2.41) for fetal death during pregnancy weeks 13-16. No increased risk was found for fetal death after 16 weeks of pregnancy.

Conclusions: Even low amounts of alcohol consumption during early pregnancy increased the risk of spontaneous abortion substantially. The results indicate that the fetus is particularly susceptible to alcohol exposure early in pregnancy.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22253313

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Neuroscience. 2012 Mar 29;206:122-35. Epub 2012 Jan 8.

109. LITHIUM PREVENTS LONG-TERM NEURAL AND BEHAVIORAL PATHOLOGY INDUCED BY EARLY ALCOHOL EXPOSURE

Sadrian B, Subbanna S, Wilson DA, Basavarajappa BS, Saito M.
Emotional Brain Institute, Nathan S. Kline Institute for Psychiatric Research, 140 Old Orangeburg Road, Orangeburg, NY 10962, USA. Benjamin.Sadrian@nyumc.org

ABSTRACT
Fetal alcohol exposure can cause developmental defects in offspring known as fetal alcohol spectrum disorder (FASD). FASD symptoms range from obvious facial deformities to changes in neuroanatomy and neurophysiology that disrupt normal brain function and behavior. Ethanol exposure at postnatal day 7 in C57BL/6 mice induces neuronal cell death and long-lasting neurobehavioral dysfunction. Previous work has demonstrated that early ethanol exposure impairs spatial memory task performance into adulthood and perturbs local and interregional brain circuit integrity in the olfacto-hippocampal pathway. Here we pursue these findings to examine whether lithium prevents anatomical, neurophysiological, and behavioral pathologies that result from early ethanol exposure. Lithium has neuroprotective properties that have been shown to prevent ethanol-induced apoptosis. Here we show that mice co-treated with lithium on the same day as ethanol exposure exhibit dramatically reduced acute neurodegeneration in the hippocampus and retain hippocampal-dependent spatial memory as adults. Lithium co-treatment also blocked ethanol-induced disruption in synaptic plasticity in slice recordings of hippocampal CA1 in the adult mouse brain. Moreover, long-lasting dysfunctions caused by ethanol in olfacto-hippocampal networks, including sensory-evoked oscillations and resting state coherence, were prevented in

mice co-treated with lithium. Together, these results provide behavioral and physiological evidence that lithium is capable of preventing or reducing immediate and long-term deleterious consequences of early ethanol exposure on brain function.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22266347

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PubMed, J Addict Dis. 2012 Jan;31(1):29-44. doi: 10.1080/10550887.2011.642766.

110. COGNITIVE AND SOCIAL DEVELOPMENT IN PRESCHOOL CHILDREN BORN TO WOMEN USING SUBSTANCES

Irner TB, Teasdale TW, Olofsson M.
The Family Center, Department of Obstetrics and Gynaecology, Copenhagen University Hospital, Hvidovre, Hvidovre, Denmark. tina.irner@psy.ku.dk

ABSTRACT
Prenatal substance exposure is associated with physical birth defects and increased risk of regulatory and neuropsychological difficulties of children born to mothers using substances while pregnant. Myriad factors, such as maternal psychopathology, stress, and poor living circumstances, may influence childhood development in addition to the teratological effect of prenatal substance exposure. This study explores the long-term developmental consequences in children from birth to age 7 born to women using substances and are in treatment. A series of t tests were performed to explore group effects on the cognitive and social dimensions of Griffiths Mental Development Scales compared with Swedish norms. The results showed significant effects on eye and hand coordination in children aged birth to 7 years and on hearing and speech, practical reasoning, and the general quotient in children aged 3 to 7 years. Children who were exposed primarily to alcohol in utero scored significantly lower on the personal and social skills subscale, eye and hand coordination subscale, and the general quotient than children exposed primarily to substances other than alcohol. These effects did not appear to be mediated by the mothers' social background or treatment history. The results suggest that children who are exposed to substances, in particular alcohol, in utero are vulnerable overall, but especially in eye and hand coordination and personal and social skills.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22356667

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PubMed, Zhongguo Ying Yong Sheng Li Xue Za Zhi. 2012 Jan;28(1):13-4, 83.

111. EFFECT OF ALCOHOL EXPOSURE DURING PREGNANCY ON LEARNING AND MEMORY AND EXPRESSION OF CDK5 IN THE HIPPOCAMPUS OF INFANT RATS

Li S, Xu CY, Jiang HB, Hao W, Zhang RL.

ABSTRACT

No Abstract Available

Link to the Article,
http://www.ncbi.nlm.nih.gov/pubmed/22493884

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American Journal of Obstetrics & Gynecology, Volume 206, Issue 1, Supplement, Page S262, January 2012

112. PRENATAL RISK-DRINKING SCREENING: T-ACER3 REDUCES T-ACE FALSE POSITIVES

Lisa Chiodo, Robert Sokol, John Hannigan, James Janisse, Grace Patterson, Virginia Delaney-Black

ABSTRACT

Objective: Preventing Fetal Alcohol Spectrum Disorders (FASD) depends on obstetricians detecting maternal risk drinking during antenatal care. ACOG and NIAAA recommend using the T-ACE, an economical/sensitive screen. We have reported that a more stringent T-ACE total score cut-point (3 vs. 2) increased specificity in identifying maternal risk drinking & alcohol-related neurobehavioral dysfunction in children (Chiodo et al, 2010). Our aim was to assess how increasing the T-ACE cut-point could increase efficiency of clinical practice.

Study Design: Self-reported peri-conceptional & in-pregnancy drinking were assessed with semi-structured interviews and alcohol screens in a prospective sub-sample of 239 African-American mothers given an in-pregnancy T-ACE. The original T-ACE risk criterion (total score of 2) and the revised T-ACER3 criterion with a total score of 3 were analyzed. ANOVA and post-hoc comparisons compared pre- and in-pregnancy alcohol consumption quantity and frequency measures by risk category. Categories were: 1) No Risk Group (NRG) = no risk for pregnancy alcohol use based on both T-ACE and T-ACER3 criteria (n=140); 2) At Risk Group (ARG) based on both T-ACE and T-ACER3 criteria (n=28); and 3) Change Risk Group (CRG) identified as at-risk with the original T-ACE criterion but not at-risk using the revised T-ACER3 criterion (n=71).

Results: The 71 women (30%) in the Change Risk Group (CRG) had patterns of alcohol use similar to the no risk group (NRG). As predicted, post-hoc analysis revealed that the at-risk group (ARG) had significantly more alcohol use both prior to and during pregnancy than either the NRG or CRG groups.

Conclusion: The results provide further evidence that adjusting the T-ACE total score cut-point to 3 in the T-ACER3 is clinically appropriate. Using the T-ACER3 criteria, only 12% of women would require intervention, compared with 41% for T-ACE. Increasing the total T-ACE score criterion from 2 to 3 results in fewer “false positives,” allowing a more intensive targeted clinical response with pregnant women correctly identified by the T-ACER3 as drinking at fetal risk levels.

Read Full Article,
http://www.ajog.org/article/S0002-9378(11)01883-7/fulltext

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American Journal of Obstetrics & Gynecology
Volume 206, Issue 1, Supplement, Page S28, January 2012

113. THE ROLE OF CELL SIGNALING MECHANISMS IN THE DOWNREGULATION OF ANTIOXIDANT ENZYMES IN THE FETAL BRAIN IN A MOUSE MODEL OF FETAL ALCOHOL SYNDROME

Nathan Drever, Talar Kechichian, Nkechi Okonkwo, Debdeep Banerjee, Monica Longo, George Saade, Egle Bytautiene

ABSTRACT

Objective: Fetal alcohol syndrome (FAS) is the most common non genetic cause of mental retardation. Prenatal alcohol exposure decreases antioxidant enzymes in the fetal brain in a mouse model of fetal alcohol syndrome. Exposure to alcohol is known to decrease expression of Glycogen Synthase Kinase-3β (GSK-3β) a serine threonine kinase that regulates glycogen

synthesis through phosphorylation and inactivation of glycogen synthase. In addition to regulation of glycogen, GSK-3β is positively correlated with expression of nuclear transcription factors, NF-κβ and Nrf2, which are inactive in the cytosol and active within nucleus. Our objective was to evaluate if the effect of prenatal alcohol exposure on fetal brain antioxidants can be explained by an effect on GSK-3β, NF-κβ and Nrf2.

Study Design: A well-characterized FAS model was used (Webster, 1980). Timed, pregnant C57BL6/J mice were treated on gestational day 8 (E8) with alcohol (0.03 mL/g) or vehicle. Fetuses were harvested on gestational day 18 (E18), and the brain prepared for protein analysis. Western Blots were performed assessing the protein expression of GSK-3β and cytosolic and nuclear expression of NF-κβ and Nrf2 normalized to Glyceraldehyde 3-phosphate dehydrogenase (GAPDH). Statistical analysis was performed using Student t-test (significance: $p<0.05$).

Results: GSK-3β protein levels were significantly lower (0.73 vs 1.06 ratio protein/GAPDH; $p=0.01$) and cytosolic Nrf2 protein levels were significantly higher (0.72 vs 0.43 ratio protein/GAPDH; $p=0.01$) in the brain of pups exposed to alcohol compared with control. There was no significant difference in cytosolic expression of NF-κβ or nuclear expression of NF-κβ and Nrf2 between the two groups.

Conclusion: In utero exposure to alcohol reduces GSK-3β and increases cytosolic Nrf2 protein levels in the fetal brain. These findings provide a mechanism for the decrease in antioxidants in the brain of FAS offspring, and a potential target for preventive strategies.

Read Full Article,
http://www.ajog.org/article/S0002-9378(11)01359-7/fulltext

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American Journal of Obstetrics & Gynecology, Volume 206, Issue 1, Supplement, Page S199, January 2012

114. PROTEIN EXPRESSION AND ENZYMATIC ACTIVITY OF SUPEROXIDE DISMUTASE IN THE FETAL BRAIN IN A MOUSE MODEL OF FETAL ALCOHOL SYNDROME

Nathan Drever, Talar Kechichian, Nkechi Okonkwo, Debdeep Banerjee, Monica Longo, George Saade, Egle Bytautiene,

ABSTRACT

Objective: Fetal alcohol syndrome (FAS) is the most common non genetic cause of mental retardation. Prenatal alcohol exposure decreases the fetal brain mRNA expression of the antioxidant enzyme, superoxide dismutase (SOD). Our objective was to elucidate the role of oxidant/antioxidant mechanisms in the etiology of FAS by determining whether decrease in SOD mRNA expression is associated with reduced protein expression and/or enzymatic activity.

Study Design: A well-characterized FAS model was used (Webster, 1980). Timed, pregnant C57BL6/J mice were treated on gestational day 8 (E8) with alcohol (0.03 mL/g) or vehicle. Fetuses were harvested on gestational day 18 (E18) and their brain processed for protein analysis and enzymatic activity. Western Blots were performed assessing protein expression of SOD normalized to Glyceraldehyde 3-phosphate dehydrogenase (GAPDH). SOD activity was measured using an assay based on inhibition of XTT reduction by superoxide. The rate of superoxide dismutation was calculated by the reciprocal rate of formazan formation as measured calorimetrically at an absorbance of 490nm over 30 minutes. Statistical analysis was performed using Student t-test (significance: $p<0.05$).

Results: Fetal alcohol exposure caused a significant increase in protein expression (0.63 vs 0.27 ratio protein/GAPDH; $p=0.02$) and no difference in enzymatic activity of SOD (31.9 vs. 32.3 arbitrary units; $p>0.10$) at E18 when compared to vehicle treated controls.

Conclusion: The previously reported decrease in fetal brain SOD mRNA expression in FAS is not associated with a decrease in protein expression, nor with altered SOD activity. In fetuses exposed to alcohol, increased SOD protein expression is not accompanied by increased activity of SOD. These findings suggest that post-transcriptional mechanisms contribute to the preservation of SOD protein expression and activity in a mouse model of fetal alcohol syndrome.

Read Full Article,
http://www.ajog.org/article/S0002-9378(11)01739-X/fulltext

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Lippincott Williams and Wilkins – Obstetrics and Gynecology
January 2012 - Volume 119 - Issue 1 - p 102–110
doi: 10.1097/AOG.0b013e31823d427d

115. EARLY START: A COST–BENEFICIAL PERINATAL SUBSTANCE ABUSE PROGRAM

Goler, Nancy C. MD; Armstrong, Mary Anne MA; Osejo, Veronica M. BS; Hung, Yun-Yi PhD; Haimowitz, Monica LCSW; Caughey, Aaron B. MD, PhD

ABSTRACT

Objective: To conduct a cost–benefit analysis of Early Start, an integrated prenatal intervention program for stopping substance use in pregnancy.

Methods: A retrospective cohort study was conducted of 49,261 women who had completed prenatal substance abuse screening questionnaires at obstetric clinics and who had undergone urine toxicology screening tests. Four study groups were compared: women screened and assessed positive and followed by Early Start (screened-assessed-followed, n=2,032), women screened and assessed positive without follow-up (screened-assessed, n=1,181), women screened positive only (screened-positive-only, n=149), women in the control group who screened negative (control, n=45,899). Costs associated with maternal health care (prenatal through 1 year postpartum), neonatal birth hospitalization care, and pediatric health care (through 1 year) were adjusted to 2009 dollars. Mean costs were calculated and adjusted for age, race, education, income, marital status, and amount of prenatal care.

Results: Screened-positive-only group adjusted mean maternal total costs ($10,869) were significantly higher than screened-assessed-followed, screened-assessed, and control groups ($9,430; $9,230; $8,282; all P<.001). Screened-positive-only group adjusted mean infant total costs ($16,943) were significantly higher than screened-assessed-followed, screened-assessed, and control groups ($11,214; $11,304; $10,416; all P<.001).

Screened-positive-only group adjusted mean overall total costs ($27,812) were significantly higher than screened-assessed-followed, screened-assessed, and control groups ($20,644; $20,534; $18,698; all P<.001). Early Start implementation costs were $670,600 annually. Cost–benefit analysis showed that the net cost benefit averaged $5,946,741 per year.

Conclusion: Early Start is a cost-beneficial intervention for substance use in pregnancy that improves maternal–infant outcomes and leads to lower overall costs by an amount significantly greater than the costs of the program.

Level Of Evidence: II

Read Full Artilce,
http://journals.lww.com/greenjournal/Abstract/2012/01000/Early_Start__A_Cost_Beneficial_Perinatal_Substance.15.aspx

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PubMed, Zh Nevrol Psikhiatr Im S S Korsakova. 2012;112(1):60-67.

116. SYNAPTOGENESIS AND FORMATION OF BENZODIAZEPINE RECEPTORS IN THE HUMAN BRAIN IN CONDITIONS OF PRENATAL ALCOHOLIZATION

Shushpanova TV, Solonskiĭ AV.
Laboratoriia neĭrobiologii NII psikhicheskogo zdorov'ia Sibirskogo otdeleniia RAMN, Tomsk.

ABSTRACT

The aim was to study correlations between the development of synaptic connections and benzodiazepine receptors functionally linked to the brain GABA system in the brain of embryos and 8-15 week fetuses obtained from women with alcoholism.

Material from 33 women with alcoholism, stage II (ICD-10 F10.201 and F10.202), and 30 healthy people (controls) was studied. The retardation in the formation of synaptic benzodiazepine receptors and increase in their density was seen in brain cells developing in conditions of prenatal alcoholization compared to controls. The authors consider these findings as a manifestation of compensatory reactions directed towards the adaptation of the fetal nervous system to the action of alcohol and as functional insufficiency of the brain GABA system.

Link to the Article,
http://www.ncbi.nlm.nih.gov/pubmed/22678678

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ScienceDIrect, Experimental Neurology, Volume 234, Issue 1, March 2012, Pages 127–135
Received 9 August 2011. Revised 25 November 2011. Accepted 14 December 2011. Available online 29 December 2011.
http://dx.doi.org/10.1016/j.expneurol.2011.12.022

117. HUMAN EMBRYONIC STEM CELL MODEL OF ETHANOL-MEDIATED EARLY DEVELOPMENTAL TOXICITY

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ABSTRACT

Background: Fetal alcohol syndrome is an important clinical problem. Human embryonic stem cells (hESC) have not been widely used to study developmental alcohol toxicity. Here we document the phenotype of hESC exposed to clinically-relevant, low dose ethanol (20 mM).

Methods: All cultures were maintained in 3% O2 to reflect normal physiologic conditions. Undifferentiated hESC were expanded with basic fibroblast growth factor (bFGF), with or without ethanol, then differentiated without ethanol. Proliferation and apoptosis in response to ethanol were assayed, and PCR used to examine expression of GABA receptor subunits. Whole cell patch clamping was used to examine GABAA receptor function in undifferentiated hESC. Immunocytochemistry and western blotting were used to follow differentiation of early neurons, astrocytes, and oligodendrocytes,

Principal findings: Exposure to 20 mM ethanol resulted in larger colonies of undifferentiated hESC despite an increase in apoptosis, because proliferation of the undifferentiated cells (and neuroblasts) was significantly increased. Differentiation of hESC (following a week of ethanol exposure) resulted in decreased expression of GFAP (by western) compared to unexposed cells, suggesting that astrocyte differentiation was reduced, while markers of oligodendrocyte and neuron differentiation were unchanged. At the message level, undifferentiated hESC express all GABAA receptor subunits, but functional receptors were not found by whole cell patch clamping.

Conclusion: Our results in hESC suggest a complex mix of ethanol-induced phenotypic changes when ethanol exposure occurs very early in development. Not only increased apoptosis, but inappropriate proliferation and loss of trophic astrocytes could result from low-dose ethanol exposure very early in development. More generally, these studies support a role for hESC in developing hypotheses and focusing questions to complement animal studies of developmental toxicities.

Read Full Article,
http://www.sciencedirect.com/science/article/pii/S0014488611004742

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PubMed, Sheng Li Xue Bao. 2011 Dec 25;63(6):479-90.

118. ALCOHOL-INDUCED PROLIFERATION OF NEURONS IN MOUSE HIPPOCAMPAL DENTATE GYRUS: A POSSIBLE ROLE OF CERAMIDE

Deng TX, Wang ZX, Gao XQ, Shi YY, Ma ZY, Jin HX, Deng JB.
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ABSTRACT
To investigate the role and mechanism of ceramide (Cer) regulation in alcohol-induced neuronal proliferation and the newborn neurons formation, we used sphingomyelin synthase 2 (predominant enzyme of Cer metabolism) knockout (SMS2(-/-)) and wild type (WT) female mice to establish the model of prenatal alcohol exposure. In 24 h after being given birth (postnatal day 0, P0), the offspring of model mice received blood sphingomyelin (SM) measurement with enzymatic method. On P0, P7, P14 and P30, the proliferation of granule cells in the dentate gyrus and newborn neurons were investigated with immunofluorescent labeling.

The expression of protein kinase Cα (PKCα) in the hippocampus was tested with Western blot analysis. The results showed that the SM level of blood in SMS2(-/-) pups was significantly lower than that in WT pups. No matter in SMS2(-/-) or WT mice, the prenatal alcohol exposure down-regulated the SM levels in pups with dose-dependency. In both SMS2(-/-) and WT pups, the number of proliferative neurons and newborn neurons in the dentate gyrus gradually decreased with the growing age. Compared with the WT pups, SMS2(-/-) pups showed significantly more proliferative neurons and newborn neurons in the dentate gyrus. Notably, prenatal alcohol exposure dose-dependently increased proliferative neurons and newborn neurons in the dentate gyrus in both WT and SMS2(-/-) pups.

The hippocampal expression of PKCα protein in SMS2(-/-) mice was lower than that in WT mice, and prenatal alcohol exposure could up-regulate the PKCα protein expression in both WT and SMS2(-/-) mice with dose dependency. These results suggest that alcohol exposure during pregnancy can induce the compensatory neural cell proliferation and the production of newborn neurons in offspring, and the Cer-ceramide-1-phosphate (C1P) pathway is involved in alcohol-induced neural cell proliferation. The activation of PKCα may be a key step to start the Cer-C1P pathway and up-regulate the alcohol-induced neural cell proliferation and the newborn neurons formation.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22193441

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Thieme eJournals, Fortschr Neurol Psychiatr
DOI: 10.1055/s-0031-1281846

119. WHAT IS KNOWN ABOUT THE OUTCOME AS ADULTS FOR CHILDREN WITH FETAL ALCOHOL SYNDROME (FAS)/FETAL ALCOHOL SPECTRUM DISORDERS (FASD)?

J. E. Walloch, P. H. Burger, J. Kornhuber
Psychiatrische und Psychotherapeutische Klinik, Universitätsklinikum Erlangen

ABSTRACT

In the field of adult psychiatry in German-speaking countries, little attention is as yet paid to the psychic defects that a fetus can sustain as a result of prenatal exposure to alcohol. Although children of alcohol-dependent mothers do present to psychiatric institutions as adults with manifold symptoms, e. g., attention deficit disorders, affective disorders or intellectual disability, fetal alcohol spectrum disorders are rarely diagnosed as an underlying cause.

Appropriate therapy guidelines do not exist. Current review papers within the German-speaking countries usually stem from paediatric and adolescent psychiatry or medicine. Based on a selected review of the literature, the following paper addresses and discusses the disease entity of fetal alcohol spectrum disorders and fetal alcohol syndrome and their significance for adult psychiatry and also identifies open questions and research requirements, e. g., the development of diagnostic instruments or the establishment of diagnostic categories.

Read Full Article,
https://www.thieme-connect.com/DOI/DOI?10.1055/s-0031-1281846

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Wiley Online Library, Alcoholism: Clinical and Experimental Research
Volume 36, Issue 3, pages 417–424, March 2012
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120. ASSESSMENT OF PRENATAL EXPOSURE TO ETHANOL BY MECONIUM ANALYSIS: RESULTS OF AN ITALIAN MULTICENTER STUDY

Simona Pichini, Emilia Marchei, Federica Vagnarelli, Luigi Tarani, Francesco Raimondi, Rosalba Maffucci, Bruno Sacher, Massimo Bisceglia, Gherardo Rapisardi, Maria Rosaria Elicio, Paolo Biban, Piergiorgio Zuccaro, Roberta Pacifici, Andrea Pierantozzi, Luca Morini

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ABSTRACT

Background: This study estimated in 7 Italian cities the prevalence of prenatal exposure to ethanol by determining fatty acid ethyl esters (FAEEs; palmitic, palmitoleic, stearic, oleic, linoleic, linolenic, and arachidonic esters) and ethyl glucuronide (EtG) in neonatal meconium samples.

Methods: A total of 607 meconium samples were obtained from neonatal wards of 7 public hospitals: Verona and San Daniele del Friuli in the northeast of the country, Reggio Emilia in the middle east, Florence and Rome in the center, and Naples and Crotone in the southwest of the peninsula.

Meconium biomarkers were assessed by a validated methodology using liquid chromatography–tandem mass spectrometry and the results categorized using the accepted cutoff of 2 nmol/g total amount of 7 FAEEs and 2 nmol/g EtG, to differentiate between heavy maternal ethanol use during pregnancy and occasional or no use at all.

Results: On the basis of the above-reported cutoffs, the overall prevalence of newborns prenatally exposed to maternal ethanol was 7.9%: 0% in Verona, 4.0% in San Daniele del Friuli, 4.9% in Naples, 5.0% in Florence, 6.2% in Crotone, up to 10.6% in Reggio Emilia, and 29.4% in Rome.

Low maternal education level and younger maternal age were associated with biomarker scores over the cutoff. There was also a significant correlation between the highest percentage of prenatal exposure in the capital and certain maternal sociodemographic characteristics.

Conclusions: These results indicate considerable variability in the prevalence of fetal exposure to ethanol in different Italian cities, as determined by the objective measurement of biomarkers in meconium. These data, together with previous ones obtained in Barcelona, Spain, indicate that gestational ethanol exposure is widespread, at least in parts of Europe.

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http://onlinelibrary.wiley.com/doi/10.1111/j.1530-0277.2011.01647.x/abstract

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Wiley Online Library, Drug and Alcohol Review, Special Issue: Low Risk Drinking Guidelines.
Volume 31, Issue 2, pages 156–161, March 2012
Article first published online: 12 DEC 2011, DOI: 10.1111/j.1465-3362.2011.00395.x

121. DIFFERENT GUIDELINES FOR DIFFERENT COUNTRIES? ON THE SCIENTIFIC BASIS OF LOW-RISK DRINKING GUIDELINES AND THEIR IMPLICATIONS

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ABSTRACT
The scientific evidence for low-risk drinking guidelines was examined in a narrative review focusing on three points: definition of exposure, the best way to select outcomes and risk relations and how to determine thresholds. With respect to exposure, at least two dimensions should be incorporated: average volume of alcohol consumption and patterns of irregular heavy drinking occasions. Mortality should be selected as the most severe outcome, and a disaggregated approach should be adopted incorporating the regional demographic and cause of death structure. Finally, our plea is for establishing a general threshold for acceptable risk on a societal level rather than ad hoc specific committees setting norms for specific risks. Acceptable thresholds will be different if the risk is to oneself or to others.[Rehm J, Patra J. Different guidelines for different countries? On the scientific basis of low-risk drinking guidelines and their implications.

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http://onlinelibrary.wiley.com/doi/10.1111/j.1465-3362.2011.00395.x/abstract

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Sage Journals, Published online before print December 12, 2011, doi: 10.1177/1524839911404232
Health Promot Pract December 12, 2011 1524839911404232

122. DEVELOPMENT OF A MEDIA CAMPAIGN ON FETAL ALCOHOL SPECTRUM DISORDERS FOR NORTHERN PLAINS AMERICAN INDIAN COMMUNITIES

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Austin Winberg, MA, Amy Elliott, PhD

ABSTRACT
Alcohol-exposed pregnancies are especially of concern for American Indians. The Indian Health Service reported that 47% to 56% of pregnant patients admitted to drinking alcohol during their pregnancy. In addition, rates of Fetal Alcohol Syndrome are estimated to be as high as 3.9 to 9.0 per 1,000 live births among American Indians in the Northern Plains, making prevention of alcohol-exposed pregnancies an important public health effort for this population. The goal of this article is to add to the literature on universal prevention of Fetal Alcohol Spectrum disorders by describing the development, dissemination, and evaluation of a media campaign on Fetal Alcohol Spectrum Disorders that was created by and for American Indian communities in the Northern Plains.

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http://hpp.sagepub.com/content/early/2011/12/12/1524839911404232.abstract

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Wiley Online Library, Alcoholism: Clinical and Experimental Research
Article first published online: 7 DEC 2011
DOI: 10.1111/j.1530-0277.2011.01679.x

123. CALLOSAL THICKNESS REDUCTIONS RELATE TO FACIAL DYSMORPHOLOGY IN FETAL ALCOHOL SPECTRUM DISORDERS

Yaling Yang, Owen R. Phillips, Eric Kan, Kathleen K. Sulik, Sarah N. Mattson, Edward P. Riley, Kenneth L. Jones, Colleen M. Adnams, Philip A. May, Mary J. O'Connor, Katherine L. Narr, Elizabeth R. Sowell
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ABSTRACT
Background: Structural abnormalities of the corpus callosum (CC), such as reduced size and increased shape variability, have been documented in individuals with fetal alcohol spectrum disorders (FASD). However, the regional specificity of altered CC structure, which may point to the timing of neurodevelopmental disturbances and/or relate to specific functional impairments, remains unclear. Furthermore, associations between facial dysmorphology and callosal structure remain undetermined.

Methods: One hundred and fifty-three participants (age range 8 to 16) including 82 subjects with

FASD and 71 nonexposed controls were included in this study. The structural magnetic resonance imaging data of these subjects was collected at 3 sites (Los Angeles and San Diego, California, and Cape Town, South Africa) and analyzed using classical parcellation schemes, as well as more refined surface-based geometrical modeling methods, to identify callosal morphological alterations in FASD at high spatial resolution.

Results: Reductions in callosal thickness and area, specifically in the anterior third and the splenium, were observed in FASD compared with nonexposed controls. In addition, reduced CC thickness and area significantly correlated with reduced palpebral fissure length.

Conclusions: Consistent with previous reports, findings suggest an adverse effect of prenatal alcohol exposure on callosal growth and further indicate that fiber pathways connecting frontal and parieto-occipital regions in each hemisphere may be particularly affected. Significant associations between callosal and facial dysmorphology provide evidence for a concurrent insult to midline facial and brain structural development in FASD.

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http://onlinelibrary.wiley.com/doi/10.1111/j.1530-0277.2011.01679.x/abstract

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Wiley Online Library, Alcoholism: Clinical and Experimental Research
Article first published online: 7 DEC 2011, DOI: 10.1111/j.1530-0277.2011.01682.x

124. ETHANOL AFFECTS DIFFERENTIATION-RELATED PATHWAYS AND SUPPRESSES WNT SIGNALING PROTEIN EXPRESSION IN HUMAN NEURAL STEM CELLS

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From the Children's Research Center of Michigan, The Carman and Ann Adams Department of Pediatrics (SDV, WDL), Wayne State University School of Medicine, Detroit, Michigan; and Children's Hospital of Michigan, Detroit, Michigan.

ABSTRACT
Background: Prenatal exposure of the fetus to ethanol (EtOH) can be teratogenic. We previously showed that EtOH alters the cell fate of human neural stem cells (NSC). As Wnt signaling plays an important role in fetal brain development, we hypothesized that EtOH suppresses Wnt signaling protein expression in differentiating NSC and thereby contributes to fetal alcohol spectrum disorder.

Methods: NSC isolated from fetal human brains were cultured in mitogenic media to induce neurospheres, which were dissociated into single-cell suspensions and used for all experiments. Equal numbers of NSC were cultured on lysine/laminin-coated plates for 96 hours in differentiating media containing 0, 20, or 100 mM EtOH. Total mRNA was isolated from samples containing 0 or 100 mM EtOH and changes in expression of 263 genes associated with neurogenesis and NSC differentiation were determined by Oligo GEArray technology. The biological impact of gene changes was estimated using a systems biology approach with pathway express software and KEGG database. Based on the pathways identified, expression of Wnt proteins (Wnt3a and Wnt5a), Wnt-receptor complex proteins (p-LRP6, LRP6, DVL2, and DVL3), Wnt antagonist Naked-2 (NKD-2), and downstream Wnt proteins (β-catenin, Tyr-p-GSK3β, Ser-p-GSK3β) were analyzed by Western blot.

Results: Of the 263 genes examined, the expressions of 22 genes in differentiating NSC were either upwardly or downwardly affected by EtOH. These genes are associated with 5 pathways/cellular processes: axon guidance; hedgehog signaling; TGF-β signaling; cell adhesion molecules; and Wnt signaling. When compared to controls, EtOH, at both 20 and 100 mM concentrations, suppressed the expression of Wnt3a and Wnt5a, receptor complex proteins p-LRP6, LRP6 and DVL2, and cytoplasmic proteins Ser-p-GSK3β and β-catenin. Expression of NKD-2 and DVL3 remained unchanged and the expression of active Tyr-p-GSK3β increased

significantly.

Conclusions: EtOH can significantly alter neural differentiation pathway-related gene expression and suppress Wnt signaling proteins in differentiating human NSC.

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http://onlinelibrary.wiley.com/doi/10.1111/j.1530-0277.2011.01682.x/abstract

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PubMed, Neuroscience. 2012 Jan 27;202:465-73. Epub 2011 Dec 6.

125. ACTIVITY-DEPENDENT NEUROTROPHIC FACTOR-DERIVED PEPTIDE PREVENTS ALCOHOL-INDUCED APOPTOSIS, IN PART, THROUGH BCL2 AND C-JUN N-TERMINAL KINASE SIGNALING PATHWAYS IN FETAL BRAIN OF C57BL/6 MOUSE

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ABSTRACT
Fetal alcohol exposure is known to induce alteration in fetal brain development. In this study, we focused on neuroprotection against the effects of alcohol exposure using ADNF-9, a peptide derived from activity-dependent neurotrophic factor. We used a mouse model of fetal alcohol exposure to identify the intracellular mechanisms underlying the neuroprotective effects of ADNF-9. On embryonic day 7 (E7), weight-matched pregnant females were assigned to the following groups: (1) ethanol liquid diet (ALC) of 25% (4.49%, v/v) ethanol-derived calories; (2) pair-fed control (PF); (3) ALC combined with administration (i.p.) of ADNF-9 (ALC/ADNF-9); and (4) pair-fed combined with administration (i.p.) of ADNF-9 (PF/ADNF-9). On E13, fetal brains were collected, weighed, and apoptosis was determined using TdT-mediated dUTP nick-end labeling (TUNEL) assay. Bcl2 protein and phospho-c-Jun N-terminal kinase (JNK) levels were determined using Western blot and enzyme immunometric assay, respectively. ADNF-9 administration significantly prevented alcohol-induced reductions in fetal brain weight. In addition, ADNF-9 prevented an alcohol-induced increase in cell death in the primordium of the cerebral cortex and ganglionic eminence. Western blot analysis of the mitochondrial protein fractions revealed that ADNF-9 administration prevented an alcohol-induced reduction in the Bcl2 level. Moreover, an analysis of the proteins in the upstream signaling pathway revealed that ADNF-9 downregulated the phosphorylation of JNK. These data indicate that the mitochondrial Bcl2 pathway and JNK upstream signaling pathway are the intracellular targets of ADNF-9. The neuroprotective mechanism of action of ADNF-9 provides a direction for potential therapeutics against alcohol-induced neural damage involving mitochondrial dysfunction.

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http://www.ncbi.nlm.nih.gov/pubmed/22178609

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Wiley Online Library, Alcoholism: Clinical and Experimental Research
article first published online: 5 DEC 2011, DOI: 10.1111/j.1530-0277.2011.01676.x

126. ETHANOL EXPOSURE DURING PREGNANCY PERSISTENTLY ATTENUATES CRANIALLY DIRECTED BLOOD FLOW IN THE DEVELOPING FETUS: EVIDENCE FROM ULTRASOUND IMAGING IN A MURINE SECOND TRIMESTER EQUIVALENT MODEL

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ABSTRACT
Background: Ethanol (EtOH) consumption during pregnancy can lead to fetal growth retardation,

mental retardation, and neurodevelopmental delay.

The fetal brain initiates neurogenesis and vasculogenesis during the second trimester, and depends on maternal-fetal circulation for nutrition and growth signals. We used high-resolution in vivo ultrasound imaging to test the hypothesis that EtOH interferes with fetal brain-directed blood flow during this critical developmental period.

Methods: Pregnant mice were lightly anesthetized on gestational day 12 with an isoflurane/oxygen mixture. We assessed the effect of single and repeated binge-like maternal EtOH exposures at 3 g/kg, administered by intragastric gavage or intraperitoneal injection, on maternal circulation and fetal umbilical, aortic, internal carotid, and middle cerebral arterial circulation.

Results: Binge maternal EtOH exposure, regardless of exposure route, significantly reduced fetal arterial blood acceleration and velocity time integral (VTI), from umbilical to cerebral arteries, without a change in fetal heart rate and resistivity indices.

Importantly a single maternal binge EtOH exposure induced persistent suppression of fetal arterial VTI for at least 24 hours. Repeated binge episodes resulted in a continuing and persistent suppression of fetal VTI. Qualitative assessments showed that maternal EtOH exposure induced oscillatory, nondirectional blood flow in fetal cerebral arteries. Maternal cardiac and other physiological parameters remained unaltered.

Conclusions: These data show that binge-type maternal EtOH exposure results in rapid and persistent loss of blood flow from the umbilical artery to the fetal brain, potentially compromising nutrition and the maternal/fetal endocrine environment during a critical period for neuron formation and angiogenesis in the maturing brain.

Read Full Article,
http://onlinelibrary.wiley.com/doi/10.1111/j.1530-0277.2011.01676.x/abstract

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PubMed, Alcohol Clin Exp Res. 2011 Dec 5. doi: 10.1111/j.1530-0277.2011.01670.x.

127. DELIVERING ALCOHOL NEUROTOXICITY INTO NUCLEUS, WHEN CLUSTERIN MEETS BCL(XL) : A COMMENTARY

Suk K.
From the Department of Pharmacology (KS), Brain Science & Engineering Institute, Kyungpook National University School of Medicine, Daegu, Korea.

ABSTRACT

The study by Kim and colleagues (in press) demonstrated that the expression of nuclear isoform of clusterin is induced by ethanol (EtOH) to participate in apoptotic cell death of neurons in developing rodent brain. EtOH-induced nuclear clusterin interacts with Bcl(XL) , thereby liberating proapoptotic Bax. This study indicates the proapoptotic role of nuclear clusterin in EtOH-exposed neurons, linking specific nuclear events to alcohol neurotoxicity. The study provides novel insights into the molecular mechanisms underlying fetal alcohol spectrum disorders.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22142431

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PubMed, Brain Behav Immun. 2012 Mar;26(3):439-50. Epub 2011 Dec 1.

128. PRENATAL ALCOHOL EXPOSURE ALTERS THE COURSE AND SEVERITY OF ADJUVANT-INDUCED ARTHRITIS IN FEMALE RATS

Zhang X, Lan N, Bach P, Nordstokke D, Yu W, Ellis L, Meadows GG, Weinberg J.
Department of Cellular and Physiological Sciences, University of British Columbia, 2350 Health Sciences Mall, Vancouver, BC, Canada V6T 1Z3.

ABSTRACT

Prenatal alcohol exposure (PAE) has adverse effects on the development of numerous physiological systems, including the hypothalamic-pituitary-adrenal (HPA) axis and the immune system. HPA hyper-responsiveness and impairments in immune competence have been demonstrated. The present study investigated immune function in PAE females utilizing an adjuvant-induced arthritis (AA) model, widely used as a model of human rheumatoid arthritis. Given the effects of PAE on HPA and immune function, and the known interaction between HPA and immune systems in arthritis, we hypothesized that PAE females would have heightened autoimmune responses, resulting in increased severity of arthritis, compared to controls, and that altered HPA activity might play a role in the immune system changes observed. The data demonstrate, for the first time, an adverse effect of PAE on the course and severity of AA in adulthood, indicating an important long-term alteration in functional immune status. Although overall, across prenatal treatments, adjuvant-injected animals gained less weight, and exhibited decreased thymus and increased adrenal weights, and increased basal levels of corticosterone and adrenocorticotropin, PAE females had a more prolonged course of disease and greater severity of inflammation compared to controls. In addition, PAE females exhibited blunted lymphocyte proliferative responses to concanavalin A and a greater increase in basal ACTH levels compared to controls during the induction phase, before any clinical signs of disease were apparent. These data suggest that prenatal alcohol exposure has both direct and indirect effects on inflammatory processes, altering both immune and HPA function, and likely, the normal interactions between these systems.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/22155498

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PubMed, Nihon Arukoru Yakubutsu Igakkai Zasshi. 2011 Dec;46(6):576-84.

129. PROMISING THERAPY OF NEURAL STEM CELL TRANSPLANTATION FOR FASD MODEL--NEURAL NETWORK RECONSTRUCTION AND BEHAVIOR RECOVERY

Shirasaka T, Ukai W, Yoshinaga T, Watanabe K, Kaneta H, Kigawa Y, Igarashi T, Tateno M, Hashimoto E, Saito T.

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ABSTRACT

Objectives: It has been elucidated that psychiatric disorders are associated with impairment of the brain neural network. Reduction in brain size and hypoplasia of the basal ganglia and corpus callosum have been reported in Fetal Alcohol Spectrum Disorder (FASD). It is believed that the formation of the neural network is influenced by alcohol exposure during the fetal period. Additionally, it is well known that the functional expression of CNS consequences of prenatal alcohol exposure includes cognitive and attentional processes, as well as social behavioral problems. It has also been reported that abnormal 5-HT neuron development can be reversed by treatment with a 5-HT1A agonist in a prenatal alcohol exposure model. However, these treatments are prophylactic.

Without early intervention, the consequences of FASD are permanent. Recently, emerging evidence suggest that many clinical symptoms observed in psychiatric disease are likely related to neural network disruptions including neurogenesis dysfunction.

Neural stem cell (NSC) transplantation has been investigated in areas such as brain injury, stroke and neurodegenerative diseases and may be a way to reverse neurogenesis dysfunction. In the present work, we evaluated the usefulness of intravenous transplantation of NSCs in the FASD model rat focusing on the possibility of regenerative therapy, particularly regarding behavioral abnormalities, for FASD rats.

Results: Abnormal behaviors FASD model rats suggest that reduced social activity , and cognitive dysfunction are major symptoms in FASD patients. Intravenous NSC transplantation appeared to partially correct these behavioral abnormalities in FASD model rats. In the Amygdala areas intravenous NSC transplantation appears to have partially regaenerates expression of PSD95 in FASD model rats.

Conclusions: The results suggest that intravenous NSC transplantation may be an advanced approach to recover neural network damage and CNS dysfunction in FASD and possibly other psychiatric disorders.

Link to the Article,
http://www.ncbi.nlm.nih.gov/pubmed/22413566

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PubMed, Isr Med Assoc J. 2011 Dec;13(12):725-9.

130. FETAL ALCOHOL SPECTRUM DISORDER IN ISRAEL: INCREASED PREVALENCE IN AN AT-RISK POPULATION

Tenenbaum A, Hertz P, Dor T, Castiel Y, Sapir A, Wexler ID.
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ABSTRACT

Background: Maternal exposure to alcohol during pregnancy can lead to a wide range of clinical manifestations in their offspring, termed fetal alcohol spectrum disorder (FASD). In Israel, relatively few cases of FASD have been diagnosed and the prevalence has not been systematically evaluated.

Objectives: To determine the number of children with FASD or at risk for FASD in a select population of high risk patients seen at a clinic evaluating foster and adopted children.

Methods: Israeli children under 2 years old who were candidates for domestic adoption or in foster care were prospectively evaluated for clinical manifestations of FASD and information was obtained regarding parental use of alcohol or other illicit drugs.

Results: Of the 100 patients prospectively evaluated, 8 had mothers with a known history of alcohol consumption during pregnancy. Two of the children had fetal alcohol syndrome (FAS) without known maternal exposure to alcohol and two had partial FAS. Eleven other children were at risk for development of one of the diagnostic categories of FASD.

Conclusions: In a population of pre-adoption and foster children, 15% either had manifestations of FASD or were at risk for developing FASD. Although this is a select high risk population, the data from this study strongly suggest a greater prevalence of FASD than previously assumed. Under-diagnosis of FASD is detrimental to affected children who could benefit from interventions

designed to meet the needs of FASD victims.

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PubMed, J Cardiovasc Pharmacol. 2011 Dec;58(6):589-601.

131. FUNCTIONAL CHARACTERIZATION OF TRANSMEMBRANE INTRACELLULAR PH REGULATORS AND MECHANISM OF ALCOHOL-INDUCED INTRACELLULAR ACIDOSIS IN HUMAN UMBILICAL CORD BLOOD STEM CELL-LIKE CELLS

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ABSTRACT

Changing intracellular pH (pHi) exerts considerable influence on many cellular functions. Different pHi regulators, such as the Na-H exchanger (NHE), Na/(Equation is included in full-text article.)symporter, and Cl/OH exchanger (CHE), have been identified in mature mammalian cells.

The aims of the present study were to investigate the physiological mechanisms of pHi recovery and to further explore the effects of alcohol on the pHi in human umbilical cord blood CD34 stem cell-like cells (HUCB-CD34STs). HUCB-CD34STs were loaded with the pH-sensitive dye, 2',7'-bis(2-carboxethyl)-5(6)-carboxyfluorescein, to examine pHi. In isolated HUCB-CD34STs, we found that (1) the resting pHi is 7.03 ± 0.02; (2) 2 Na-dependent acid extruders and a Cl-dependent acid loading carrier exist and are functional; (3) alcohol functions in a concentration-dependent manner to reduce pHi and increase NHE activity, but it does not affect CHE activity; and (4) fomepizole, a specific alcohol dehydrogenase inhibitor, does not change the intracellular acidosis and NHE activity-induced by alcohol, whereas 3-amino-1, 2,4-trizole, a specific catalase inhibitor, entirely abolishes these effects. In conclusion, we demonstrate that 2 acid extruders and 1 acid loader (most likely NHE, NBC, and CHE, respectively) functionally existed in HUCB-CD34STs. Additionally, the intracellular acidosis is mainly caused by catalase-mediated alcohol metabolites, which provoke the activity of NHE.

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http://www.ncbi.nlm.nih.gov/pubmed/22146403

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J Popul Ther Clin Pharmacol Vol 18(3):e544-e563; November 30, 2011

132. THE ROLE OF THE MOTHER-CHILD RELATIONSHIP IN DEVELOPMENTAL OUTCOMES OF INFANTS AND YOUNG CHILDREN WITH AND WITHOUT PRENATAL ALCOHOL EXPOSURE

Mary Motz, Stacey D Espinet, Jessica J Jeong, Danielle Major, Nicole Racine, Julie Chamberlin, Debra J Pepler

ABSTRACT

Background: Prenatal alcohol exposure has been associated with deficits in many developmental areas. Effects on developmental outcomes can be exacerbated by cumulative risk across the pre- and postnatal environments. Given that the parent-infant relationship provides the primary context for healthy child development, it is possible that maternal caregiving may play a substantial role in mitigating these effects.

Objectives: To clarify the role of the quality of the mother-child relationship in the relation between

cumulative risk and neurodevelopmental outcomes.

Methods: Participants were 40 infants/children and their mothers with substance-use problems who were taking part in an early mental health intervention program. Cumulative risk, across the pre- and postnatal period was measured, and quality of the mother-child relationship was rated based on clinical file reviews and observation of mother-child interactions. Outcome measures were infant/child IQ, and neurobehavioral functioning rated across several developmental domains.

Results: The quality of the mother-child relationship mediated the direct relation between cumulative risk and neurobehavioral functioning, and cumulative risk was related with IQ indirectly through the mother-child relationship.

Conclusions: These findings indicate an important role for quality of the mother-child relationship in determining outcomes for infants and young children of substance-using women, and emphasize the need to consider both the larger context of risk, as well as the mother-child relationship for best intervention outcomes.

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http://www.cjcp.ca/pubmed.php?articleId=351

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Front Psychiatry. 2011; 2: 66.
Published online 2011 November 30. doi: 10.3389/fpsyt.2011.00066
Received April 18, 2011; Accepted November 11, 2011.

133. SOCIAL BEHAVIOR OF OFFSPRING FOLLOWING PRENATAL COCAINE EXPOSURE IN RODENTS: A COMPARISON WITH PRENATAL ALCOHOL

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ABSTRACT
Clinical and experimental reports suggest that prenatal cocaine exposure (PCE) alters the offsprings' social interactions with caregivers and conspecifics. Children exposed to prenatal cocaine show deficits in caregiver attachment and play behavior. In animal models, a developmental pattern of effects that range from deficits in play and social interaction during adolescence, to aggressive reactions during competition in adulthood is seen. This review will focus primarily on the effects of PCE on social behaviors involving conspecifics in animal models. Social relationships are critical to the developing organism; maternally directed interactions are necessary for initial survival. Juvenile rats deprived of play behavior, one of the earliest forms of non-mother directed social behaviors in rodents, show deficits in learning tasks and sexual competence. Social behavior is inherently complex. Because the emergence of appropriate social skills involves the interplay between various conceptual and biological facets of behavior and social information, it may be a particularly sensitive measure of prenatal insult. The social behavior surveyed include social interactions, play behavior/fighting, scent marking, and aggressive behavior in the offspring, as well as aspects of maternal behavior. The goal is to determine if there is a consensus of results in the literature with respect to PCE and social behaviors, and to discuss discrepant findings in terms of exposure models, the paradigms, and dependent variables, as well as housing conditions, and the sex and age of the offspring at testing. As there is increasing

evidence that deficits in social behavior may be sequelae of developmental exposure alcohol, we compare changes in social behaviors reported for prenatal alcohol with those reported for prenatal cocaine. Shortcomings in the both literatures are identified and addressed in an effort to improve the translational value of future experimentation.

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Australian Journal of Primary Health 18(1) 68-73 http://dx.doi.org/10.1071/PY10077
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134. WHAT INFLUENCES AUSTRALIAN WOMEN TO NOT DRINK ALCOHOL DURING PREGNANCY?

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ABSTRACT

There is a strong social norm against consuming alcohol during pregnancy. However, many women do not realise they are pregnant until the sixth week and are not provided with information about the risks of consuming alcohol until they visit a health professional in the second trimester. We conducted semi-structured interviews with 12 midwives and 12 pregnant women from two regions in NSW in 2008–09 to explore attitudes towards alcohol consumption during pregnancy, and the factors that may encourage or inhibit women from following the recommendation to abstain from drinking while pregnant. Both groups noted the social issues around pregnant women consuming alcohol due to perceived social norms and the challenges in not revealing early pregnancy status at social events.

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Journal of Mental Health
October 2011, Vol. 20, No. 5 , Pages 473-483 (doi:10.3109/09638237.2011.577113)

135. MENTAL HEALTH ISSUES IN FETAL ALCOHOL SPECTRUM DISORDER

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ABSTRACT

Background: High numbers of individuals with Fetal Alcohol Spectrum Disorders (FASD) have been described as having mental health problems.

Aims: This article summarizes research about mental health problems in FASD and considers related developmental and environmental issues.

Method: A computer-based literature search was conducted in the databases Medline, PsycINFO, Google Scholar, Academic Search Complete, and Education Resources Information Centre for articles addressing the prevalence and types of mental health issues in individuals affected by FASD.

Results: High rates of mental disorders within the FASD and prenatal alcohol exposure (PAE) population were found to be consistently reported for both internalizing and externalizing disorders. Moreover, problems that emerge in childhood may reflect a convergence of genetic, environmental, and neurophysiological factors that persist into adulthood.

Conclusions: Researchers are beginning to document the impacts of PAE on later mental health development. Further longitudinal study is needed to determine whether there is an increasing severity of mental health deficits and consequences with age, and whether any such changes reflect increasingly deteriorating environmental factors or brain-based factors. Additionally, research is needed to design interventions to better address the unique mental health needs of this population.

Read Full Article,
http://informahealthcare.com/doi/abs/10.3109/09638237.2011.577113

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PubMed, S Afr Med J. 2011 Sep 27;101(10):724, 726-7.

136. CHILDHOOD BEHAVIOURAL AND DEVELOPMENTAL DISORDERS: ASSOCIATION WITH MATERNAL ALCOHOL CONSUMPTION IN CAPE TOWN, SOUTH AFRICA

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ABSTRACT

Current maternal alcohol consumption, especially binge drinking, is strongly associated with childhood behavioural and/or developmental disorders (BDDs) in a population attending tertiary hospital ambulatory services. BDDs were also associated with maternal alcohol use 6 months before pregnancy. An association with BDDs could not be conclusively demonstrated for drinking during pregnancy, but this may have been influenced by under-reporting and reduced study power due to misclassification of exposure. We cannot rule out the a priori suspicion that some mild BDDs in children in the Western Cape could be undiagnosed fetal alcohol spectrum disorder. Nonetheless, the study highlighted the important impact of current maternal alcohol use on behaviour and development of children. Future research on the impact of maternal alcohol use on childhood development should include examination of environmental and social factors contributing to this increased risk. Upstream interventions aimed at reducing alcohol-related harms may also contribute to reducing the burden of BDDs.

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http://www.ncbi.nlm.nih.gov/pubmed/22272851

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ScienceDirect, Drug and Alcohol Dependence
Volume 120, Issues 1–3, 1 January 2012, Pages 113–118
Received 20 January 2011. Revised 7 July 2011. Accepted 8 July 2011. Available online 4 August 2011.
http://dx.doi.org/10.1016/j.drugalcdep.2011.07.004, How to Cite or Link Using DOI

137. ALCOHOL CONSUMPTION AMONG HIV-POSITIVE PREGNANT WOMEN IN KWAZULU-NATAL, SOUTH AFRICA: PREVALENCE AND CORRELATES

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ABSTRACT

Background: HIV-positive pregnant women who drink put their children at risk of both HIV and fetal alcohol spectrum disorders. The province of KwaZulu-Natal (KZN) has the highest prevalence of HIV in South Africa, but has not before been considered an area of high alcohol consumption among women. This paper analyzes a large sample of HIV+ pregnant women in KZN to examine alcohol consumption in that population.

Methods: Data came from assessments of women enrolled in Prevention of Mother-To-Child Transmission programs at 8 clinics in KZN. Descriptive statistics and logistic regressions were used to examine the prevalence and correlates of alcohol consumption and binge drinking.

Results: Of 1201 women assessed, 18% reported drinking during pregnancy, and 67% of drinkers usually binged when drinking (had 3+ drinks in one sitting). Over one-third of drinkers binged twice a month or more. Women living in urban and peri-urban locations were more likely to drink, as were those with indicators of higher economic status and greater social engagement. Married women were less likely to drink, while women who had poorer mental health, used tobacco, or had a greater history of sexual risk-taking were more likely to drink.

Conclusion: Health care workers in KZN should be aware that pregnant women who drink are likely to do so at a level that is dangerous for their babies. Some factors associated with drinking indicate social/environmental influences that need to be counteracted by greater dissemination of information about the dangers of drinking, and greater support for abstinence or moderation.

Read Full Article,
http://www.sciencedirect.com/science/article/pii/S0376871611003085

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PubMed, Adv Neonatal Care. 2011 Aug;11(4):255-67.

138. IMPLEMENTING A PERINATAL SUBSTANCE ABUSE SCREENING TOOL

Wallman CM, Smith PB, Moore K.
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ABSTRACT

Newborns exposed to illicit drugs or alcohol in utero can face physical, social, and emotional obstacles. Outcomes for children with fetal alcohol syndrome disorders are well documented in the literature. Data exist on the effects of maternal illicit drug use. Identifying perinatal substance abuse can increase positive outcomes for newborns and create the opportunity for mothers to access assistance through referrals to community resources.This article provides insight on how

hospitals can implement an effective screening tool through patient surveying and testing, nurse education, and collaboration with community agencies in a multidisciplinary advisory committee setting.

This discussed method of universal perinatal screening results in increased positive screens and increased referrals for care and support. Emphasis is placed on universal screening and testing methods. Nurses are trained in motivational interview techniques that convey empathy, listening, and objectivity. Community agencies partner with hospital staff through onsite meetings with families that determine the best discharge plan for the newborn. The multidisciplinary advisory committee meets continually to discuss future enhancements.

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http://www.ncbi.nlm.nih.gov/pubmed/22123347

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Wiley Online Library - Journal of Child Psychology and Psychiatry
Volume 53, Issue 1, pages 64–72, January 2012
Article first published online: 28 JUL 2011,
DOI: 10.1111/j.1469-7610.2011.02432.x

139. IS SENSORY OVER-RESPONSIVITY DISTINGUISHABLE FROM CHILDHOOD BEHAVIOR PROBLEMS? A PHENOTYPIC AND GENETIC ANALYSIS

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ABSTRACT

Background: Although impaired sensory processing accompanies various clinical conditions, the question of its status as an independent disorder remains open. Our goal was to delineate the comorbidity (or lack thereof) between childhood psychopathology and sensory over-responsivity (SOR) in middle childhood using phenotypic and behavior-genetic analyses.

Method: Participants (N = 970) were drawn from the Wisconsin Twin Project, a population-based sample of twins and their families. Mothers completed a sensory responsivity checklist when their offspring were on average 7 years old, followed by a diagnostic interview (Diagnostic Interview Schedule for Children; DISC) within 6–12 months. We examined the incidence of DISC diagnoses – attention deficit hyperactivity disorder, conduct disorder, oppositional defiant disorder, agoraphobia, general anxiety, obsessive-compulsive disorder, panic disorder, separation anxiety, social phobia, specific phobia, depression, enuresis, trichtollomaniatics, selective mutism, and pica – among children with SOR, and vice versa.

Children with autism or pervasive developmental disorders were excluded from the present study. In addition, we examined parent-reported physical health diagnoses among nondiagnosed children and three groups of children with SOR and/or DISC diagnoses. Biometric models explored common underlying genetic and environmental influences on symptoms of SOR and psychopathology.

Results: A majority of individuals who screened positive for SOR did not qualify for a DISC diagnosis (58.2%), and vice versa (68.3%). Children who screened positive for SOR only and typical children had similar rates of physical health problems. Turning to a dimensional approach, multivariate twin models demonstrated that modest covariation between SOR and DISC symptoms could be entirely accounted for by common underlying genetic effects.

Conclusions: Our results suggest that SOR occurs independently of recognized childhood

psychiatric diagnoses but is also a relatively frequent comorbid condition with recognized diagnoses. Genetic sources of this comorbidity are implicated.

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http://onlinelibrary.wiley.com/doi/10.1111/j.1469-7610.2011.02432.x/abstract

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American Journal of Obstetrics & Gynecology
Volume 204, Issue 1, Supplement , Page S182, January 2011

140. CYTOKINE ALTERATIONS IN FETAL ALCOHOL SYNDROME
Robin Roberson, Thea Kuddo, Ines Benassou, Catherine Spong

ABSTRACT
No abstract available

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http://www.ajog.org/article/S0002-9378(10)01731-X/

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PubMed, Conf Proc IEEE Eng Med Biol Soc. 2011;2011:789-92.

141. AN AUDITORY GO/NO-GO STUDY OF EVENT-RELATED POTENTIALS IN CHILDREN WITH FETAL ALCOHOL SPECTRUM DISORDERS
Steinmann TP, Andrew CM, Thomsen CE, Kjær TW, Meintjes EM, Molteno CD, Jacobson JB, Jacobson SW, Sorensen HB.
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ABSTRACT

In this study event-related potentials (ERPs) were used to investigate the effects of prenatal alcohol exposure on response inhibition identified during task performance. ERPs were recorded during a auditory Go/No Go task in two groups of children with mean age of 12.8 years (11 years to 14.7 years): one diagnosed with fetal alcohol syndrome (FAS) or partial FAS (FAS/PFAS; n = 12) and a control group of children of same age whose mothers abstained from alcohol or drank minimally during pregnancy (n = 11).

The children were instructed to push a button in response to the Go stimulus and not to press the button when the No Go stimulus were heard. Task performance accuracy did not differ between the two groups, however differences were observed in the ERP components: P2, N2, and P3. The P2 amplitude were larger for Go trials in both groups. The FAS/PFAS group showed slower N2 response to Go trials, suggesting a less efficient early classification of the stimulus. P3 showed larger amplitudes to No-Go vs. Go in both groups.

The study has provided new evidence for inhibition deficits in FAS/PFAS subjects identified by ERPs.

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http://www.ncbi.nlm.nih.gov/pubmed/22254429

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Journal Of Developmental And Physical Disabilities
Volume 23, Number 2 (2011), 143-167, DOI: 10.1007/s10882-010-9204-2

142. FETAL ALCOHOL SPECTRUM DISORDER: A REVIEW OF NEURODEVELOPMENTAL FINDINGS AND INTERVENTIONS

Krista Davis, Mary Desrocher and Timothy Moore

No Abstract Available

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ARTICLE ABSTRACTS

12th Annual Fetal Alcohol Canadian Expertise (FACE) 2011 Poster Competition Abstracts
13th September 2011

1. SOCIAL PERSPECTIVE TAKING AND SOCIAL AFFECTIVE PROCESSING IN CHILDREN WITH FETAL ALCOHOL SPECTRUM DISORDERS

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5 Motherisk Program, The Hospital for Sick Children, Toronto, Ontario

ABSTRACT

Background: Children with Fetal Alcohol Spectrum Disorders (FASD) show severe behaviour and cognitive impairments. These children also display striking difficulties in adaptive and social functioning, which extend beyond their cognitive delays. However, the factors that contribute to these social impairments are not well understood. The objective of the present study was to investigate two areas of social cognition, namely social perspective taking and affect processing as potential contributing factors to the cognitive, behaviour and social impairments observed in FASD.

Methods: Studied at The Hospital for Sick Children were 42 children between 8 to 12 years of age; 25 had a diagnosis of a FASD (mean age 10.35) and 17 were typically developing control (TDC) children (mean age 10.25). The Theory of Mind and Affect Recognition subtests from the NEPSY-II (Korkman, Kirk & Kemp, 2009) were used to investigate social perspective taking and affect processing, respectively.

Results: Children with FASD had poorer social perspective taking skills compared to TDC, as observed by their lower percentiles on the Theory of Mind subtest. The FASD group also had poorer affect processing compared to the TDC group.

Of note, the mean score on the Affect Recognition subtest for the FASD group remained in the average range, whereas the mean score for TDC approached the high average range. The FASD group, however, made more errors on happy, neutral and angry facial expressions.

Conclusions: Children with FASD displayed reduced social perspective taking and affect processing abilities, compared to TDC. These social cognitive weaknesses may contribute to the poor behaviour regulation observed in children with FASD, and significantly impacts their daily social functioning.

Keywords: Social cognition, theory of mind, affect processing
Source of funding for the study: CFFAR; CIHR
Conflict of interest: None
Student/Trainee: Full-time PhD student
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http://www.cjcp.ca/pubmed.php?articleId=332

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2. EVALUATION OF THE NEUROBEHAVIORAL SCREENING TOOL IN CHILDREN WITH FETAL ALCOHOL SPECTRUM DISORDER (FASD) AND PRENATAL ALCOHOL EXPOSURE (PAE)

La France M[1], Nash K[2], Koren G[3], Andrew G[4], Rasmussen C[1]
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4 Glenrose Rehabilitation Hospital, Alberta

ABSTRACT
Background: The Neurobehavioral Screening Tool (NST) is a part of the National Screening Tool Kit for FASD. The NST is a parental rating scale consisting of ten questions identified by Nash et al. (2006) to be predictive of FASD based on the child's current behaviour. The purpose of this study was to field test the NST to determine how well it differentiates between children diagnosed with FASD, those with PAE but no FASD diagnosis, and typically developing controls.

Methods: We administered the NST to three groups of children (6 to 17 years): 36 with FASD and 21 PAE assessed at the Glenrose Hospital FASD clinic in Edmonton, and 21 controls recruited locally.

Results: Significantly more children with FASD screened positive on the NST compared to the PAE and the control group. The sensitivity of the NST in our sample was 36.1%. Within the FASD group, there was a higher sensitivity among older participants (50%) 12- 17 years, than younger participants (22%) 6-11 years. The NST had 100% specificity against the control group; none of the controls tested positively.

Discussion: Despite overall low sensitivity, the specificity in our sample is very high indicating that the selected items are specific to FASD. This supports the use of the NST as a national screening tool which would lead to large numbers of children being screened and undergoing the process of diagnosis.

Keywords: Neurobehavioralscreening tool, fetal alcohol spectrum disorder, prenatal alcohol exposure
Source of funding: The Public Health Agency of Canada
Conflict of interest: None
Student/Trainee: Trainee
Corresponding author: mtl5@ualberta.ca

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13th September 2011

3. THE PATTERNS OF SLEEP DISORDERS AND CIRCADIAN RHYTHM DISRUPTIONS IN CHILDREN AND ADOLESCENTS WITH FETAL ALCOHOL SPECTRUM DISORDERS

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1 Youthdale Child and Adolescent Sleep Centre, Toronto, Ontario;
2 Collaborative Program in Neurosciences, University of Toronto, Toronto, Ontario;
3 Department of Neuropsychiatry, Toronto Western Hospital, Toronto, Ontario

ABSTRACT
Background: Sleep disorders have been poorly described in children and adolescents diagnosed

with Fetal Alcohol Spectrum Disorders (FASD). The objective of this study is to describe the sleep and circadian rhythm characteristics of children with FASD using overnight polysomnography, sleep questionnaires, and the Dim Light Melatonin Onset (DLMO) test. To our knowledge, no comprehensive studies of this nature have been conducted.

Methods: Thirty six children aged 6-18 years diagnosed with FASD were recruited from various FASD clinics to the Youthdale Child and Adolescent Sleep Centre in Toronto. After a medical consultation, each participant had one night of overnight polysomnography, as well as an additional night of DLMO. Participants completed various sleep and FASD questionnaires. Data was analyzed using SPSS 19.

Results: Significant differences were found when comparing the sleep architecture of FASD participants to normative data. There was a high prevalence of sleep disorders in this sample. Most of the melatonin profiles of the FASD participants were found to be abnormal, suggesting HPA axis disturbances. The melatonin results are congruous with the brain pathology in studies of animals prenatally exposed to alcohol.

Conclusion: The high prevalence of sleep disorders and melatonin abnormalities in this population warrants sleep assessments, as part of and/or in conjunction with the diagnostic process.

Keywords: Fetal alcohol spectrum disorders, sleep disorders, circadian rhythm
Source of funding: Youthdale Foundation
Conflict of interest: None
Student/Trainee: Full-time
Corresponding author: s.goril@utoronto.ca

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4. EXECUTIVE FUNCTIONING PREDICTS ADAPTIVE FUNCTIONING IN CHILDREN WITH FASD AND PAE

Denys K[1], Zwaigenbaum L[1], Andrew G[1], Tough S[2], Rasmussen C
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2 Department of Pediatrics and Department of Community Health Sciences, University of Calgary

ABSTRACT
Background/Objectives: Adaptive functioning (AF) deficits are common in individuals with FASD. Although IQ is predictive of AF in other populations (Liss et al., 2001)1, this is not the case in FASD. Schonfeld and colleagues (2006)2 found that executive functioning (EF) predicted social skills in individuals with FASD, however, there are no studies linking EF with AF in FASD.

Methods: Participants (aged 6 - 16) were in two groups, FASD (N=41) and prenatal alcohol exposure, not meeting criteria for FASD (PAE; N=26). Parents rated their child's EF and AF, using the Behavioral Rating Inventory of Executive Function (BRIEF) and the Adaptive Behavior Assessment System Second Edition (ABAS-II), respectively. We also examined whether IQ, age of assessment, SES, and home stability were related to AF.

Results: Based on one-way ANOVAs, the PAE group performed significantly better than the FASD group on all of the ABAS composites, the BRIEF Metacognitive Index (MI), and approached significance on the BRIEF Global Executive Composite. Stepwise regression analyses revealed

that the BRIEF MI was the best predictor of the General Adaptive, Conceptual, Practical ABAS-II composites, but nothing predicted and the Social composite. IQ only predicted scores on the Conceptual composite.

Conclusions/Discussion: We found EF to be a better predictor of AF than IQ and other environmental factors. This has implications for policy and services: many disability services are not available to individuals with FASD and PAE because they have IQs above 70, whereas EF measures may be more informative for severe impairments in AF.

Keywords: Executive functioning, adaptive functioning, IQ, FASD, PAE

References:
1. Liss M, Fein D, Allen D, Dunn M, Feinstein C, Morris R, Waterhouse L, Rapin I. Executive functioning in high-functioning children with autism. Journal of Child Psychology and Psychiatry and Allied Disciplines 2001;42:261-270.
2. Schonfeld AM, Paley B, Frankel F, O'Connor MJ. Executive functioning predicts social skills following prenatal alcohol exposure. Child Neuropsychology 2006;12:439-452.

Sources of funding for the study: CIHR Operating Grant
Conflict of interest: None
Student/Trainee: Full-time MSc student
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13th September 2011

5. ECOGENETIC AND EPIGENETIC INFLUENCES IN FASD

Kapalanga J[1,2,3,4], Wong D[1,2], Gandy A[1,2], Avind Singh A[2], Bhoka G[5]
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3 Grey Bruce Health Services, Owen Sound, Ontario;
4 Cambridge Memorial Hospital, Cambridge, Ontario;
5 Obongi Hospital,W Moyo, Uganda

ABSTRACT
Background/Objectives: Alcohol is globally consumed, and its long term adverse effects on fetal development are well characterized. However, in utero alcohol exposure does not always result in Fetal Alcohol Spectrum Disorders (FASD). This study explores the role of ecogenetic and epigenetic factors in FASD. We hypothesize that ecogenetic and epigenetic factors determine susceptibility to FASD.

Methods: To test this hypothesis we examined the prevalence of FASD between two disparate population groups; one in Prince Edward Island (PEI), Canada and the other in Moyo, Northern Uganda. Moyo was chosen because alcohol consumption in the district is especially widespread and yet alcohol-related behavioral and academic issues are rare. In PEI, FASD is a significant public health concern. Both Moyo and PEI are remote regions with stable populations of comparable size. Children aged 2 years to 10 years exposed in utero to alcohol as determined by maternal history were assessed by a dysmophologist/paediatricianfor clinical features of FASD.

Results: Fifty seven (57/87 or 65.5%) Moyo mothers and 49 (49/89 or 55%) PEI mothers admitted to consuming alcohol during pregnancy. Based on maternal history a total of 65 Moyo children and

52 PEI children were exposed in utero to alcohol. Twentysix or 40% Moyo children and 42 or 80% PEI children were confirmed to have FASD.

Conclusions/Discussions: PEI children exposed in utero to alcohol are significantly more likely to have FASD than Moyo children ($p<0.01$). Differences in ecogenetic and epigenetic factors are postulated to be the explanation for the two-fold difference in FASD prevalence between Moyo and PEI children. These findings are preliminary and warrant further and larger studies that analyze ecogenetic and epigenetic factors that could influence risk susceptibility to FASD.

Keywords: Ecogenetics, epigenetics, risk susceptibility, clinic assessment
Source of funding: None
Conflict of interest: None
Corresponding author: jkapalanmdphd@pol.net

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13th September 2011

6. AVAILABLE INTERVENTIONS AND SERVICES USED BY CANADIAN ADOLESCENTS WITH FASD AND THEIR FAMILIES

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2 York University, Department of Psychology,Toronto,Ontario

ABSTRACT
Background/Objectives: FASD is a leading cause of developmental disability; however, little research has focused on management post-diagnosis. This study aims to describe the availability and usage of interventions and services by Canadian adolescents with FASD and their families.

Methods: This observational, follow-up study included 44 adolescents between 11.0 and 18.9 years of age (31 males, 13 females), diagnosed with a FASD at the Motherisk Clinic at The Hospital for Sick Children in Toronto, Canada. Quantitative and qualitative data were collected via semi-structured telephone interviews with primary caregivers. Qualitative data were analyzed using thematic analysis.

Results: Psychopharmacotherapy was the most common type of intervention utilized by adolescents (68%). Commonly used medications included stimulant/non-stimulant attention medications (52%), antipsychotics (43%) and antidepressants (16%). Thirty-two percent of adolescents were receiving individual counselling/therapy and 7% were participating in group interventions. Only 14% of adolescents had ever been referred to or received any FASD-specific interventions/services. Forty-three percent of caregivers had received FASD training and 16% attended caregiver support groups. The mean caregiver rating of accessibility of interventions/services for adolescents was 1.85 (SD = 1.14) on a numerical scale of 1 to 5 (1 = very difficult to get, 5 = very easy to get).

The most common categories of interventions/services reported by caregivers as needed, yet difficult to obtain or unavailable, were FASD-specific interventions/services and social skills interventions.

Discussion/Conclusions: The majority of Canadian adolescents with FASD are not receiving interventions, other than psychopharmacotherapy. Despite significant advancements in the

diagnosis of FASD, little progress has been made in supporting these individuals postdiagnosis.

Keywords: FASD, adolescents, observational
Source of funding for the study: CIHR and Glendon College
Conflict of interest: None
Student/Trainee: Full-time
Corresponding author: michelle.todorow@sickkids.ca

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7. HOW TO APPROACH SLEEP PROBLEMS IN CHILDREN WITH FASD: THE FIRST CANADIAN FASD & SLEEP CONSENSUS PAPER

Ipsiroglu OS[1], Andrew G[2], Carmichael-Olson H[3], Chen M[4], Collet JP[5], Pei J[6], Garden J[1], Hanlon-Dearman A[7], Houben R[8], Jan JE[1], Keivers K[9], McNaughton D[10], Loock C[11], Vitale-Cox L[12], Yo W[13], Veer D[1], Weinberg J[13], Witmans M[14]
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3 Psychiatry and Behavioral Sciences, University of Washington, Seattle;
4 University of Washington School of Medicine, Seattle;
5 Child and Family Research Institute, British Columbia Children's Hospital, Vancouver;
6 Canada NorthWest FASD Research Network/Intervention Team/ Educational Psychology, University of Alberta, Edmonton;
7 Manitoba FASD Centre/ Children's Hospital of Winnipeg, Winnipeg;
8 Health2Media, Vancouver;
9 University of the Fraser Valley, Abbotsford;
10 Social Work, Children's & Women's Health Centre of British Columbia, Vancouver;
11British Columbia Children's Hospital, Sunny Hill Health Center for Children, Vancouver;
12 Elispogtog First Nation Education Division EHWC Eastern Door Diagnostic Team New Brunswick, New Brunswick;
13 Department of Cellular and Physiological Sciences, University of British Columbia, Vancouver;
14 Stollery Children's Hospital, University of Alberta, Edmonton

ABSTRACT

Background: Although up to 85% of children with FASD experience sleep problems (SP) which significantly impact functioning and quality-of-life (QoL), SP are rarely addressed by health care providers (HCP). In order to develop a framework for a structured approach to SP and to determine skills and knowledge necessary to diagnose and treat children with SP and/or disorders, an interdisciplinary consensus meeting was organized prior to the 4th International FASD Conference in Vancouver (2011).

Methods: Existing published/submitted clinical research exploring the problem and trends of medical treatment (medication) were reviewed. Based on the Canadian Health Care Systems' universal services philosophy, a 3-Level-Curriculum was proposed and the needs for future research and knowledge dissemination were discussed as a guideline for screening/assessing SP.

Results: Level I includes screening with a focus on behaviour, day- and night time situations and a simple measure for quality-of-life. All HCPs should be "empowered to close the screening based sleep service- related gaps at the level of care they offer". Level II includes assessments by HCPs

(e.g. occupational/behavioural therapist or community paediatrician) using sleep-logs/-diaries as clinical monitoring/evaluation tools and validated sleep questionnaires, thus requires a formal training. Level III represents regional health care services and is the highest level of the curriculum in regards to structured knowledge dissemination.

Discussion: Knowledge dissemination is needed to enable this proposed approach and will be provided through the collaboration of the Consensus Group with the Intervention Team of the Canada Northwest FASD Research Network. Evaluation of activities will guide and coordinate future research needs.

Keywords: Sleep problems, 3-Level-Curriculum, knowledge dissemination
Source of funding: Victoria Foundation FASD-Action-Fund
Conflict of interest: None
Corresponding author: oipsiroglu@cw.bc.ca

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8. RESTLESS LEG SYNDROME (RLS) IN CHILDREN AND YOUTH WITH FASD AND PRENATAL SUBSTANCE EXPOSURE (PSE) – A CLINICALLY MISSED DIAGNOSIS AGGRAVATING THE CHALLENGING BEHAVIOUR?

Ipsiroglu OS[1], Black A[2], Garden J[1], Jan JE[1]
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ABSTRACT
Background: RLS can cause sleep problems (SP), and is a "disorder characterized by disagreeable leg sensations that usually occur prior to sleep onset and that cause an almost irresistible urge to move the legs." Sleep related day- and night-time symptoms may not be recognized, or are missed in children with FASD/PSE, as FASD/PSE is usually associated with challenging behaviour including daytime hyperactivity and bedtime resistance. Results of clinical sleep assessments suggest that optimizing our understanding before triaging patients for further diagnostic/therapeutic care would be helpful.

Methods: We initially used an ethnographic approach adapted from medical anthropology to explore parent(s)/caregiver(s) perceptions of "challenging behaviour" and SP; then we developed and piloted home-based over-night-video-sleep-studies with which we could describe observable and reproducible SP; out of the video-reports we developed standardized descriptions.

Results: We are describing day- and night-time related RLS related clinical symptoms in 27 patients with FASD/PSE: "tossing and turning around"; "messy bed"; "fighting sleep"; 'restless sleep" etc; 23/27 patients had insomnia, 22/27 parasomnias, 22/27 had symptoms strongly suggestive of RLS; 12 cases were diagnosed, 10 cases are re-evaluated.

Discussion: RLS related discomfort/urge-to-move/pain seems to be a main cause of SP. Children may develop movement based abilities and/or extreme adaptive movement strategies (trampoline jumping until collapsing) to overcome difficulties falling asleep, which are interpreted as part of their challenging behaviour. History and analysis of behavioural patterns in conjunction seems to be a key in patients with expressive language difficulties. Our observations open a new causality related diagnostic/therapeutic care option.

Keywords: RLS, challenging behaviour, sleep

Source of funding: Victoria Foundation FASD-Action-Fund
Conflict of interest: None
Corresponding author: oipsiroglu@cw.bc.ca

Link to the Article,
http://www.cjcp.ca/pubmed.php?articleId=332

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9. INSOMNIA & RESTLESS LEGS SYNDROME (RLS) IN CHRONIC CARE MANAGEMENT OF CHILDREN AND ADOLESCENTS WITH NEURODEVELOPMENTAL CONDITIONS. AN APPLIED MEDICAL ANTHROPOLOGY AND DEVELOPMENTAL PEDIATRICS (AMA/DP) COURSE FOR UNDERGRADUATES

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3 Department of Anthropology, University of British Columbia, Vancouver

ABSTRACT
Background: The purpose of this course is to introduce undergraduate students to the concepts of AMA/DP in chronic care management of children with neurodevelopmental conditions (NDC), such as FASD. Special attention was given to the topics of insomnia due to RLS in MA/DP research context and to analytic matters as the nature of description, conceptualization, generalization and content analysis.

Methods: Selected methods from AMA/DP were applied to observe, describe, and interpret the medical phenomena of RLS in cultural and medical context and social organization in children and youth with NDC. The course covered participant observation, disease and life histories, narratives, ethnographic semantics, information gathering techniques, with a focus on narratives, videos, and questionnaires.

Results: Undergraduate students were able to develop 1) new perspectives to NDC and/or to patients who have different life experiences, and understand how to interpret and make use of obtained qualitative and quantitative information, and 2) their own focused research questions, and try new explorative research tools in their own areas of expertise (e.g. nutritional sciences, pharmacology, kinesiology, …) presented at www.chroniccare4sleep.org .

Discussion: Sleep spans across various organ-specific conditions and as a theme has involved most disciplines and natural sciences, including humanities and arts, but in medicine the content has been 'medicalized' from subspecialty viewpoints. In applied AMA/DP the focus is more on the contextual relationship of the biological experience sleep with the concept of medicalization, along with cultural and social phenomena helping individuals broaden their understanding and overcome personal/professional prejudices.

Keywords: Undergraduate training, applied medical anthropology, developmental paediatrics
Source of funding: Victoria Foundation FASD-Action-Fund
Conflict of interest: None
Corresponding author: oipsiroglu@cw.bc.ca

Link to the Article,
http://www.cjcp.ca/pubmed.php?articleId=332

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10. THE BRIDGE THE GAPS APPROACH. WEB-BASED HELP FOR PARENTS AND HEALTH CARE PROFESSIONALS (HCP) FOR SLEEP PROBLEMS IN CHILDREN & ADOLESCENTS WITH FASD AND OTHER NEURODEVELOPMENTAL CONDITIONS

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3 FASD & Sleep Research Group BCCH/SHHCC, University of British Columbia, Vancouver/Department of Anthropology, University of British Columbia, Vancouver;
4 Health2Media, Vancouver; 5British Columbia Children'sHospital, Sunny Hill Health Center for Children, Vancouver

ABSTRACT

Background: Individuals with FASD and other neurodevelopmental conditions are at high-risk for developing sleep problems (SP). In consequence the sleep deprivation leads to additional co-morbidity, e.g. hyperactive behaviour, and further physical/cognitive/emotional impairments. Our research/clinical experience has shown that clinical symptoms and behaviour are often not recognized as sleep related, and that parents/caregivers' reports about SP are not given appropriate attention by HCPs. Children are often medicated with psychotropic substances without critical evaluation of the benefit to potential harm.

Methods: The website www.chroniccare4sleep.org is a portal and an information exchange source for SP experienced by children and adolescents with neurodevelopmental conditions; the FASD-pages are the first to go public (September 10, 2011). Published literature reviews, developed and existing screening questionnaires can be downloaded for parents'/HCPs' use. Presented information has been peer reviewed and/or is based on own experience accepted as "standard-of-care".

Results: We predict that providing parents/caregivers and HCP with different levels of information will help to bridge the current situation of neglect in regards to SP and give us valuable information about needs/requirements. The website-counter and received feedback enables us to monitor interest, acceptance and improve presentations on an ongoing-basis.

Discussion: Besides anxiety, behaviour, airway obstructions, discomfort/pain, RLS and other organic causes paired with socio-cultural factors can contribute to SP. We believe that directly enabling target groups to focus more specifically on SP and use a shared language will help to gain new information regarding the causes of challenging behaviour, mainstream medication trends and SP.

Keywords: www.chroniccare4sleep.org, medication, sleep problems

Source of funding: Victoria Foundation FASD-Action- Fund

Conflict of interest: None
Corresponding author: oipsiroglu@cw.bc.ca

Link to the Article,
http://www.cjcp.ca/pubmed.php?articleId=332

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11. BODY AND HEAD MOVEMENTS DURING SLEEP AND WAKE EPISODES IN A RAT MODEL OF FASD: PILOT FOR PROVING THE VIDEO MONITORING CONCEPT

Hung A[1], Chan F[1], Yo W[2], Ipsiroglu OS[1], Weinberg J[2]
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2 Department of Cellular and Physiological Sciences University of British Columbia, Vancouver

ABSTRACT

Background: Based on our clinical observation that Restless Legs Syndrome (RLS, a neurologic sensorimotor disorder characterized by a range of sensations from discomfort to pain) is a major cause of insomnia in children and adolescents with FASD, we hypothesized that rat offspring with prenatal alcohol exposure (PAE) may exhibit altered movements during sleep compared to those in control animals. To investigate this, we first had to test the applicability of our overnight-video-sleep studies system for monitoring activities/ sleep of rats.

Methods: 2 control, 2 PAE male Sprague Dawley rats were videotaped in specially equipped cages for a total of 8 hrs during the lights on period. Equipment included infrared-light camera, netbook with synchronized audio/video software, and live time-stamp, constant frame-rates.

Results: Awake/asleep states, defined as open/closed eyes and responsive/unresponsiveness were identified. Overall, the following were found: awake: 146/140 min, asleep: 267/278 min, transitioning between awake/sleep: 64/63 min. We were able to observe behavioural patterns including prolonged grooming/head shivering activities before falling asleep, arousals and some active limb movements while asleep, which appeared to differ between PAE and control animals. We were not able to count leg movements reliably as sleeping position hindered observation of lower/upper limbs.

Discussion: The video methodology opens a new option for behavioural-sleep-observations in the rat model. While head and body movement can clearly be described, the study must be repeated with a larger sample size, and the relationship of these observations to RLS must still be demonstrated.

Keywords: Rat model of FASD, sleep, behavioural observations
Source of funding: Victoria Foundation FASD-Action-Fund, NIH/NIAAA R37AA007789
Student/Trainee: Trainee
Conflict of interest: None
Corresponding author: oipsiroglu@cw.bc.ca

Link to the Article,
http://www.cjcp.ca/pubmed.php?articleId=332

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BBC News, Health, 20th June 2012

A. MODERATE DRINKING IN EARLY PREGNANCY BRANDED 'SAFE'

Drinking a low or moderate level of alcohol in early pregnancy is not linked to developmental problems in five-year-olds, researchers say.

Women were asked about drinking in early pregnancy

The Danish research, published in the BJOG journal, suggested one to eight drinks a week was not linked to harm.

In Denmark a standard drink has 12g of alcohol, compared with the UK's 7.9g.

UK pregnant women are advised not to drink, but experts say those who do should have no more than one or two units, once or twice a week.

Heavy drinking during pregnancy is known to be linked to miscarriage, foetal alcohol syndrome and low birth weight.

Binge drinking
The Danish researchers produced five papers on drinking in pregnancy.

More than 1,600 pregnant women took part, recruited at their first antenatal visit. Half were first-time mothers, and just under a third smoked during pregnancy.

They were asked about their alcohol intake.

Low average consumption was defined as one to four per week, moderate as five to eight drinks and high levels as nine or more per week.

Binge drinking, which women were also questioned about, was defined as having five or more drinks on one occasion. Pregnant women who did not drink during pregnancy were included in the research.

The scientists looked at the effects of alcohol on IQ, attention span, executive functions such as planning, organisation, and self-control in the five-year-olds.

They found low to moderate weekly drinking in early pregnancy had no significant effect on neurodevelopment of children at the age of five - and neither did binge drinking.

There were no differences in IQ test results in children whose mothers drank one to four units per week or five to eight units per week in pregnancy compared with children of abstaining mothers.

But drinking more than nine drinks per week was associated with lower attention span among the children.

'Minimising risk'
The lead authors of the work, Ulrik Schiøler Kesmodel of Aarhus University and Prof Erik Lykke Mortensen of the University of Copenhagen, said: "High prenatal exposure to alcohol has consistently been associated with adverse effects on neurodevelopment.

"Areas such as intelligence, attention and executive functions have been found to be particularly vulnerable.

"Our findings show that low to moderate drinking is not associated with adverse effects on the children aged five."

Patrick O'Brien, a spokesman for the Royal College of Obstetricians and Gynaecologists (RCOG) and a consultant obstetrician said the research was very well designed.

This was because it asked women about their alcohol intake at the time - rather than asking them to look back as past studies have done - and because it followed children for such a ling time and assessed such a range of developmental markers, he said.

The RCOG advises that women abstain from alcohol while pregnant, but if they do decide to drink evidence suggests "one or two units, once or twice a week, is acceptable after 12 weeks of pregnancy".

Dr O'Brien said: "These findings suggest low to moderate drinking has no significant effect on children aged five. However, this does not mean that women can use this as an excuse to indulge in more than the recommended amount in the UK.

"This evidence suggests that the UK guidance is erring on the side of caution - but that's sensible in pregnancy."

A spokeswoman for the Department of Health echoed the RCOG advice, but said it would always "take note" of new evidence.

Women are advised to talk to their midwife or doctor if they have any concerns about the amount of alcohol they are drinking in pregnancy.

Read Full Article,
http://www.bbc.co.uk/news/health-18506174

RESPONSE FROM PROFESSORS THERESA GRANT AND SUSAN ASTLEY

DANISH STUDIES SUGGEST LOW AND MODERATE DRINKING IN EARLY PREGNANCY HAS NO ADVERSE EFFECTS ON CHILDREN AGED FIVE

Is it safe to drink alcohol during pregnancy? A new Danish study on alcohol and pregnancy reports that drinking up to 8 alcoholic drinks per week during pregnancy has no effect on children's intelligence or activity levels. But this goes against everything we have heard about the dangers of drinking during pregnancy. What about the Surgeon General's warning on liquor bottles? *"Women should not drink alcoholic beverages during pregnancy because of the risk of birth defects"*. How are we to trust medical research if we receive conflicting messages like these?

Let's start with the most important question…*Is it safe to drink during pregnancy?* The answer is NO. And if you look closely, even the Danish researchers come to this conclusion. But nevertheless, press headlines read "Moderate drinking in early pregnancy branded 'safe': BBC News". Most women in the U.S. who drink alcohol quit when they're pregnant. But some women will be influenced by press like this.

So is the Danish study wrong? Well, let's look at it in more detail. It was an impressively large study. Mothers were interviewed during pregnancy about their alcohol use, and about half of them agreed to participate in the follow-up study five years later. The researchers studied 870 preschool

children whose mothers reported drinking during pregnancy and compared them to 758 preschool children whose mothers reported not drinking during pregnancy. They measured the children's IQ and attention levels at age 5 years. Children exposed prenatally to 1-8 drinks per week had the same IQ and attention levels as children with no exposure to alcohol. **The reason the children in this study did not appear to be harmed by the alcohol is because the children were too young to measure the full impact alcohol may have had on their brains.** At 5 years of age, the brain is still developing. A 5-year-old's brain is not developed enough to perform complex tasks like remembering and following multiple instructions, writing an essay, communicating abstract ideas effectively, exercising good judgment. Over 30 years of research on fetal alcohol syndrome (FAS) confirms that alcohol has its greatest impact on complex brain functions. This is why children exposed to and damaged by prenatal alcohol exposure look deceptively good in the preschool years. The full impact of their alcohol exposure will not be evident until their adolescent years.

So if the news reports have you believing that 1-8 drinks per week during pregnancy are safe, please consider the following. The statistics below are based on 2,600 children who received a diagnostic evaluation for FAS in the Washington State FAS Diagnostic & Prevention Network clinics over the past 18 years.

- 1 out of every 7 children diagnosed with FAS (the most severe outcome caused by prenatal alcohol exposure) had a reported exposure of 1-8 drinks per week. (The Danish study did not conduct FAS diagnostic evaluations on the children).
- Half of the children with FAS had developmental scores in the normal range as preschoolers. But all had severe brain dysfunction confirmed by age 10. (The Danish study only assessed preschoolers).
- Only 10% of the children with FAS had attention problems by age 5. 60% had attention problems by the age of 10. (The Danish study only assessed attention at age 5).
- Only 30% of the children with FAS have an IQ below normal. But 100% had severe dysfunction in other areas like

Which children are most vulnerable? We have no way of knowing because risk is not just based on how much alcohol the mother drank. We know from twin studies that genetics also plays a role. When genetically different twins are exposed to the same levels of alcohol, one twin can be born with FAS while the other twin is normally developed. We also know that metabolism plays a part. Every person absorbs and metabolizes alcohol differently, and a pregnant woman simply can't know how "just one drink" might be affecting her developing fetus.

So, while the science may be complicated and studies sometimes yield conflicting messages, the message for women is simple: to have *the healthiest baby possible*, don't drink alcohol when you're trying to get pregnant and during pregnancy. When a pregnant woman drinks, her child is at risk. If she drinks heavily, her child is at higher risk.

FAS was first identified at the University of Washington in 1973. Washington State, through a collaboration between the State and University, has led the field in FAS diagnosis, intervention, and prevention since that time.

If you are pregnant or trying to get pregnant and you drink alcohol, you should stop. If you cannot stop drinking, please contact us! We are here to help you and your family.

Susan Astley Ph.D. is a professor of Epidemiology and Pediatrics and director of the WA State FAS Diagnostic & Prevention Network of clinics (fasdpn.org). Therese Grant Ph.D. is the Ann Streissguth Endowed Professor in Fetal Alcohol Spectrum Disorders and director of the Fetal Alcohol and Drug Unit in the Dept. of Psychiatry & Behavioral Sciences (http://depts.washington.edu/fadu/). Both are research affiliates at the Center on Human Development & Disability at the University of Washington.

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J Popul Ther Clin Pharmacol Vol 19(2):e223;
June 3, 2012
Gideon Koren

B. THE FIRST DESCRIPTION OF FETAL ALCOHOL SYNDROME BY FRENCH PEDIATRICIAN PAUL LEMOINE

Dr. Lemoine's account demonstrates how much knowledge about the toxic fetal effects of alcohol was published and known in Europe in the early 1900's, half a century before it became public knowledge and a public health issue.

Read Full Article,
http://www.jptcp.com/pubmed.php?articleId=372

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J Popul Ther Clin Pharmacol Vol 19(2):e224-e226;
June 3, 2012
Paul Lemoine

C. THE HISTORY OF ALCOHOLIC FETOPATHIES

Dr Paul Lemoine was the Chief of the Pediatric Service at the Centre Hospitalier Universitaire (CHU) in Nantes, France.

During this time he noted the effects of parental alcoholism on offspring outcome. Here we publish his personal account of researching the cause of a strange dystrophy around the 1960's with certain children and alcoholism in their mothers.

Read Full Article,
http://www.jptcp.com/pubmed.php?articleId=373

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The Republic,
The Associated Press
First Posted: April 15, 2012 - 10:32 pm

D. ALASKA LEGISLATURE APPROVES BILL TO ALLOW JUDGES TO CONSIDER FASD IN SENTENCING

JUNEAU, Alaska — The Alaska Legislature has approved a bill that would give judges flexibility when handing down criminal sentences to people with fetal alcohol spectrum disorders. Judges are not currently allowed to consider conditions caused by prenatal exposure to alcohol during sentencing. Senate Majority Leader Kevin Meyer introduced SB151 after working with judges, activists and researchers. He says the bill was carefully worded to give judges more options without letting criminals get off easy. SB151 passed the Senate earlier this month and passed the House unopposed on Sunday.

Link to the Article,
http://www.therepublic.com/view/story/7641ff2d03ed4473926a2f3677d50cac/AK-XGR--FASD-in-Alaska/

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Native American Times, Grant Schulte, Associated Press
27th February 2012

E. TRIBE SEEKS ORDER TO LIMIT BEER SALES

In this June 3, 2004, file photo, Pine Ridge police officers Mirian Laybad, left, Sgt. Oscar Hudspeth, center, and Lt. Mitch Wisecarver confiscate cases of beer at a checkpoint just north of Whiteclay, Neb. The Oglala Sioux Tribe announced Thursday, Feb. 6, that it will file a $500 million federal lawsuit against some of the nation's largest beer distributors, alleging that they knowingly contributed to the chronic alcoholism, health problems and other social ills on the Pine Ridge Indian Reservation. The lawsuit also targets the four beer stores in Whiteclay, a Nebraska town (pop. 11) on the South Dakota border that sells about 5 million cans of beer per year. WILLIAM LAUER/LINCON JOURNAL STAR FILE PHOTO

One in four children born on the reservation suffer from fetal alcohol syndrome.

LINCOLN, Neb. (AP) – Leaders of a South Dakota American Indian tribe who are suing beer makers, distributors and retailers are now asking a judge to restrict alcohol sales in a tiny Nebraska town that borders their reservation.

The Oglala Sioux Tribe added the request late last week to a federal lawsuit that seeks $500 million in damages for the alcohol-related problems on the Pine Ridge Indian Reservation.

The tribe's attorney, Tom White of Omaha, said he will argue that Nebraska officials have failed to enforce their own laws by allowing beer sales that far surpass the amount that can legally be consumed in the area.

Alcohol is officially banned on Pine Ridge, a reservation the size of Connecticut in southwestern South Dakota. Nebraska state law prohibits drinking outside of the stores, and the nearest non-reservation town is more than 20 miles to the south. Yet the four beer retailers in Whiteclay, which has fewer than a dozen residents, sold the equivalent of nearly 5 million cans in 2010.

"The defendants have failed to make reasonable efforts to ensure their products are distributed and sold in obedience to the laws of the state of Nebraska and the Oglala Sioux tribe," White said Feb. 22.

The lawsuit says the tribe has "no adequate remedies to protect its federally granted rights" to protect its sovereignty and enforce its own alcohol ban. He pointed to statements by Attorney General Jon Bruning, the state's top law enforcement officer, who has said shutting down the beer stores will not solve the problem.

The lawsuit in U.S. District Court of Nebraska targets some of the world's largest beer makers, as well as their distributors and the four stores in Whiteclay.

Tribal leaders and activists blame the Whiteclay businesses for chronic alcohol abuse and bootlegging on the Pine Ridge. They say most of the stores' customers come from the reservation, which spans southwest South Dakota.

The $500 million lawsuit seeks reimbursement for the cost of health care, social services and child rehabilitation caused by chronic alcoholism on the reservation, which encompasses some of the nation's most impoverished counties.

One in four children born on the reservation suffer from fetal alcohol syndrome or fetal alcohol spectrum disorder, and the average life expectancy is estimated between 45 and 52 years – the shortest in the North Hemisphere except for Haiti, according to the lawsuit. The average American life expectancy is 77.5 years.

The lawsuit alleges that the beer makers and stores sold to Pine Ridge residents, knowing they would smuggle the alcohol into the reservation to drink or resell.

Matthew Fletcher, a Michigan State University associate law professor who specializes in American Indian issues, said the suit offers a novel approach to a problem that has plagued the reservation for more than a century. But, Fletcher said, "my sense is, it doesn't have much of a chance."

For years, Nebraska lawmakers have struggled to curb the problem, and are considering legislation that would limit the types of alcohol sold in areas like Whiteclay. The measure would require local authorities to ask the state to designate the area an "alcohol impact zone." The state liquor commission could then limit the hours that alcohol sellers are open, as well as ban the sale of certain products.

The beer store owners, distributors and retailers have all declined to comment.

Link to the Article,
http://www.nativetimes.com/news/tribal/6902-tribe-seeks-order-to-limit-beer-sales

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HarvardScience
By Peter Reuell, Harvard Staff Writer
Wednesday, January 18, 2012

F. CLUES TO ADDICTION. RESEARCH SHOWING HOW NEURONS INTERACT COULD LEAD TO NEW TREATMENTS

Harvard scientists have developed the fullest picture yet of how neurons in the brain interact to reinforce behaviors ranging from learning to drug use, a finding that might open the door to new treatments for addiction.

The finding is the result of a yearlong effort by a team of researchers led by Naoshige Uchida, associate professor of molecular and cellular biology, to examine a brain process known as reward prediction error. Thought to be a key component of learning, prediction error has long been considered the product of dopamine neurons firing in response to an unexpected "reward," thus reinforcing the behavior that led to the reward.

But Uchida and colleagues from Harvard and Beth Israel Deaconess Medical Center report in the Jan. 18 issue of Nature that reward prediction error is actually the product of a complex interplay between two classes of neurons — one that relies on dopamine and an inhibitory class of neurons that uses the neurotransmitter GABA.

"Until now, no one knew how these GABA neurons were involved in the reward and punishment cycle," Uchida said. "What we believe is happening is that they are inhibiting the dopamine neurons, so the two are working together to make the reward error computation."

Before Uchida and his team could prove that GABA neurons were involved in the computation, however, they had to be sure what type of cells they were observing.

Jon Chase/Harvard Staff Photographer
Naoshige Uchida (above) and colleagues report in the Jan. 18 issue of Nature that reward prediction error is actually the product of a complex interplay between two classes of neurons — one that relies on dopamine and an inhibitory class of neurons that uses the neurotransmitter GABA.

The challenge in studying both dopamine and GABA neurons is that the two cell types are intermingled in a relatively small area of the brain, making it difficult for researchers to definitively know which type they are observing. Ultimately, researchers developed an elegant solution to the problem.

Researchers genetically altered the neurons in two groups of mice — one for the dopamine neurons, the other for GABA neurons — to fire when hit by a pulse of laser light. Once researchers were certain they were measuring the correct type of neuron, they used electrodes to measure whether and when the neurons fired in response to expected and actual rewards.

The results, Uchida said, showed that while firing of dopamine neurons signaled reward prediction error, firing of GABA neurons signaled an expected reward. Taken together, GABA neurons help dopamine neurons calculate reward prediction error.

The finding is particularly important, Uchida said, because it sheds new light on how behaviors can be reinforced, either through normal brain function, or by damaging the way the two types of neurons interact.

“What happens with drug abuse is that many drugs, such as opioids and cannabinoids, target the GABA neurons,” he said. “What we are hypothesizing is that, by inhibiting those GABA neurons, you can lose this feedback cycle, so you keep getting reinforcing signals from the dopamine neurons.

“This is a new way of thinking about addiction in general,” Uchida continued. “Based on this theory, I believe you may be able to develop new theories or treatments for addiction.”

Funding for the research was provided by the Howard Hughes Medical Institute, the Human Frontier Science Program, the Richard and Susan Smith Family Foundation, the Alfred P. Sloan Foundation, and the Milton Fund.

Link to the Article,
http://news.harvard.edu/gazette/story/2012/01/clues-to-addiction/

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The Partnership at Drugfree.org
By Join Together Staff | January 4, 2012

G. PROGRAM FOR PREGNANT WOMEN AT RISK OF SUBSTANCE ABUSE COULD LEAD TO BIG COST SAVINGS

A prenatal intervention program, for stopping substance use in pregnancy, could save almost $2 billion annually if it were implemented nationwide, a new study suggests.

The Kaiser Permanente Early Start program helps women at risk of substance abuse to achieve health outcomes for mothers and babies that are similar to women who do not use cigarettes, alcohol or drugs, Medical News Today reports. The study of almost 50,000 women found the program decreases illness in mothers and their babies, as well as stillbirths.

“Now, we’re able to show everyone that not only is it the right thing to do, we will save money,” lead author Nancy C. Goler, MD, said in a news release. “This program is a very low-technology intervention that has an enormous net cost savings.”

The program screens pregnant women with urine tests and substance abuse screening questionnaires. It is located in the same clinic where women receive their prenatal care, and a licensed substance abuse expert sees patients at the same time as their prenatal care appointments. All health care providers and patients in the program are educated about the effects of alcohol, drug and cigarette use during pregnancy.

The findings are published in the journal Obstetrics & Gynecology.

Link to the Article,
http://www.drugfree.org/join-together/addiction/program-for-pregnant-women-at-risk-of-substance-abuse-could-lead-to-big-cost-savings

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ScienceNetwork, Western Australia
Written By Geoff Vivian
Friday, 16 December 2011 12:00

H. FITZROY CHILDREN CENTRE OF FOETAL ALCOHOL DISORDER STUDY

ABORIGINAL organisations in Fitzroy Valley have called for research into social, health and wellbeing issues associated with alcohol abuse.

Paediatrician James Fitzpatrick is leading one investigation into Foetal Alcohol Spectrum Disorders (FASD) in Fitzroy Valley children.

“The effects of alcohol in pregnancy are the leading cause of environmental intellectual impairment worldwide,” Dr Fitzpatrick says.

“The foetus can suffer permanent brain damage, as well as other birth defects affecting the face, heart, lungs, kidneys and musculoskeletal system.”

Dr Fitzpatrick says his research is part of the Lililwan Project, a program initiated by Aboriginal women’s organisation Marninwarntukuru and Nindilingarri Cultural Health Service.

“High-risk drinking behaviour in some remote communities was leading to rates of FASD that could wipe out an entire generation's chance of a healthy and productive life,” Dr Fitzpatrick says.

The effects of alcohol in pregnancy are the leading cause of environmental intellectual impairment worldwide — Dr Fitzpatrick.

“Compounding this is the threat to Aboriginal culture that relies on learned stories and traditions.”

The team designed an active case ascertainment population-based prevalence study to determine the problem’s prevalence in the community.

Children born from 2001-2002 who were living in Fitzroy Valley communities in 2009-2010 were tested.

Ninety-five per cent of that age group were properly assessed.

“We expect to find out accurately the number of children in this age range exposed to alcohol during pregnancy, as well as the proportion of these children with one of the three specific diagnoses along the FASD spectrum,” Dr Fitzpatrick says.

Foetal Alcohol Syndrome, Partial Foetal Alcohol Syndrome, and Neurodevelopmental Disorder-Alcohol Exposed are the three types of FASD.

An exhaustive three hour questionnaire was completed with the help of each child’s parent or carer, to ascertain whether the mother had any health or other issues during pregnancy.

“We also asked questions about the child’s behaviour and learning and educational outcomes,” Dr Fitzpatrick says.

“In addition to these questionnaires each child participated in approximately six hours of clinical assessments.”

He says these included examinations by ophthalmologists and audiologists, and assessments by paediatricians, speech and language therapists, occupational therapists, child psychologists and physiotherapists.

“We have an on-going relationship with the community in the establishment of a community model of care for supporting children who have FASD, and also in following up on the referrals and recommendations that we made during the study.”

The final report will be submitted to government in June 2013, with research papers due to come out during 2012.

Link to the Article,
http://sciencewa.net.au/topics/health-a-medicine/item/1127-fitzroy-children-centre-of-foetal-alcohol-disorder-study

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Wayne State University, Public Relations
December 5, 2011

I. WAYNE STATE UNIVERSITY RESEARCH TEAM RECEIVES NEW NIH GRANT TO STUDY NUMERICAL PROCESSING IN INFANTS WITH FETAL ALCOHOL DISORDERS

DETROIT - Fetal alcohol spectrum disorders (FASDs) are one of the most common causes of birth defects worldwide and are particularly prevalent in some South African communities where heavy drinking during pregnancy is a major public health issue, particularly in the wine-growing areas of the Western Cape.

FASDs have long-term, significant effects on neurocognitive and behavioral development, including problems with attention, learning, memory and social skills. They can also cause heart defects, facial dysmorphic features, poor growth, and decreased muscle tone and coordination. A team of researchers led by Sandra W. Jacobson, Ph.D., and Joseph L. Jacobson, Ph.D., professors of psychiatry and behavioral neurosciences in Wayne State University's School of Medicine recently received a $413,440 grant from the National Institute on Alcohol Abuse and Alcoholism of the National Institutes of Health to conduct a new study designed to improve the diagnosis of FASDs. Improved diagnosis can lead to the development of better-targeted treatments for specific deficits found in children with these disorders.

According to S. Jacobson, infants as young as 5 months of age can look at a display of stimuli that involve simple numbers and mentally manipulate them. However, alcohol-exposed infants do not show the same ability to process this numerical information when shown the same stimuli.

"Infants exposed to heavy prenatal alcohol exposure do not exhibit the same response as non-exposed infants," said Jacobson. "In the newly funded study, we will use event-related potentials (ERP) that measure brain waves to examine the time course and specific components of information processing in alcohol-exposed and non-exposed infants. We then can specify which components of number processing are affected by fetal alcohol exposure."

The study will examine whether magnitude comparison, which is the ability to detect larger or smaller quantities, and/or error monitoring, which is an early precursor of executive function, are affected when the exposed infants perform a simple numerical discrimination task. The team's earlier research shows that alcohol-exposed infants do not perform well on this test. The ERP version of this test will help determine which aspects of numerical processing are likely impaired by the alcohol exposure.

This type of research on these two early-developing potential neurocognitive biobehavioral markers could improve understanding of what is impaired by studying specific aspects of central nervous system function that can be linked biologically to fetal alcohol exposure. This, in turn, can provide important information about the pathophysiology of FASDs and contribute to the development of improved treatments for the specific deficits of this disorder.

"Dr. S. Jacobson's research is addressing a critical research area that currently lacks specific diagnostic criteria and an understanding of the neural structures that underlie specific cognitive deficits due to repeated fetal alcohol exposure," said Hilary Ratner, Ph.D., vice president for

research at Wayne State University. "She is a leader in the field of fetal alcohol research and this is another example of the high impact research Wayne State University faculty are engaged in."

Wayne State University is one of the nation's pre-eminent public research institutions in an urban setting. Through its multidisciplinary approach to research and education, and its ongoing collaboration with government, industry and other institutions, the university seeks to enhance economic growth and improve the quality of life in the city of Detroit, state of Michigan and throughout the world. For more information about research at Wayne State University, visit http://www.research.wayne.edu/.

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Link to the Article,
http://media.wayne.edu/2011/12/05/wayne-state-university-research-team-receives-new

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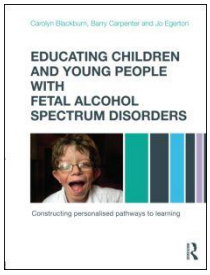
FASD BOOKS AND SEGMENTS

❖ EDUCATING CHILDREN AND YOUNG PEOPLE WITH FETAL ALCOHOL SPECTRUM DISORDERS

Constructing Personalised Pathways to Learning
By Carolyn Blackburn, Barry Carpenter, Jo Egerton
Published May 7th 2012 by Routledge – 114 pages



The range of learning difficulties associated with children and young people who have Fetal Alcohol Spectrum Disorders (FASD) has been highlighted as an emerging but little understood area of Special Educational Needs.

This engaging, timely and highly practical book will raise awareness about FASD and its associated difficulties across the entire education workforce. It provides a range of specialist, tried-and-tested practical teaching and learning strategies from which teachers and support staff may construct personalised learning plans for students with FASD, and will help improve outcomes for all their children. It also:

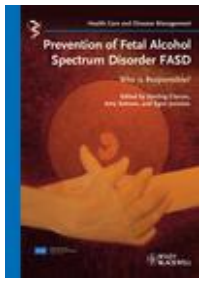
- Explains the impact that FASD can have on the child's brain.
- Discusses the overlapping and co-existing disorders, such as ADHD and autism spectrum disorders.
- Shows how to support and empower teachers.
- Provides ready to use teaching resources and strategies that can be used directly in the classroom.

Informed by the very latest research and written by leading experts in the field, Educating Children and Young People with Fetal Alcohol Spectrum Disorders will prove invaluable for experienced teachers and teaching assistants who are engaging in Continuing Professional Development, newly qualified and training Initial Teacher Training students.

http://www.routledge.com/books/details/9780415670203/

❖ PREVENTION OF FETAL ALCOHOL SPECTRUM DISORDER FASD: WHO IS RESPONSIBLE?

Sterling Clarren (Editor), Amy Salmon (Editor), Egon Jonsson (Editor)
ISBN: 978-3-527-32997-7
Hardcover, 384 pages
April 2011



The perfect complementation to "Fetal Alcohol Spectrum Disorder" this title for the first time gives a complete overview of prevention strategies of FASD and their effectiveness on a global scale. A real Must-Have for everyone involved in decisions about FASD prevention measures.

http://eu.wiley.com/WileyCDA/WileyTitle/productCd-3527329978.html

❖ **FETAL ALCOHOL SPECTRUM DISORDERS: UNDERSTANDING THE EFFECTS OF PRENATAL ALCOHOL EXPOSURE**

National Institute of Alcohol Abuse and Alcoholism (NIAAA)
Alcohol Alert
Number 82

Source material for this Alcohol Alert originally appeared in Alcohol Research & Health, 2011, Volume 34, Number 1.

Alcohol Research & Health, 34(1) "Fetal Alcohol Spectrum Disorders" examines the latest research on prenatal alcohol exposure. Articles explore what is known about the harmful effects of drinking during pregnancy. Includes a look at the latest technology and how it is helping scientists to better understand the effects of alcohol on the developing brain. Articles also describe new diagnostic tools for identifying children at risk for FASD as well as the latest findings in the treatment, intervention, and prevention of these harmful effects

For more information on the latest advances in alcohol research, visit NIAAA's Web site, www.niaaa.nih.gov

http://pubs.niaaa.nih.gov/publications/AA82/AA82.htm

❖ **ALCOHOL IN THE EUROPEAN UNION - CONSUMPTION, HARM AND POLICY APPROACHES**

EURO NON SERIAL PUBLICATIONS
Anderson, P., Moller, L., Galea, G.
WHO Regional Office for Europe
ISBN-13 9789289002646
ISBN-10 9289002646
Order Number 13400118
Format Paper Back

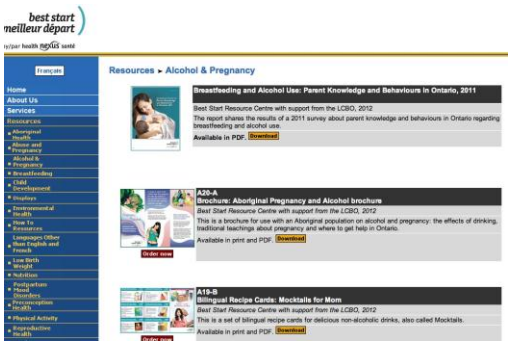
This new report uses information gathered in 2011 to update key indicators on alcohol consumption, health outcomes and action to reduce harm across the European Union (EU). It gives an overview of the latest research on effective alcohol policies, and includes data from the EU, Norway and Switzerland on alcohol consumption, harm and policy approaches. The data were collected from a 2011survey, carried out as part of a project of the European Commission and the WHO Regional Office for Europe. The report updates the evidence base for some important areas of alcohol policy, and provides policy-makers and other stakeholders in reducing the harm done to health and society by excessive drinking with useful information to guide future action.

Alcohol is one of the world's top three priority areas in public health. Even though only half the global population drinks alcohol, it is the world's third leading cause of ill health and premature death, after low birth weight and unsafe sex. In Europe, alcohol is the third leading risk factor for disease and death after tobacco and high blood pressure.

http://apps.who.int/bookorders/anglais/detart1.jsp?codlan=1&codcol=34&codcch=118

❖ **CANADIAN RESOURCES, BEST START**

Resources on Alcohol in Pregnancy from Best Start, Canada



Brochure: Aboriginal Pregnancy and Alcohol brochure
This is a brochure for use with an Aboriginal population on alcohol and pregnancy: the effects of drinking, traditional teachings about pregnancy and where to get help in Ontario.

Bilingual Recipe Cards: Mocktails for Mom
This is a set of bilingual recipe cards for delicious non-alcoholic drinks, also called Mocktails.

Be Safe: Have an Alcohol Free Pregnancy Tear-off Pads
Pad of tear-off colour sheets (10 cm by 10 cm) with information for expectant parents about alcohol use in pregnancy. One side says "Be Safe: Have an alcohol-free pregnancy" and includes visuals that illustrate the dangers to the baby from alcohol use in pregnancy. The other side includes phone contact information for Motherisk, Telehealth etc.

See full list of resources on http://beststart.org/resources/alc_reduction/index.html

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Fetal Alcohol Spectrum Disorder in Israel: Increased Prevalence in an At-Risk Population

Ariel Tenenbaum MD*, Pnina Hertz PhD, Talia Dor MD, Yael Castiel RN, Alon Sapir MD and Isaiah D. Wexler MD PhD*

Medical Unit for Adoption and Foster Care, Department of Pediatrics, Hadassah University Medical Center, Mount Scopus Campus, Jerusalem, Israel

ABSTRACT: **Background:** Maternal exposure to alcohol during pregnancy can lead to a wide range of clinical manifestations in their offspring, termed fetal alcohol spectrum disorder (FASD). In Israel, relatively few cases of FASD have been diagnosed and the prevalence has not been systematically evaluated.

Objectives: To determine the number of children with FASD or at risk for FASD in a select population of high risk patients seen at a clinic evaluating foster and adopted children.

Methods: Israeli children under 2 years old who were candidates for domestic adoption or in foster care were prospectively evaluated for clinical manifestations of FASD, and information was obtained regarding parental use of alcohol or other illicit drugs.

Results: Of the 100 patients prospectively evaluated, 8 had mothers with a known history of alcohol consumption during pregnancy. Two of the children had fetal alcohol syndrome (FAS) without known maternal exposure to alcohol and two had partial FAS. Eleven other children were at risk for development of one of the diagnostic categories of FASD.

Conclusions: In a population of pre-adoption and foster children, 15% either had manifestations of FASD or were at risk for developing FASD. Although this is a select high risk population, the data from this study strongly suggest a greater prevalence of FASD than previously assumed. Under-diagnosis of FASD is detrimental to affected children who could benefit from interventions designed to meet the needs of FASD victims.

IMAJ 2011; 13: 725–729

KEY WORDS: adoption, alcohol, birth defects, fetal alcohol syndrome (FAS), fetal alcohol spectrum disorder (FASD)

Fetal alcohol spectrum disorder is the leading cause of acquired developmental delay in the world [1]. FASD is caused by ethanol consumption throughout pregnancy, especially during the first trimester. Since ethanol is a teratogen, fetal cellular exposure to this toxin causes birth defects associated with dysmorphic features (e.g., smooth philtrum, thin upper lip, small palpebral fissures, upturned nose, microcephaly), other birth anomalies, neurodevelopmental abnormalities, and neuroanatomic defects [1,2]. The effects of in utero exposure of the fetus to alcohol are long term and are associated with developmental disabilities, cognitive impairments, psychiatric disorders, and social maladjustment [2,3]. The diagnosis of FASD is important for the patient and family since early intervention, appropriate medical follow-up, and social support are crucial for improving outcome and quality of life of both the patients and their families [4].

FASD is highly prevalent in many countries for which statistics are available. It is estimated that the prevalence of FASD among younger schoolchildren may be as high as 2%–5% in the United States and some West European countries [5]. In an Italian study the rate of FASD reached 4.1% in children attending 25 primary schools in the Lazio region [6]. Based on epidemiologic studies, risk factors for having a child with FASD have been identified; these include mothers in their teenage years, single mothers, previous children with FASD, and adopted children from Eastern Europe [7].

The number of children with the diagnosis of FASD in Israel is extremely low and there are no registries or databases in Israel listing children suspected of having FASD. Senecky et al. [8] reported that during a 10 year period (1998–2007), only six patients with a diagnosis of FASD were listed in two of the four health insurance funds in Israel. This may reflect a true low incidence, a lack of awareness among health professionals regarding the diagnostic features of FASD, the absence of systematic data collection in Israel related to FASD, confusion regarding the updated diagnostic criteria of FASD as related to classical fetal alcohol syndrome, or unawareness of the importance of listing FASD as a diagnostic entity [8]. With regard to the possibility of FASD being rare in Israel, this would be quite unlikely given the increase in alcohol consumption in the country [9], a lifestyle among a large segment of the population that is similar to western countries where the prevalence of FASD is high [10,11], and lack of awareness on the part of expectant mothers and adolescent girls regarding the minimal amounts of alcohol that are considered toxic [12,13].

We investigated a group of children at high risk for FASD. The Medical Unit for Adoption and Foster Care at Hadassah

***Dr. Tenenbaum and Dr. Wexler contributed equally to the study**

FASD = fetal alcohol spectrum disorder

Medical Center evaluates Israeli infants and children referred by the Child Services Unit of the Ministry of Welfare and Social Services who are in foster care, are residing in institutions, are candidates for adoption or were recently adopted. We evaluated this group to determine the level of fetal exposure to alcohol and the frequency of children having clinical manifestations consistent with FASD.

PATIENTS AND METHODS

The first 100 children under the age of 2 years who were referred to the medical adoption unit of Hadassah Mount Scopus for comprehensive medical and developmental assessment were prospectively evaluated regarding epidemiological and clinical manifestations of FASD. Information collected during the evaluation was recorded in the patient's medical record and subsequently collated. The study was approved by the Hadassah University Medical Center Institutional Review Board.

All children were examined by a physician, a psychologist and a nurse. The infants' medical and developmental condition and their anthropometric measurements were recorded. The medical history of the child and the biological parents was obtained. However, for many of the patients, complete information was not available either because the child had been abandoned or the parent(s) were not forthcoming with the information. As part of the evaluation, children underwent testing for human immunodeficiency virus, hepatitis, syphilis and, when indicated, other congenital infections. For children with microcephaly or cranial malformations, we recommend a neurologic evaluation and a cranial ultrasound. In nearly all cases follow-up in a child developmental center is recommended.

The diagnostic criteria for FASD and clinical manifestations associated with FASD were based on the U.S. Institute of Medicine's diagnostic classification with modifications based on a Canadian Task Force [14,15]. In brief, the diagnostic categories included:

- Fetal alcohol syndrome with known maternal alcohol exposure and with the child displaying typical dysmorphology associated with FAS (i.e., flat upper lip, flattened philtrum, midline facial hypoplasia), evidence of growth retardation, and central nervous system neurodevelopmental or neuroanatomic abnormalities
- FAS without confirmed maternal alcohol consumption with all the criteria mentioned above
- Partial FAS with confirmed maternal alcohol consumption
- Alcohol-related birth defects
- Alcohol-related neurodevelopmental disorders showing evidence of both neurodevelopmental and social/behavioral abnormalities.

FAS = fetal alcohol syndrome

RESULTS

Demographic data for children and patients are shown in Table 1. For 8% of the mothers there was a known history of alcohol consumption. Most likely this is an underestimation since information regarding alcohol exposure during pregnancy was not available for many of the mothers. This is even more true for the fathers as their identity was often unknown. As seen in Table 1, many mothers are involved in high risk behaviors such as drug use and promiscuity (e.g., prostitution, multiple partners). The median age for the children in our study population was 4.5 months and the mean age 5.9 ± 5.2 months at the initial visit. Almost all the children were evaluated prior to their first birthday.

The clinical characteristics of the children are listed in Table 2. Of the children with documented exposure to alco-

Table 1. Demographic characteristics of patients and biological parents

Patient's age (mos)	
Mean (SD)	5.9 ± 5.2
Median	4.5
Range	.5–24
Gender	
Males	42
Females	58
Religion of parents	
Jewish	59
Moslem	16
Christian	9
Mixed	13
Unknown	3
Maternal history	
Alcohol use	5
Drug use	14
Both	3
Promiscuity	3
Paternal history	
Alcohol use	0
Drug use	6
Both	2

Table 2. Clinical characteristics of patients

	Confirmed maternal alcohol consumption (n=8)	Alcohol consumption denied or unknown (n=92)
Facial dysmorphology	1	6
FAS facial characteristics	1	3
Prematurity	2	10
Low birth weight	1	11
Failure to thrive	2	13
Microcephaly	1	3
Developmental delay	0	15
Other neurological findings*	0	5

*Irritability, abnormal tone, appetite dysregulation, hearing loss, craniofacial abnormalities

hol, only two had manifestations that could be associated with partial FAS (a diagnostic category of FASD), and none had the facial dysmorphology associated with in utero alcohol exposure. One child was premature and had a low birth weight adjusted for gestational age, while the second child had microcephaly and failure to thrive.

For children with no documented in utero exposure to alcohol, one child had all the manifestations of FAS including a flat upper lip, microcephaly, low birth weight and failure to thrive. A second child, who was examined at age 12 months, also had all the features of FAS including microcephaly, failure to thrive, developmental delay, and midline facial hypoplasia. As shown in Table 2, many children without alcohol exposure had clinical characteristics associated with FASD including characteristic facial features, low birth weight or failure to thrive, and neurological deficits including developmental delay and microcephaly. Nineteen children had more than one problem. Over two-thirds of the mothers (10/14) with known drug usage but without alcohol exposure had children with one or more of the following: dysmorphism, low birth weight, failure to thrive, developmental delay, microcephaly, and/or other neurological abnormalities. This is in contrast to maternal alcohol consumption with or without drug usage where most of the children were asymptomatic.

Table 3 is a summary of our findings regarding the diagnosis of FASD. As part of our analysis, we characterized children at risk for attaining a diagnosis of FASD and this included the six children with known maternal ethanol exposure but no clinical manifestations (at risk for ARND), and five children with two of the three criteria for FAS but without confirmed maternal ethanol exposure. Of the latter five, one patient was a 5 month old child with FAS dysmorphology and developmental delay without growth failure, and the other four were children with developmental delay/microcephaly and growth failure, but without recognizable facial features of FAS.

ARND = alcohol-related neurodevelopmental disorders

Table 3. Summary of findings

FAS /+ history of alcohol exposure	0
FAS /- history of alcohol exposure	2
Partial FAS	2
ARBD	0
ARND	0
Potential to develop FASD*	5
Alcohol exposure/no signs of FASD	6

* No history of ethanol exposure but two of three clinical characteristics (FAS dysmorphology, growth failure, neurologic abnormalities)

ARBD = alcohol-related birth defects, ARND = alcohol-related neurodevelopment disorders

DISCUSSION

Among the children evaluated in a national medical adoption unit, 4% of the children met the criteria for a diagnosis of FASD, and another 11% were highly likely to receive a diagnosis of FASD either because of known alcohol exposure so that any neurologic, psychological or social adjustment abnormality discovered subsequently would place them in the category of ARND, or because they had two of three characteristics of FAS and would then meet the definition for FAS without confirmed maternal alcohol exposure.

It is likely that we are underestimating the true incidence of FASD and the number of children at risk in our population. Having a history of maternal alcohol exposure significantly impacts on the diagnosis of FASD. Without a confirmed history of maternal alcohol exposure, it is impossible to diagnose partial FAS, ARBD, or ARND so that the only diagnostic entity obtainable is FAS, which requires typical facial dysmorphology, growth failure and neurological manifestations. In contrast, in the presence of a history of maternal alcohol consumption, a child can be classified as FASD even with one of these abnormalities (partial FAS) or can be classified as ARBD or ARND if he or she has birth defects related to ethanol or neurological or social dysfunction.

In our patient population we often did not have information pertaining to the mother. Even when there was information, we could not be sure of its veracity as the information was often obtained by Child Protective Services personnel, and the parent may not have disclosed that she had consumed alcohol so as not to prejudice the legal and social status of her infant. In fact, one of the children in our study is listed as at risk for FAS without a history of maternal alcohol consumption (no information was available). Subsequently, when we examined a younger sibling, it became clear that the mother was an alcoholic who drank during her pregnancies. Based on the new information, this child should be reclassified as having FAS, thereby increasing the proportion of children with FASD to 5%.

Another reason for the underestimation of FASD in our population is that most of the children seen in our clinic were under the age of 1 year. Since many children were subsequently adopted, they were not followed in our clinic. This placed limits on our ability to diagnose FASD as many of the syndromatic and cognitive features of FASD do not become apparent until after 1 year of life. Reviewing studies performed in other countries, most children with FASD were diagnosed later in life, often after years of follow-up [5-7].

The high rate of both FASD and the risk for developing FASD in our selected population is not surprising when compared with studies conducted in other countries. FASD features were found in more than half the Russian orphans residing in

ARBD = alcohol-related birth defects

baby homes in Murmansk, Russia [16]. Similar findings were found for adopted children from Eastern Europe who were followed for a long time. Previous studies have also shown that children with FASD are over-represented in foster care and adoption [17]. For example, in a study done in Washington State, 50% of the surveyed children with FASD or FAS had at least one adoptive parent and 15.4% had foster parents [18].

A limitation of the current study is its generalizability to the general Israeli population. Clearly, the patients in our group were high risk and the incidence of FASD would be expected to be much lower among the general population of newborns. However, it would be reasonable to assume that there are mothers who ingested alcohol and did not give their children up for adoption or have them removed from the home. There are also mothers who would be considered normative and may have consumed alcohol early in the pregnancy prior to becoming aware of their pregnancy. This is quite common in other countries, and in the USA at least 50% of pregnant women drank alcohol during the 3 months prior to pregnancy recognition, and 1 in 20 of these women drank at moderate to heavy rates [19].

Our study is also in line with other recent reports suggesting an increase in alcohol abuse among Israelis, especially teenagers and young adults. This is reflected in statistics regarding the increasing abuse of alcohol in the general population [9], alcohol levels in fatal casualties in motor vehicle accidents [20], and the number of children brought to the emergency room with alcohol poisoning [21].

This raises the question why the reported incidence of FASD is so low in Israel given the findings in our study. Taking a conservative estimate that the incidence or risk of FASD in the general population is 100 to 1000 times lower than the 15% seen in our study, it would be expected that between 22.5 and 225 children born per year would be at risk for FASD (annual birth rate in Israel is approximately 150,000). This is far higher than the number of children carrying a diagnosis of FASD listed in hospitals or health insurance funds [8]. It is also far below the rates reported for other countries (USA, South Africa, Italy, Sweden) where the incidence of FASD in the general population ranges from 5% to 7% [5].

One possibility is that the rate of FASD in Israel is extremely low except in very high risk populations such as those seen in our clinic. This could be due to low rates of alcohol consumption and low rates of alcohol abuse among the different constituents of the Israeli population. While it is true that there are higher rates of abstinence in the population as compared to European countries, a large-scale survey sponsored by the Ministry of Health showed that the lifetime prevalence rate of alcohol abuse or dependence is 4.3% and that 5% of the population drank alcoholic beverages three or more times a week, which is comparable to European countries [10]. Specific segments of the population are at greater risk, including young adults, males, and immigrants from the former Soviet Union. Other studies have also shown that ethanol abuse is a significant problem among immigrants from the former Soviet Union and Ethiopia [22,23], and that the frequency of intoxication and binge drinking was increasing [9]. Hence, a low prevalence of alcohol intake or dependence is not a reason for the lower reported rates.

It is also possible that despite the high prevalence of drinking in the general population, pregnant women are very careful to avoid drinking during pregnancy. Weiss and colleagues [24] found that among 2477 woman who had given birth at a single medical center during the years 1999–2000, only 1.13% admitted to consuming alcohol during pregnancy – mostly small amounts and infrequently. However, the authors were very skeptical of their findings as they did not concur with reported prevalence rates in other countries; they suggested that there was significant under-reporting because of the fear of stigmatization, denial, and/or the reluctance to share personal information. This was borne out by a more recent survey done in 2010 sponsored by the Israel Anti-Drug Authority, which found that among 3815 postpartum women in three Israeli hospitals 17.1% reported that they consumed alcohol during pregnancy and 0.8% admitted to binge drinking at least once during the last 3 months of their pregnancy [13].

A third possibility is that there are protective genes for FASD that are either associated with diminished alcohol consumption or reduced teratogenic effects of alcohol on the fetus. The alcohol dehydrogenase 1B genotype is related to the risk for alcoholism, and there is a greater prevalence (20%) of the protective allele among Jews. While it is true that younger individuals (< 33 years old) carrying the protective allele had lower alcohol consumption (mean number of drinks per occasion 2.6), in those without the protective allele the mean number of drinks was 6.2, which is considered risky and unsafe drinking and within the ranges known to be associated with FASD [25]. There are also genes that are protective of the fetus, but there is no increased prevalence of this gene among Jews.

The most likely reason for under-reporting is the lack of awareness of health care providers or their lack of effort to either solicit a history of maternal alcohol consumption or examine children for features of FASD. In their study [8], Senecky and co-authors interviewed geneticists and child development specialists throughout Israel. Fifty percent of the respondents felt that "tens" or even "hundreds" of children with a potential diagnosis of FASD had been missed. Among the respondents, approximately 60% reported that there was low or insufficient awareness of FASD among physicians in Israel.

The current study suggests that the number of children with FASD being diagnosed is only the tip of the iceberg. This is unfortunate as early intervention may minimize many of the cognitive, behavioral and social problems associated with FASD [4]. Identification of mothers with a history of ethanol

consumption and the appropriate follow-up by the social services may prevent future cases in the same family. Attributing antisocial behavior to FASD may facilitate more appropriate interventions and lower the rate of recidivism [3].

CONCLUSIONS

This study shows that there is a high rate of FASD and a risk for developing FASD in a selected population of adopted or foster children in Israel. The study is limited in that it observed the patients for a short time and at a very early stage of development. While direct extrapolation to the general population is not possible, this study can confirm previous studies in Israel suggesting that FASD is under-diagnosed. Since intervention is important and potentially beneficial, it is crucial to identify children with FASD or at risk for developing FASD. Steps to improve the diagnosis of FASD in Israel would include large-scale studies of the pediatric population to determine the true incidence of maternal alcohol consumption and FASD as well as interventions to enhance the awareness of health care personnel regarding the need to assess pregnant women for ethanol exposure and clinical manifestations of FASD among children and adults.

Acknowledgments

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References

1. Sokol RJ, Delaney-Black V, Nordstrom B. Fetal alcohol spectrum disorder. *JAMA* 2003; 290: 2996-9.
2. Guerri C, Bazinet A, Riley EP. Foetal alcohol spectrum disorders and alterations in brain and behaviour. *Alcohol Alcohol* 2009; 44: 108-14.
3. Fast DK, Conry J. Fetal alcohol spectrum disorders and the criminal justice system. *Dev Disabil Res Rev* 2009; 15: 250-7.
4. Jirikowic T, Gelo J, Astley S. Children and youth with fetal alcohol spectrum disorders: summary of intervention recommendations after clinical diagnosis. *Intellect Dev Disabil* 2010; 48: 330-44.
5. May PA, Gossage JP, Kalberg WO, et al. Prevalence and epidemiologic characteristics of FASD from various research methods with an emphasis on recent in-school studies. *Dev Disabil Res Rev* 2009; 15: 176-92.
6. May PA, Fiorentino D, Gossage JP, et al. Epidemiology of FASD in a province in Italy: prevalence and characteristics of children in a random sample of schools. *Alcohol Clin Exp Res* 2006; 30: 1562-75.
7. May PA, Gossage JP, Marais AS, et al. Maternal risk factors for fetal alcohol syndrome and partial fetal alcohol syndrome in South Africa: a third study. *Alcohol Clin Exp Res* 2008; 32: 738-53.
8. Senecky Y, Inbar D, Diamond G, et al. Fetal alcohol spectrum disorder in Israel. *IMAJ Isr Med Assoc J* 2009; 11: 619-22.
9. Bar-Hamburger R, Ezraci Y, Steinberg D, et al. Israel illegal use of drugs and alcohol in the sixth national epidemiological survey, 2005. National Anti-Drug and Anti-Alcohol Authority of Israel.
10. Isralowitz RE, Reznik A. Former Soviet Union immigrant and native-born adolescents in Israel: substance use and related problem behavior. *J Ethn Subst Abuse* 2007; 6: 131-8.
11. Neumark YD, Lopez-Quintero C, Grinshpoon A, et al. Alcohol drinking patterns and prevalence of alcohol-abuse and dependence in the Israel National Health Survey. *Isr J Psychiatry Relat Sci* 2007; 44: 126-35.
12. Weiss S. Israeli Arab and Jewish youth knowledge and opinion about alcohol warning labels: pre-intervention data. *Alcohol Alcohol* 1997; 32: 251-7.
13. Senecky Y, Weiss N, Shalev SA, et al. Alcohol consumption during pregnancy among women in Israel. *J Popul Ther Clin Pharmacol* 2011; 18: e261-72.
14. Stratton K, Howe C, Battaglia F. Fetal Alcohol Syndrome: Diagnosis, Epidemiology, Prevention and Treatment. Washington DC: National Academy Press, 1996.
15. Chudley AE, Conry J, Cook JL, et al. Public Health Agency of Canada's National Advisory Committee on Fetal Alcohol Spectrum Disorder. Fetal alcohol spectrum disorder: Canadian guidelines for diagnosis.*CMAJ* 2005; 172 (5 Suppl): S1-21.
16. Miller LC, Chan W, Litvinova A, et al. Fetal alcohol spectrum disorders in children residing in Russian orphanages: a phenotypic survey. *Alcohol Clin Exp Res* 2006; 30: 531-8.
17. Landgren M, Svensson L, Strömland K, et al. Prenatal alcohol exposure and neurodevelopmental disorders in children adopted from Eastern Europe. *Pediatrics* 2010; 125 (5): e1178-85.
18. Olson HC, Oti R, Gelo J, et al. "Family matters:" fetal alcohol spectrum disorders and the family. *Dev Disabil Res Rev* 2009; 15: 235-49.
19. Floyd RL, Decouflé P, Hungerford DW. Alcohol use prior to pregnancy recognition. *Am J Prev Med* 1999; 17: 101-7.
20. Jaffe DH, Savitsky B, Zaistev K, Hiss J, Peleg K. Alcohol and driver fatalities in Israel: an examination of the current problem. *IMAJ Isr Med Assoc J* 2009; 11: 725-9.
21. Kozer E, Bar-Hamburger R, Rosenfeld NY, Zdanovitch I, Bulkowstein M, Berkovitch M. Illicit drug and alcohol users admitted to the pediatric emergency department. *IMAJ Isr Med Assoc J* 2008; 10: 779-82.
22. Weiss S. Alcohol use and problems among immigrants from the former Soviet Union in Israel. *Subst Abus* 2008; 29: 5-17.
23. Weiss S. Alcohol use and abuse among Ethiopian immigrants in Israel: A review. *African J Drug Alcohol Stud* 2008; 7: 27-39.
24. Weiss S, Sharan H, Merlob P, Self-reported alcohol use among pregnant women in the center of Israel. *Int J Risk Safety Med* 2000; 13: 225-32.
25. Spivak B, Frisch A, Maman Z, et al. Effect of ADH1B genotype on alcohol consumption in young Israeli Jews. *Alcohol Clin Exp Res* 2007; 31: 1297-301.

"Neither a man nor a crowd nor a nation can be trusted to act humanely or to think sanely under the influence of a great fear"

Bertrand Russell (1872-1970), British philosopher, mathematician, author, and Nobel Prize laureate

"The memories of a man in his old age are the deeds of a man in his prime"

Pink Floyd, English rock band that achieved worldwide success with their progressive and psychedelic rock music. Their work is marked by the use of philosophical lyrics, sonic experimentation, innovative album art, and elaborate live shows

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Fetal alcohol spectrum disorders: is it something we should be more aware of?

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KEYWORDS Fetal alcohol syndrome, fetal alcohol spectrum disorders, overview, diagnosis, risk, clinical presentation

DECLARATION OF INTERESTS The FASD clinic has received honoraria with respect to Dr Mukherjee's lectures at conferences and educational meetings related to FASD from various charities and an industrial sponsor including TACT, PFC, NOFAS UK, Jannsen Cilag, Flynn Pharma and Oak Group. He is medical advisor to FASAware UK, FASD Trust UK and NOFAS UK.

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INTRODUCTION

Fetal alcohol spectrum disorders (FASD) describes a range of conditions caused by exposure to alcohol by a developing fetus.[1] The range of disorders vary from the most recognised fetal alcohol syndrome (FAS), where physical signs are obvious, to problems with behaviour and the brain with no obvious external physical signs (alcohol related neurodevelopmental disorders [ARND]).[2,3] This selective review will give an overview of many of the clinically relevant areas of FASD, including diagnostic difficulties, epidemiology, risk, knowledge among the general public, cognitive profiles and secondary disabilities.

BACKGROUND

The damaging effects to the fetus of maternal alcohol consumption during pregnancy have been recognised and commented on since as long ago as the Ancient Greek philosophers.[4] There are references in the Bible relating to the ill effects of alcohol in pregnant women: 'You will conceive and give birth to a son. Now then drink no wine or fermented spirit…'.[5] While it could be argued that these quotes are taken out of context as the actual pathology of the damage was not known in the past, they do represent a long historical recognition that the effects of alcohol on a developing fetus can be harmful.

Calhoun and Warren[4] describe the modern history of FAS, beginning with a case series published by Paul Lemoine in 1967.[6] A French paediatrician and his midwifery colleagues reported on 127 cases of children who had all been exposed to alcohol prenatally, and their later difficulties. The study did not, however, set out the diagnostic features required to identify the condition. The article that led to a change in the recognition and definition of the condition was published in 1973 in *The Lancet* by Smith and Jones. They reported on children living on an American Indian reserve who all presented with consistent, common and recognisable features, which they called fetal alcohol syndrome.[7] All of the children's mothers had consumed high levels of alcohol during pregnancy. Smith and Jones set out a series of criteria which formed the basis for the development of diagnostic criteria for FASD.

The publication of Smith and Jones' articles and an increasing recognition of the condition generated a great deal of interest in FAS. Case studies considering the presentation of behavioural difficulties, neurological damage and physical characteristics of the disorder were published.[8] Further, it was becoming clearer that alcohol could affect the brain without resulting in the distortions of facial features attributed to FAS. This led to a debate as to whether the term fetal alcohol effect (FAE) was a more accurate representation of the condition.[9] Insights were obtained from both human examination and animal studies. It was through animal studies that perhaps our greatest understanding about the effects of alcohol during pregnancy were achieved, both from a pathological point of view, but also in clarifying the teratogenicity of alcohol. By using multiple animal models, allowing controlled experiments not possible in humans for ethical reasons, it became possible to begin to identify pathological processes, confirm cognitive profiles and explore possible therapeutic interventions.[10]

The Institute of Medicine (IOM) defined a set of criteria in 1996 for classifying the disorder. It was also recognised that prenatal exposure to alcohol could present with a spectrum of difficulties that would vary among individuals.[4] The term FASD came into wider use towards the end of the 1990s, superseding the term FAE.

DIAGNOSTIC ISSUES IN FAS AND FASD

The diagnostic elements of FAS are made up of three facial features (short palpebral fissures, elongated and flattened philtrum and a thin upper lip vermillion), pre- and postnatal growth deficits (below the tenth percentile), neurocognitive deficits and a history of maternal alcohol consumption during pregnancy. It is only in the case of FAS that confirmation of alcohol consumption is not required for the diagnosis.[1,2] Timing is also important for diagnosis. When a child is still very young, features such as brain damage or developmental delay may not be obvious, while even at birth there may be signs of the distinct facial features, growth problems and evidence of maternal alcohol exposure. However, without the central nervous system (CNS) deficits, which are not always obvious at birth, a diagnosis of FAS is not valid. Despite this, a possible diagnosis, in anticipation of potential cognitive findings as a child grows older, should be noted and followed up.

While there is general agreement over the diagnostic features of FAS, debate continues over specific criteria. The cut-off figure used for palpebral fissure length for example is different across several diagnostic frameworks.[2,11] Table 1 summarises the similarities and differences between the four main diagnostic frameworks in current practice.

The main difference between the groups remains whether or not the cut-off for palpebral fissure length should be the tenth or the third percentile. In the UK, ninth and second percentiles are now more commonly used. The difficulty in reaching agreement relates to the sensitivity and specificity of this cut-off measurement. A sensitivity test will pick up more of the true positive cases, while a specificity test will rule out more of the incorrect cases. The best test is one that has both high sensitivity and high specificity. Unfortunately, in most cases one tends to be higher than the other, leading to the discrepancies between the different diagnostic groupings and uncertainty about the reliability, accuracy and usefulness of the tests. Astley for example compared two diagnostic groupings, finding discrepancies between the methods used and the subsequent results obtained.[11] These discrepancies highlight the inherent need for local norms for diagnosis to be agreed and established. The Canadian Guidance, developed by the Canadian Advisory group on FASD, is increasingly used as the best overview of all current guidelines.[12]

OVERLAPPING SYMPTOMS

One of the other difficulties in diagnosing FAS and FASD is because many of the established symptoms of FAS and FASD tend to be characteristic rather than discriminating. A characteristic symptom is one that is commonly seen in the disorder, however it may also be found as the result of other disorders. Discriminating symptoms occur uniquely as the result of a specific disorder. As FASD cases present mainly, if not wholly, with characteristic symptoms, it is only by exclusion of other disorders through genetic testing and through confirmation of alcohol consumption, that a reliable diagnosis can be established. Unfortunately, the amount and quality of information necessary to conduct such a careful differential diagnosis is frequently missing. In his editorial, Goddlett noted the establishment of a collaborative initiative who are attempting to overcome many of these issues and to bring consistency in diagnosis and management to the field of FASD.[13]

There has been much speculation and debate about the relationship between FASD and primarily attention deficit hyperactivity disorder (ADHD), but also its links with autism. Various types of relationships have been suggested, from direct causality to separate but overlapping disorders[14,15] but as yet agreement about them

TABLE 1 Summary of the four main diagnostic frameworks and associated criteria[1]

	Centre For Disease Control	Institute of Medicine (revised)	Canadian National Advisory Committee on FASD	4-Digit Diagnostic Code
Face	Tenth percentile PFL and rank 4/5 on lip philtrum	Tenth percentile PFL and rank 4/5 on lip philtrum	Third percentile PFL and rank 4/5 on lip philtrum	Third percentile PFL and rank 4/5 on lip philtrum
Growth	Pre/postnatal growth below tenth percentile	Pre/postnatal growth below tenth percentile	Pre/postnatal growth below tenth percentile	Pre/postnatal growth below tenth percentile
Neurological	One out of several brain parameters including OFC <10%, CNS deficits	One out of one brain parameters including OFC <10%, CNS deficits Or abnormal structure	3+ soft and hard neurological signs	One out of several brain parameters including OFC <3%, CNS deficits
Alcohol	Confirmed or unknown	Confirmed to be excessive or unknown	Confirmed or unknown	Confirmed or unknown

PFL= palpebral fissure length; **OFC=** orbitofrontal cortex; **CNS=** central nervous system

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remains elusive. We recently reported higher rates of autism were being found in people with FASD diagnoses.[16] We believe the association between the two appears to be more than what might be attributed to chance. Therefore we believe, as others have for similar conditions,[17] that more research is needed in order to establish the relationship between the phenomenology and the underlying neurological pathology.[16]

EPIDEMIOLOGY

The original IOM criteria were used in various epidemiological studies of FAS and FASD but they were refined in 2006,[2] leading to wide variations in the reported prevalence rates from those originally stated.[18,19] Other problems were found with other studies. An investigation by May et al. for example in Lazio, Italy, showed that the prevalence rates of the full spectrum of FASD was estimated at 35 people per 1,000.[20] Unfortunately, despite actively recruiting study participants (proactively identifying cases rather than passively waiting for them to be reported) in order to establish prevalence, the study group moved people between diagnostic groupings based on clinical review rather than strict adherence to the study design. This led to criticism of the study, most specifically in regards to the validity of the results.[20]

A later study by Petkovic and Barisic, using similar methodology as the May study, but without the same limitations and criticisms, showed similar findings in a population of school-aged children.[21] They actively recruited participants in two regions of Croatia (over 1,200 people). This study showed an overall prevalence rate of FAS and partial FAS of 40.77/1,000.[21] However these studies have yet to be widely replicated by other groups. The suggestion from studies thus far is that the prevalence rate in the full population is higher than previously thought. Table 2 summarises studies that used the same methodology, however the actual rate in many parts of the world is still unclear. The results however continue to represent the best available estimates of prevalence.

In the UK there have been no actively recruited studies. This is unfortunate, as passive surveillance studies have been shown to have poor reliability due to the under-reporting of the rates of prevalence. A recently published study completed a review of paediatric hospital episode statistics in the UK over a five-year period which showed a progressive increase in the rates, but ultimately they were much lower than expected.[22] The true prevalence in the UK therefore still needs to be established.

ALCOHOL CONSUMPTION AND THE LEVEL OF RISK

Establishing the actual level of alcohol consumed during pregnancy in order to accurately diagnose the disorder is challenging but necessary. Despite attempts to improve awareness of the need for this information, problems persist partly because alcohol exposure is not often recorded in the medical records of children or mothers.[22] It has also been reported that the recall of alcohol consumption amounts is poor.[23] Different ways of improving the recording of what women drink during pregnancy continue to be researched. Existing methods range from the use of specialist screening tools such as the T-ACE questionnaire or the Alcohol Disorders Identification Test (Audit-C) to the use of specific biomarkers.[1,14] The screening tools raise concerns because they rely on honest reporting. To circumvent these concerns, a search for biomarkers such as free fatty ethyl esters in meconium can be done.[1,24,25] Unfortunately, these biomarkers only pick up second and third trimester alcohol consumption. There are also ethical considerations around the use of biomarkers in the absence of informed consent, particularly since the level of risk has not been established.[26] The implications of this on the wider 'drinking during pregnancy' debate must also be considered.

Another challenge to collecting data on individual alcohol consumption during pregnancy is a lack of a clear definition of what is considered to be a 'safe' amount of alcohol.[27,28] Studies have shown dose-response relationships between levels of alcohol consumption and harm: higher levels of alcohol exposure were found to cause the most obvious neurodevelopmental problems;[27,29–31] lower levels of alcohol exposure are more difficult to assess, with some studies reporting no clear differences compared to a non-exposed group,[29,30,32–38] while others continue to suggest possible effects of even low exposure levels.[31,35,39–41] As such, the levels of individual risk remain uncertain.

TABLE 2 Estimated prevalence rates per thousand population from different studies[18–21]

	Fetal Alcohol Syndrome	Partial Fetal Alcohol Syndrome	Fetal Alcohol Spectrum Disorders
May et al., South Africa (2007)[18,19]	51.3–67.2	16.8–22.0	68.0–89.2
May et al., Italy (2006)[20]	3.7–7.4	15.7–31.3	20.3–40.5
Petrovic et al., Croatia (2010)[21]	6.4	34.3	40.77

Perhaps unsurprisingly, given the difficulties stated above, the advice about drinking alcohol during pregnancy currently differs from country to country.[42] In the UK for example, women are advised to avoid alcohol consumption if possible and, if they do drink alcohol, to consume no more than one to two drinks in a single sitting. However, the definition of how much alcohol constitutes one to two drinks is often confusing and unclear. Combined with the public's limited understanding of the scientific conclusions about the risks of drinking alcohol during pregnancy, this creates a dangerous situation.[28]

AWARENESS AND UNDERSTANDING OF FASD IN THE GENERAL POPULATION

Internationally and more specifically in the UK, conflicting evidence regarding the effects of alcohol during pregnancy has seemingly led to inconsistent understanding of FASD in both professionals and the public.[43–50] This has implications for the management and the prevention of the disorder. The development of a consistent public health strategy remains a challenge, particularly when the condition is one that, unlike many others, can be entirely prevented simply by not consuming alcohol while pregnant. As such, it has been argued that FASD constitutes a public health disorder that requires directed prevention strategies.[24,51,52]

The lack of consistent guidelines and the subsequent confusion over health strategy means that providing support for individuals and families can be challenging. This increases the health burden and stress faced by those with FASD and their families.[53–58] The publicly funded health and social care system in the UK is accessible by all and therefore may well offer better support to those with FASD and their families compared to countries in which access is more limited, although budget limitations may well mean the opposite is also true. The situation is currently unclear and requires clarification.

EVIDENCE FROM ANIMAL STUDIES ON THE EFFECTS OF ALCOHOL

Part of the difficulty of finding evidence that supports the labelling of alcohol as a teratogen relates to the ethical and practical aspects of conducting this research. Ethically, it is not possible to conduct controlled human experiments into the risks of alcohol during pregnancy. Population-based studies are inherently biased by confounding factors. For example, alcohol consumption by the mother is known to cause prematurity,[59] yet prematurity itself is also known to cause cognitive deficits regardless of what may have caused it.[60] Further, much of the population-based work is subject to bias, especially in terms of recall of alcohol consumption.[61] To overcome this and statistically correct for these confounding factors, large numbers of research participants are required. Alternative methods for researching this subject have been suggested (such as the use of natural experiments) but have yet to be widely implemented.[62]

Instead, animal studies have formed the basis for providing the basic pathology of FASD. Hannigan summarised the research undertaken in the years prior to his review in 1996.[63] He looked at the data collected using animal research, including evidence about dose-response rates, timing of exposure and risk, neuro-anatomical and neuro-behavioural dysfunctions and offered an insight into potential future treatments. Despite the availability of this evidence, the report concluded by suggesting that much more work was still needed.

Sulik's study helped identify the dose-dependent effect of alcohol on the development of facial features and clarified how the timing of alcohol exposure affects subsequent fetal development by identifying a risk period during weeks three to four of gestation. She also considered factors that may alter alcohol teratogenesis between individuals.[64] Her research has implications for humans in that, in the UK at least, women are drinking more than in the past and the patterns of this alcohol consumption both in terms of who is consuming alcohol and where it is consumed have changed.[65] More women are consuming alcohol at levels that are associated with the levels of risk identified in the animal studies.[66] The timing of pregnancy recognition is therefore crucial in helping women to refrain from alcohol consumption during the earliest period in the pregnancy when facial features of the fetus are most susceptible to damage. Many women will consume alcohol until pregnancy is detected, usually between the first and second missed period. Once pregnancy is confirmed, however, around 67% of women in the UK will significantly reduce or stop drinking.[23] As a result, it seems likely that for some fetuses, damage to the facial features may occur, but later development may continue, unaffected by alcohol, leading to the subsequent unhindered development of the brain and body. This in itself has implications for relying solely on facial features for the diagnosis of FAS and confirms the importance of the cognitive features in the diagnosis. It is clear from the literature on animal studies that the risks of alcohol consumption during pregnancy exist during all trimesters, not just the first.[63,64] Ongoing brain development in the second and third trimesters means that public health advice regarding alcohol consumption must also cover these trimesters.

While many have extrapolated the results of the animal studies to the human population, some authors have questioned the methods of animal research and the applicability of the conclusions of the results to humans. Abel argues for example that in some of the animal experiments that refer to 'low blood alcohol concentrations', rather than representing low levels of alcohol

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in the bloodstream, this actually describes concentrations that would correspond to high levels of alcohol in a human. He points out that the methods of alcohol delivery, such as putting animals onto liquid alcohol diets, do not reflect human consumption patterns, making interpretation difficult. A six gram dose of alcohol in a rat for example equates to 27 drinks per day in an average human (where a US drink equated to 1.5 UK units).[32] In their paper, Leichter and Lee confirm that the method of alcohol delivery in animal studies represents a confounding factor in their interpretation.[67]

Despite these issues, animal studies remain the only way to ethically study the effects of alcohol on fetuses and confirm its teratogenicity. By showing consistent findings in multiple animal models it is possible to make comparisons to the human findings from population-based research. Cudd describes the strengths and weaknesses of different models of exploration, concluding that substantial progress in the field will require the judicious use of multiple scientific approaches using different animal systems.[68]

COGNITIVE PROFILE OF PEOPLE WITH FASD

Population-based studies and studies of affected individuals have corroborated many of the findings from the animal studies. Jacobson and Jacobson summarised some of the early findings (Table 3).[69] Rasmussen carried out a systematic review of executive function (the set of cognitive abilities that control and regulate other abilities and behaviours) and working memory[70] and confirmed these two areas of deficit found in experimental studies contributed to the difficulties in function seen in affected individuals. Rasmussen's review complimented much of the work of Matteson and colleagues, who looked at IQ and its relationship to facial features[71] as well as how executive functioning in this group was affected by alcohol.[72] They contributed much to the understanding of how the condition presents. Others, such as Burden and colleagues, have shown how attention and pressure of time to complete a task can affect those with FASD by studying working memory and processing speed.[73,74] Sampson and colleagues have further highlighted that pressure and time affect the accuracy of an individual's results on cognitive tests. Roebuck et al. looked at the importance of the corpus callosum damage caused by maternal alcohol consumption and the impact that this has on cognitive presentation and the ability of those affected to link information between cerebral hemispheres.[75] All of these findings have implications for the management of FASD, by establishing that performance can be improved by reducing external expectations and time pressures on those affected.[76]

Increased understanding of the condition and the correlation of psychometric findings with neuro-imaging studies has led to a more refined profile.[77] Using data from two sites of a multi-site FASD international project, Matteson et al. found that executive functioning and spatial processing were especially sensitive to prenatal alcohol exposure.[78] The remaining challenge is to identify how these cognitive profiles can help to integrate and establish management strategies on the basis of individual diagnostic profiles. Kodituwakku for example, has argued that in reality, a broader framework of understanding and subsequent intervention is needed in order to bring together the cognitive profiles to allow better management of this condition.[79]

TABLE 3 Summary of main cognitive deficits seen in people with fetal alcohol spectrum disorder

- Intellectual disabilities and reduced IQ
- Executive functioning difficulties
  - Hyperactivity
  - Inattention
  - Social communication problems
  - Integration of information problems
  - Concrete thinking
  - Difficulty with abstract concepts
  - Cognitive flexibility issues
- Working memory problems
- Processing speed deficits
- Spatial awareness problems
- Mathematical problems
- Cerebellar problems
- Hemispheric linkage problems

SECONDARY DISABILITIES

As a child grows into an adult, the secondary disabilities associated with FASD are increasingly recognised. Secondary disabilities are defined as those caused by inadequate recognition and support management of the primary disability. Streissguth et al. published a list of secondary disabilities from a long-term cohort study where participants were followed up for over 30 years.[80,81] These deficits are summarised in Table 4. Steinhaussen and Spohr have also conducted a long-term follow-up study showing the difficulties that continue into adulthood.[82] While support on an individual level has been shown to lead to a better overall outcome, a lack of that same support is known to lead to significant

TABLE 4 Common secondary disabilities seen if individuals are not recognised and supported

- Mental health issues
  - All form of mental illness
  - Increased suicide rates
- Crime and incarceration
- Sexual inappropriateness
- Substance abuse
- Educational difficulties

EDUCATION

difficulties. Mental health issues are the most frequently recognised of these disabilities: 90% of individuals affected by FASD suffer some form of mental illness. Famy first reported on the mental health issues in this group as far back as 1997 in a brief report to *The Journal of the American Medical Association*.[83] Unfortunately, as the report was based on a very small number of participants, the findings were not widely accepted. The data, however, were corroborated in a study by Barr et al in 2006. In a larger series they showed high rates of mental illness. The larger sample also allowed statistical correction for confounding factors influencing the outcomes.[84] In addition, it has been found that people with FASD who also suffer with mental illness have been associated with higher rates of suicide.[85]

Larger scale epidemiological studies have increasingly been used to look at the wider impact of prenatal alcohol exposure on both developmental and mental health presentation. Work conducted by Kelly et al. for example, showed that, in a sample of 11,513 people, low levels of alcohol exposure had less of an impact on socio-emotional or cognitive outcomes. They did however show a clear relationship between higher levels of alcohol exposure and cognitive deficits.[30] This was in contrast to findings by Sayal et al. who found that girls aged nine were more likely to suffer mental health problems even at maternal alcohol consumption levels of less than one UK unit a week (in a sample of 8,046 people).[31] At present these contrasting findings leave almost as many questions as answers. More research will be required to clarify these issues.

CONCLUSIONS

Recognising and managing FASD is a challenge in the UK. Discrepancies in the scientific literature continue to raise questions about the condition, but there is general agreement that FASD will demand significant preventive, medical and social care resources in the future. Overall, better training on FASD and its impact is needed for all professionals working in the fields of health, social work, criminal justice and education. If the situation in the UK is to improve, with a decrease in the number of people with FASD achieved, the cause and impact of the condition must be clarified and widely and consistently communicated to professionals and the public. While some changes have already begun to happen, there is a great deal left to do.

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REFERENCES

1 BMA Board of Science. *Fetal Alcohol Spectrum Disorders, a guide for healthcare practitioners* [Internet]. London: BMA Publishing; 2007 [cited 2012 March 28]. Available from: http://www.bma.org.uk/images/FetalAlcoholSpectrumDisorders_tcm41-158035.pdf
2 Hoyme HE, May PA, Kalberg WO et al. A practical clinical approach to diagnosis of Fetal Alcohol Spectrum Disorders: clarification of the 1996 Institute of Medicine criteria. *Pediatrics* 2005; 115:39–47. http://dx.doi.org/10.1542/peds.2005-0702
3 Mukherjee RA, Hollins S, Turk J. Fetal Alcohol Spectrum Disorder: an overview. *J R Soc Med* 2006; 99:298–302. http://dx.doi.org/10.1258/jrsm.99.6.298
4 Calhoun F, Warren KR. Fetal Alcohol Syndrome: historical perspectives. *Neurosci Biobehav Rev* 2007; 31:168–71. http://dx.doi.org/10.1016/j.neubiorev.2006.06.023
5 The Bible (New international version). *Judges 13:7*. Michigan: Biblica; 2011.
6 Lemoine P, Harousseau H, Borteryu JP et al. [The children of alcoholic parents: anomalies observed in 127 cases]. *Quest Medicale* 1968; 25:476–82.
7 Jones KL, Smith DW, Ulleland CN et al. Pattern of malformation in offspring of chronic alcoholic mothers. *Lancet* 1973; 1:1267–71. http://dx.doi.org/10.1016/S0140-6736(73)91291-9
8 Warren KR, Foudin LL. Alcohol-related birth defects: the past, present, and future. *Alcohol Res Health* 2001; 25:153–8.
9 Clarren SK, Smith DW. The fetal alcohol syndrome. *N Engl J Med* 1978;298:1063–7.http://dx.doi.org/10.1056/NEJM197805112981906
10 Chandrasena AN, Mukherjee RA, Turk J. Fetal Alcohol Spectrum Disorders: an overview of interventions for affected individuals. *Child and Adolescent Mental Health* 2009; 14:162–7. http://dx.doi.org/10.1111/j.1475-3588.2008.00504.x
11 Astley SJ. Comparison of the 4-Digit code and the Hoyme diagnostic guidelines for Fetal Alcohol Spectrum Disorders. *Paediatrics* 2006; 118:1532–45. http://dx.doi.org/10.1542/peds.2006-0577
12 Chudley AE, Conry J, Cook JL et al. Fetal Alcohol Spectrum Disorder: Canadian guidelines for diagnosis. *CMAJ* 2005:172:S1–S21. http://dx.doi.org/10.1503/cmaj.1040302
13 Goodlett CR. Fetal Alcohol Spectrum Disorders: new perspectives on diagnosis and intervention. *Alcohol* 2010; 44:579–82. http://dx.doi.org/10.1016/j.alcohol.2010.10.001
14 Gray R, Mukherjee RA. A psychiatrists guide to Fetal Alcohol Spectrum Disorders in mothers who drank heavily during pregnancy. *Advances in Mental Health and Intellectual Disabilities* 2007; 1:19–26.
15 Oesterheld JR, Wilson A. ADHD and FAS. *J Am Acad Child Adolesc Psychiatry* 1997; 36:1163. http://dx.doi.org/10.1097/00004583-199709000-00004
16 Mukherjee RA, Layton M, Yacoub E et al. Autism and autistic traits in people exposed to heavy prenatal alcohol: data from a clinical series of 21 individuals and a nested case control study. *Advances in Mental Health and Intellectual Disabilities* 2011; 5:42–9. http://dx.doi.org/10.5042/amhid.2011.0015
17 McGough JJ, McCracken JT. Adult attention hyperactivity disorder: moving beyond DSM-IV. *Am J Psychiatry* 2006; 163:1673–5. http://dx.doi.org/10.1176/appi.ajp.163.10.1673
18 May PA, Gossage JP, Kalberg WO et al. Prevalence and epidemiological characteristics of FASD from various research methods with an emphasis on recent in-school studies. *Devl Disabil Res Rev* 2009; 15:176–92. http://dx.doi.org/10.1002/ddrr.68
19 May PA, Gossage JP, Marais AS et al. The epidemiology of Fetal Alcohol Syndrome and Partial FAS in a South African community. *Drug Alcohol Depend* 2007; 88:259–71. http://dx.doi.org/10.1016/j.drugalcdep.2006.11.007
20 May PA, Fiorentino D, Phillip Gossage J et al. Epidemiology of FASD in a province in Italy: prevalence and characteristics of children in a random sample of schools. *Alcohol Clin Exp Res* 2006; 30:1562–75. http://dx.doi.org/10.1111/j.1530-0277.2006.00188.x

21 Petkovic G, Barisic I. FAS prevalence in a sample of urban schoolchildren in Croatia. *Reprod Toxicol* 2010; 29:237–41. http://dx.doi.org/10.1016/j.reprotox.2009.11.006

22 Morleo M, Woolfall K, Dedman D et al. Under-reporting of Fetal Alcohol Spectrum Disorders: an analysis of hospital episode statistics. *BMC Pediatr* 2011; 11:14.

23 Smith LA, Foxcroft D. *Drinking in the UK: an exploration of trends* [Internet]. York: Joseph Rowntree Foundation; 2009 [cited 2012 March 28]. Available from: http://www.jrf.org.uk/publications/drinking-in-the-uk

24 Warren KR, Hewitt BG, Thomas JD. Fetal Alcohol Spectrum Disorders: research challenges and opportunities. *Alcohol Research & Health* 2011; 34:4–14.

25 Hutson J. Meconium fatty acid ethyl esters and prediction of fetal alcohol effects. *J FAS Int* 2006; 4:1–3.

26 Mukherjee RA, Eastman N, Turk J et al. Fetal Alcohol Syndrome: law and ethics. *Lancet* 2007; 369:1149–50. http://dx.doi.org/10.1016/S0140-6736(07)60542-X

27 Henderson J, Gray R, Brocklehurst P. Systematic review of effects of low-moderate prenatal alcohol exposure on pregnancy outcome. *BJOG* 2007; 114:243–52. http://dx.doi.org/10.1111/j.1471-0528.2006.01163.x

28 Mukherjee RA, Hollins S, Abou-Saleh MT et al. Low level alcohol consumption and the fetus. *BMJ* 2005; 330:375–6. http://dx.doi.org/10.1136/bmj.330.7488.375

29 Disney ER, Iacono W, McGue M et al. Strengthening the case: prenatal alcohol exposure is associated with increased risk for conduct disorder. *Pediatrics* 2008; 122:e1225–30. http://dx.doi.org/10.1542/peds.2008-1380

30 Kelly YJ, Sacker A, Gray R et al. Light drinking during pregnancy: still no increased risk for socioemotional difficulties or cognitive deficits at 5 years of age? *J Epidemiol Community Health* 2012; 66:41–8. http://dx.doi.org/10.1136/jech.2009.103002

31 Sayal K, Heron J, Golding J et al. Prenatal alcohol exposure and gender differences in childhood mental health problems: a longitudinal population-based study. *Pediatrics* 2007; 119:e426–34. http://dx.doi.org/10.1542/peds.2006-1840

32 Abel EL. Fetal Alcohol Syndrome: same old, same old. *Addiction* 2009;104:1274–5.http://dx.doi.org/10.1111/j.1360-0443.2008.02481.x

33 Alati R, MacLeod J, Hickman M et al. Intrauterine exposure to alcohol and tobacco use and childhood IQ: findings from a parental-offspring comparison within the Avon Longitudinal Study of Parents and Children. *Pediatr Res* 2008; 64:659–66. http://dx.doi.org/10.1203/PDR.0b013e318187cc31

34 Henderson J, Kesmodel U, Gray R. Systematic review of the fetal effects of prenatal binge-drinking. *J Epidemiol Community Health* 2007; 61:1069–73. http://dx.doi.org/10.1136/jech.2006.054213

35 Kaskutas LA, Graves K. Pre-pregnancy drinking: how drink size affects risk assessment. *Addiction* 2001; 96:1199–209. http://dx.doi.org/10.1046/j.1360-0443.2001.968119912.x

36 O'Leary CM, Nasser N, Kurinczuk JJ et al. Prenatal alcohol exposure and risk of birth defects. *Pediatrics* 2010; 126:e843–50. http://dx.doi.org/10.1542/peds.2010-0256

37 D'Onoforio BM, Van Hulle CA, Waldman ID et al. Causal inferences regarding prenatal alcohol exposure and childhood externalising problems. *Arch Gen Psychiatry* 2007; 64:1296–304. http://dx.doi.org/10.1001/archpsyc.64.11.1296

38 Patra J, Bakker R, Irving H et al. Dose-response relationship between alcohol consumption before and during pregnancy and risk of low birthweight preterm birth and small for gestational age (SGA): a systematic review and meta-analyses. *BJOG* 2011; 118:1411–21. http://dx.doi.org/10.1111/j.1471-0528.2011.03050.x

39 Chaudhuri JD. Effect of a single dose of ethanol on developing skeletal muscle of chick embryos. *Alcohol* 2004; 34:279–83. http://dx.doi.org/10.1016/j.alcohol.2004.07.012

40 Ieraci A, Herrera DG. Single alcohol exposure in early life damages hippocampal stem/progenitor cells and reduces adult neurogenesis. *Neurobiol Dis* 2007; 26:597–605. http://dx.doi.org/10.1016/j.nbd.2007.02.011

41 Ikonomidou C, Bittigau P, Ishimaru MJ et al. Ethanol-induced apoptotic neurodegeneration and Fetal Alcohol Syndrome. *Science* 2000;287:1056–60.http://dx.doi.org/10.1126/science.287.5455.1056

42 International Centre for Alcohol Policies. *International guidelines on drinking in pregnancy* [Internet]. Washington DC: International Centre for Alcohol Policies; 2009. Available from: http//www.icap.org/table/internationalguidelinesondrinkinginpregnancy 2009

43 Abel EL, Kruger M. What do physicians know and say about Fetal Alcohol Syndrome?: a survey of obstetricians, pediatricians and family medicine physicians. *Alcohol Clin Exp Res* 1998; 22:1951–4. http://dx.doi.org/10.1111/j.1530-0277.1998.tb05902.x

44 Gahagan S, Sharpe TT, Brimacombe M et al. Pediatrician's knowledge, training and experience in the care of children with Fetal Alcohol Syndrome. *Pediatrics* 2006; 118:e657–68. http://dx.doi.org/10.1542/peds.2005-0516

45 Haemmerlie FM, Merx CJ, Nelson SB. College vs junior high school students' knowledge of alcohol as a teratogen. *Psychol Rep* 1992; 71:809–10. http://dx.doi.org/10.2466/pr0.1992.71.3.809

46 Kesmodel U, Kesmodel PS. Drinking during pregnancy: attitudes and knowledge among pregnant Danish women, 1998. *Alcohol Clin Exp Res* 2002; 26:1553–60.http://dx.doi.org/10.1111/j.1530-0277.2002.tb02455.x

47 Lefebvre LG, Ordean A, Midmer D et al. Physicians' knowledge of alcohol, tobacco and folic acid in pregnancy. *Subst Abus* 2007; 28:3–9. http://dx.doi.org/10.1300/J465v28n01_02

48 Mack FR. *Parental knowledge of FAS and FAE*. Conference Proceedings of the Michigan Federal Chapter of the Council of Exceptional Children; 1996 Feb 29; Grand Rapids, Michigan, USA.

49 Morse BA, Idelson RJ, Sachs WH et al. Paediatricians' perspectives on Fetal Alcohol Syndrome. *J Subst Abuse* 1992; 4:187–95. http://dx.doi.org/10.1016/0899-3289(92)90018-S

50 Nanson JL, Bolaria R, Snyder RE et al. Physician awareness of Fetal Alcohol Syndrome: a survey of pediatricians and general practitioners. *CMAJ* 1995; 152:1071–6.

51 Hankin JR. Fetal Alcohol Syndrome prevention research. *Alcohol Res Health* 2002; 26:58–65.

52 McKnight A, Merrett D. Alcohol consumption in pregnancy: a health education problem. *J R Coll Gen Pract* 1987; 37:73–6.

53 Paley B, O'Connor MJ, Frankel F et al. Predictors of stress in parents of children with Fetal Alcohol Spectrum Disorder. *J Dev Behav Paediatr* 2006; 27:396–404. http://dx.doi.org/10.1097/00004703-200610000-00005

54 Granitsas J. Experiences of adoptive parents of children with Fetal Alcohol Spectrum Disorder. *Clinical Excellence for Nurse Practitioners* 2004; 8:22–8.

55 Gardner J. Living with a child with Fetal Alcohol Syndrome. *MCN Am J Matern Child Nurs* 2000; 25:252–7. http://dx.doi.org/10.1097/00005721-200009000-00007

56 Brown JD, Bednar LM, Sigvaldason N. Causes of placement breakdown for foster children affected by alcohol. *Child and Adolescent Social Work Journal* 2007; 24:313–32. http://dx.doi.org/10.1007/s10560-007-0086-9

57 Brown JD, Sigvaldason N, Bednar LM. Foster parents perception of placement needs for children with a Fetal Alcohol Spectrum Disorder. *Children and Youth Services Review* 2005; 27:309–27. http://dx.doi.org/10.1016/j.childyouth.2004.10.008

58 Gelo J, O'Malley K. Family stress in parenting a child or adolescent with FASD. *Iceberg* 2003; 13:1–4.

59 Sokol RJ, Janisse JJ, Louis JM et al. Extreme prematurity: an alcohol-related birth defect. *Alcohol Clin Exp Res* 2007; 31:1031–7. http://dx.doi.org/10.1111/j.1530-0277.2007.00384.x

60 Volpe JJ. Cognitive deficits in premature infants. *N Eng J Med* 1991; 325:276–8. http://dx.doi.org/10.1056/NEJM199107253250409

61 Czeizel AE, Petik D, Puho E. Smoking and alcohol drinking during pregnancy. The reliability of retrospective maternal self-reported information. *Cent Eur J Public Health* 2004; 12:179–83.

62 Gray R, Mukherjee RA, Rutter M. Alcohol consumption during pregnancy and its effects on neurodevelopment: what is known and what remains uncertain. *Addiction* 2009; 104:1270–3. http://dx.doi.org/10.1111/j.1360-0443.2008.02441.x

63 Hannigan JH. What research with animals is telling us about alcohol-related neurodevelopmental disorder. *Pharmacol Biochem Behav* 1996; 55:489–99. http://dx.doi.org/10.1016/S0091-3057(96)00251-1.

64 Sulik KK. Genesis of alcohol-induced craniofacial dysmorphism. *Exp Biol Med* 2005; 230:366–75.

65 Office for National Statistics. *Drinking: adults' behaviour and knowledge in 2008* [Internet]. South Wales: ONS; 2009 [cited 2012 March 28]. Available from: http://www.ons.gov.uk/ons/rel/lifestyles/drinking--adult-s-behaviour-and-knowledge/2008-report/index.html

66 Office for National Statistics. *Smoking and drinking among adults, 2008* [Internet]. South Wales: ONS; 2010 [cited 2012 March 28]. Available from: www.ons.gov.uk/ons/rel/ghs/general-lifestyle-survey/2009-report/smoking-and-drinking-among-adults--2009.pdf

67 Leichter J, Lee M. Method of ethanol administration as a confounding factor in studies of Fetal Alcohol Syndrome. *Life Sciences* 1982; 31:221–7. http://dx.doi.org/10.1016/0024-3205(82)90581-1

68 Cudd TA. Animal model systems for the study of alcohol teratology. *Exp Biol Med (Maywood)* 2005; 230:389–93.

69 Jacobson JL, Jacobson SW. Effects of prenatal alcohol exposure on child development. *Alcohol Res Health* 2002; 26:282–6.

70 Rasmussen C. Executive functioning and working memory in Fetal Alcohol Spectrum Disorder. *Alcohol Clin Exp Res* 2005; 29:1359–67. http://dx.doi.org/10.1097/01.alc.0000175040.91007.d0

71 Mattson SN, Riley EP. Parent ratings of behaviour in children with heavy prenatal alcohol exposure and IQ-matched controls. *Alcohol Clin Exp Res* 2000; 24:226–31. http://dx.doi.org/10.1111/j.1530-0277.2000.tb04595.x

72 Mattson SN, Goodman AM, Caine C et al. Executive functioning in children with heavy prenatal alcohol exposure. *Alcohol Clin Exp Res* 1999; 23:1808–15. http://dx.doi.org/10.1111/j.1530-0277.1999.tb04077.x

73 Burden MJ, Jacobson SW, Sokol RJ et al. Effects of prenatal alcohol exposure on attention and working memory at 7.5 years of age. *Alcohol Clin Exp Res* 2005; 29:443–52. http://dx.doi.org/10.1097/01.ALC.0000156125.50577.EC

74 Burden MJ, Jacobson SW, Jacobson JL. Relation of prenatal alcohol exposure to cognitive processing speed and efficiency in childhood. *Alcohol Clin Exp Res* 2005; 29:1473–83. http://dx.doi.org/10.1097/01.alc.0000175036.34076.a0

75 Roebuck TM, Mattson SN, Riley EP. Interhemispheric transfer in children with heavy prenatal alcohol exposure. *Alcohol Clin Exp Res* 2002; 26:1863–71. http://dx.doi.org/10.1111/j.1530-0277.2002.tb02494.x.

76 Sampson PD, Kerr B, Carmichael Olson H et al. The effects of prenatal alcohol exposure on adolescent cognitive processing: a speed-accuracy tradeoff. *Intelligence* 1997; 24:329–53. http://dx.doi.org/10.1016/S0160-2896(97)90064-7

77 Spadoni AD, McGee CL, Fryer SL et al. Neuroimaging and Fetal Alcohol Spectrum Disorders. *Neurosci Behav Rev* 2007; 31:239–45. http://dx.doi.org/10.1016/j.neubiorev.2006.09.006

78 Mattson SN, Roesch SC, Fagerlund A et al. Towards a neurobehavioural profile of FASD. *Alcohol Clin Exp Res* 2010; 34:1640–50. http://dx.doi.org/10.1111/j.1530-0277.2010.01250.x

79 Kodituwakku PW. Neurocognitive profile in children with Fetal Alcohol Spectrum Disorders. *Dev Disabil Res Rev* 2009; 15:218–24. http://dx.doi.org/10.1002/ddrr.73

80 Baer JS, Sampson PD, Barr HM et al. A 21-year longitudinal analysis of the effects of prenatal alcohol exposure on young adult drinking. *Arch Gen Psychiatry* 2003; 60:377–85. http://dx.doi.org/10.1001/archpsyc.60.4.377.

81 Streissguth AP, O'Malley K. Neuropsychiatric implications and long-term consequences of Fetal Alcohol Spectrum Disorders. *Semin Clin Neuropsychiatry* 2000; 5:177–90. http://dx.doi.org/10.1053/scnp.2000.6729

82 Spohr HL, Willms J, Steinhausen HC. Prenatal alcohol exposure and long-term developmental consequences. *Lancet* 1993; 341:907–910. http://dx.doi.org/10.1016/0140-6736(93)91207-3

83 Famy C, Streissguth AP, Unis AS. Mental illness in adults with Fetal Alcohol Syndrome or Fetal Alcohol Effects. *Am J Psychiatry* 1998; 155:552–4.

84 Barr HM, Bookstein FL, O'Malley KD et al. Binge drinking during pregnancy as a predictor of psychiatric disorders on the structured clinical interview for DSM-IV in young adult offspring. *Am J Psychiatry* 2006; 163:1061–5. http://dx.doi.org/10.1176/appi.ajp.163.6.1061

85 Huggins JE, Grant T, O'Malley K et al. Suicide attemps among adults with Fetal Alcohol Spectrum Disorders: clinical considerations. *Mental Health Aspects of Developmental Disabilities* 2008; 11:33–40.

Substance use among 15-16 year olds in the UK

Key findings from the 2011 European School Survey Project on Alcohol and Other Drugs (ESPAD)

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Introduction

The European Survey Project on Alcohol and Drugs (ESPAD) is conducted every four years and collects comparable data on trends in substance use among 15-16 year old school pupils across Europe. It is a high-quality survey and provides important data to support policy, practice and research. In 2011 36 countries and around 100,000 students took part; with 1712 being from the UK. The UK has taken part in ESPAD since it began in 1995, and the survey is now in its fifth data collection stage. This briefing presents a focussed summary of key results for the UK from the 2011 survey that was undertaken on behalf of the UK by the Centre for Public Health. It should be read alongside the full ESPAD report (see www.espad.org). Longer-term trends are also discussed and comparisons are made with other surveys of substance use among school pupils in the UK (Currie *et al*., 2012[1], Fuller *et al*., 2011[2]). The full ESPAD report contains further data on a range of associated substance use and risk taking behaviours.

Summary of key trends

BEHAVIOUR	% In 1995	% In 2007	% In 2011	APPROXIMATE NUMBER OF 15 AND 16 YEAR OLDS ACROSS THE UK IN 2010*	RANK OF ESPAD COUNTRIES (1ST BEING THE COUNTRY WHERE BEHAVIOUR IS MOST PREVALENT)
Lifetime smoking	68	52	47	702,000	23rd
Last 30 days smoking	36	22	23	344,000	24th
Lifetime alcohol use	94	92	90	1,345,000	15th
Last 30 days alcohol use	74	70	65	971,000	12th
Having drank at least 5 drinks on one occasion in the last 30 days	50	54	52	777,000	7th
Lifetime use of any illicit drug	42	29	27	403,000	5th
Lifetime cannabis use	41	29	25	374,000	5th
Cannabis use in last 30 days	24	11	13	194,000	4th

** Based on 1,494,000 15 and 16 year olds in the UK in 2010*

Methodology

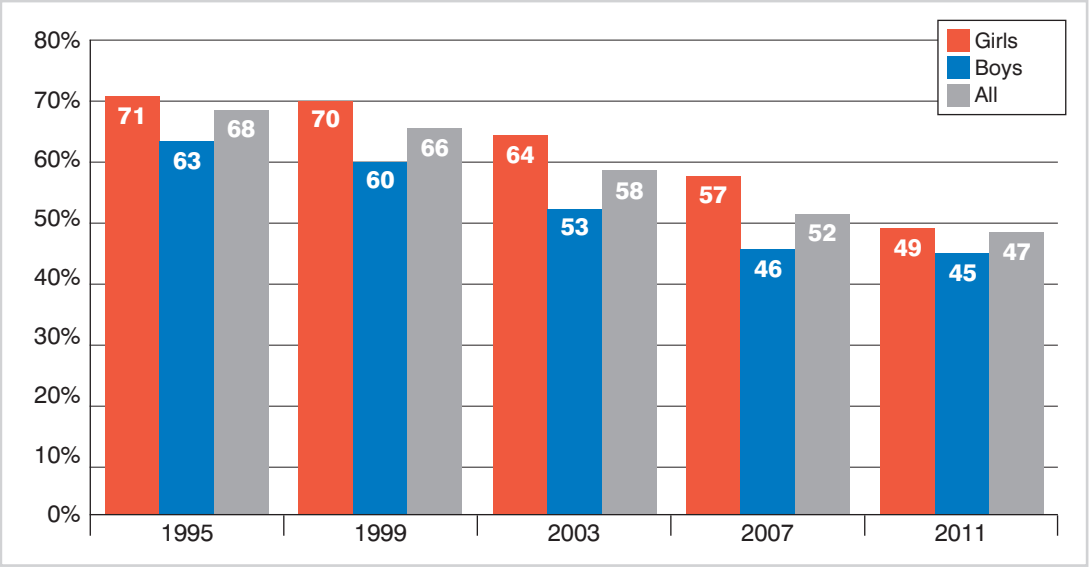
To provide comparable data across countries and time, the survey is conducted using a standardised methodology and questionnaire. The UK data was collected during March-April 2011 in a random sample of 74 secondary schools in England, Wales, Scotland and Northern Ireland (sampling was in proportion to the number of schools in each country). Participants were 1712 school children born in 1995 and this comprised a relatively even split of boys (n=865) and girls (n=847). All schools that participated in the survey reported good pupil engagement with the research. Busy school schedules and involvement in other research projects meant that the overall number of schools agreeing to take part in the survey was low (approximately 1 in 17 agreed)[3]. However, as participating schools were selected using a random sampling process the data can be considered representative of the wide variety of UK schools.

Key findings

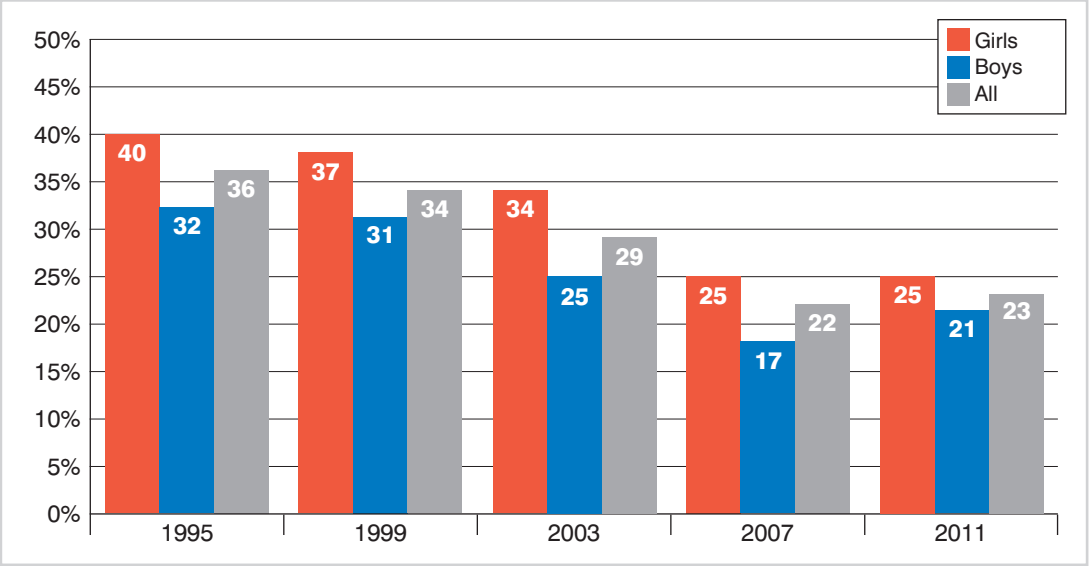
Cigarettes

In 2011, 47% of UK school pupils reported having tried smoking at least once in their lifetime (see Graph 1). 23% reported having smoked in the last 30 days (see Graph 2). 49% of girls reported lifetime smoking compared to 45% of boys. 25% of girls reported having smoked in the last 30 days compared to 21% of boys. The majority (66%) of pupils reported finding it 'fairly' or 'very' easy to obtain cigarettes. The UK is considered to have the most comprehensive set of tobacco control policies in Europe (Jooseens and Raw, 2010) and lifetime use of cigarettes by 15 and 16 year olds is below the ESPAD average of 54%. The proportion of pupils reporting smoking during the last 30 days was also below the ESPAD average of 28%. These figures are similar to the 2009/10 World Health Organization's Health Behaviours in School-aged Children survey of smoking (Currie *et al*., 2012). For example, in 2009/2010, lifetime smoking prevalence for 15 year olds in England was 41%, 42% for Wales and 39% for Scotland (Currie *et al*., 2012). There has been a decrease in lifetime and last 30 days use of cigarettes in the UK since the start of the ESPAD survey in 1995 (see Graphs 1 and 2).

Graph 1: Lifetime use of cigarettes



Graph 2: Cigarette smoking in the last 30 days



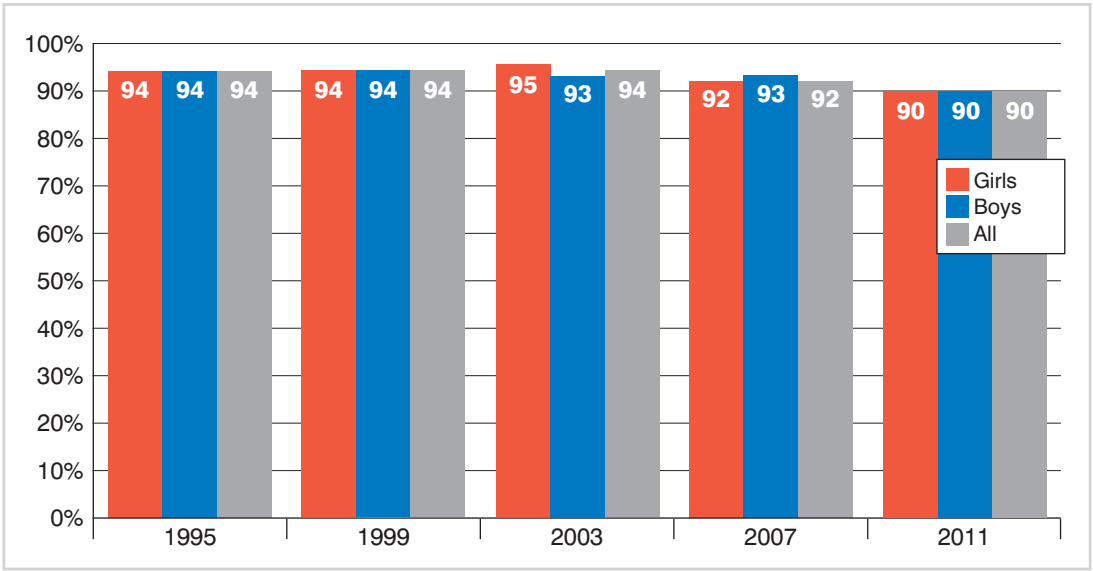
Alcohol

In 2011, 90% of 15-16 year old school pupils in the UK reported having drank an alcoholic drink at least once in their lifetime (see Graph 3). 85% reported having drank alcohol in the last 12 months and 65% in the last 30 days (see Graph 4). There were no sex differences. 52% of pupils reported having drank 5 or more drinks on at least one occasion in last 30 days (see Graph 5). 54% of girls reported having drank at least 5 drinks on one occasion in the last 30 days compared to 50% of boys. 26% of pupils reported having been drunk in the last 30 days[4]. Girls (29%) reported drunkenness more than boys (24%). The majority (82%) of pupils reported that alcohol is 'fairly' or 'very' easy to obtain. More on-premise purchasing of alcohol for own consumption (40%) was reported than off premise purchasing (26%).

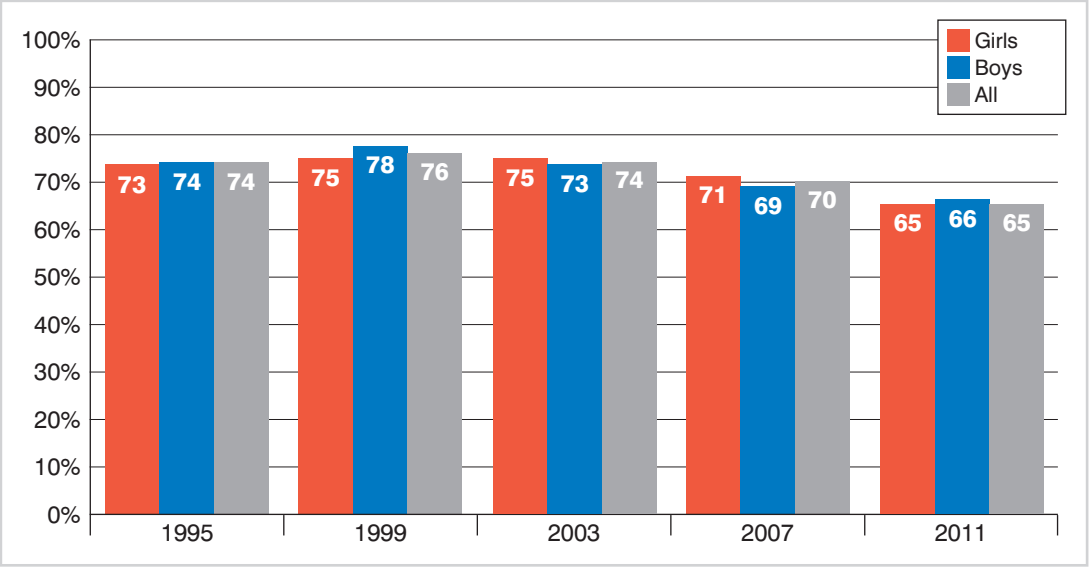
Participants reported experiencing a number of alcohol-related problems in the last 12 months. 13% of pupils reported having experienced relational problems (e.g. serious problems with friends) that they associated with their personal use of alcohol. 12% reported having experienced sexual problems (e.g. engaged in unprotected sexual intercourse) and 11% reported having experienced individual problems (e.g. performing poorly at school or work). 9% reported having experienced delinquency problems related to their own alcohol use (e.g. physical fight).

The UK is classed as a high prevalence country for alcohol use. For example, the proportions of pupils reporting having used alcohol during their lifetime (90%), the last 12 months (85%) and the last 30 days (65%) were higher than the ESPAD averages (87%, 79%, 57% respectively). However, since 1995, the proportion of pupils reporting lifetime and last 30 days alcohol use have decreased (See Graph 3 and 4). Similar downward trends have been reported in national surveys of substance use among UK school children (Fuller *et al*., 2011). Despite this, the number of pupils reporting heavy episodic drinking (i.e. at least 5 drinks on one occasion), has not fallen since 1995 (see Graph 5).

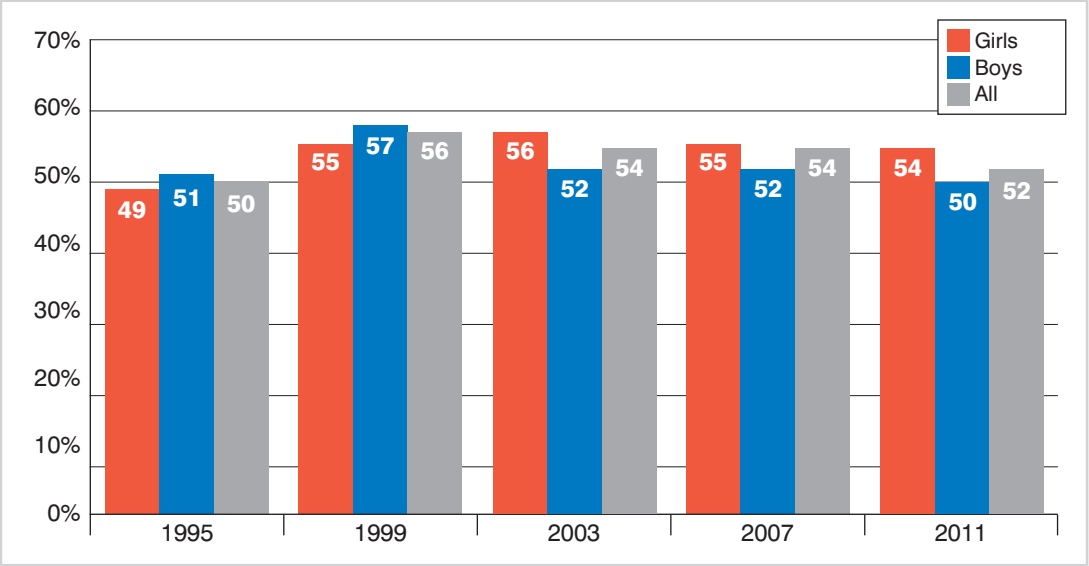
Graph 3: Lifetime use of alcohol



Graph 4: Use of alcohol in the last 30 days



Graph 5: Consuming more than five alcoholic drinks on one occasion in the last 30 days

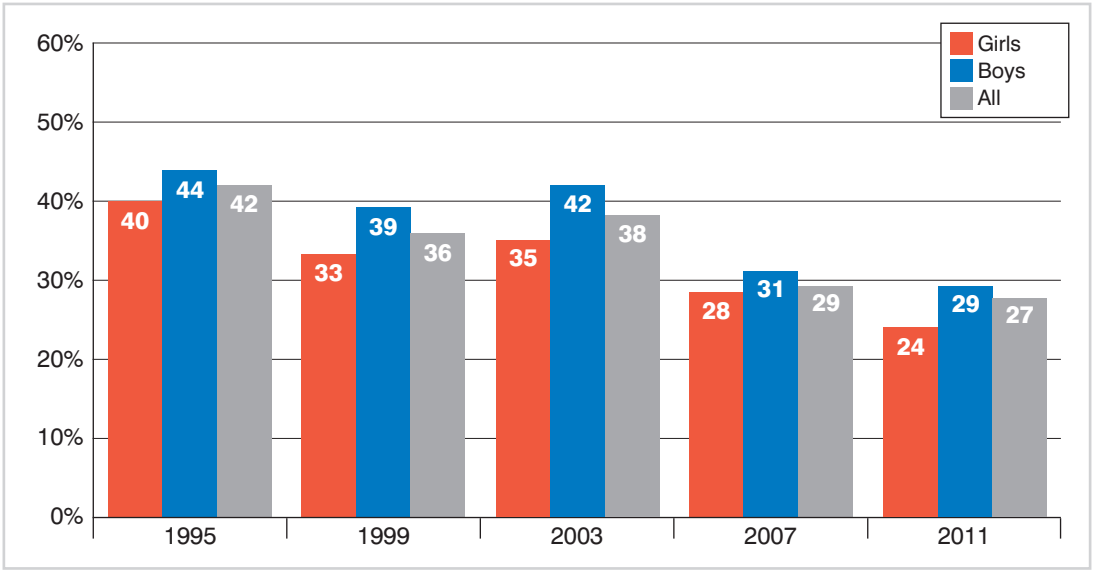


Illicit drugs

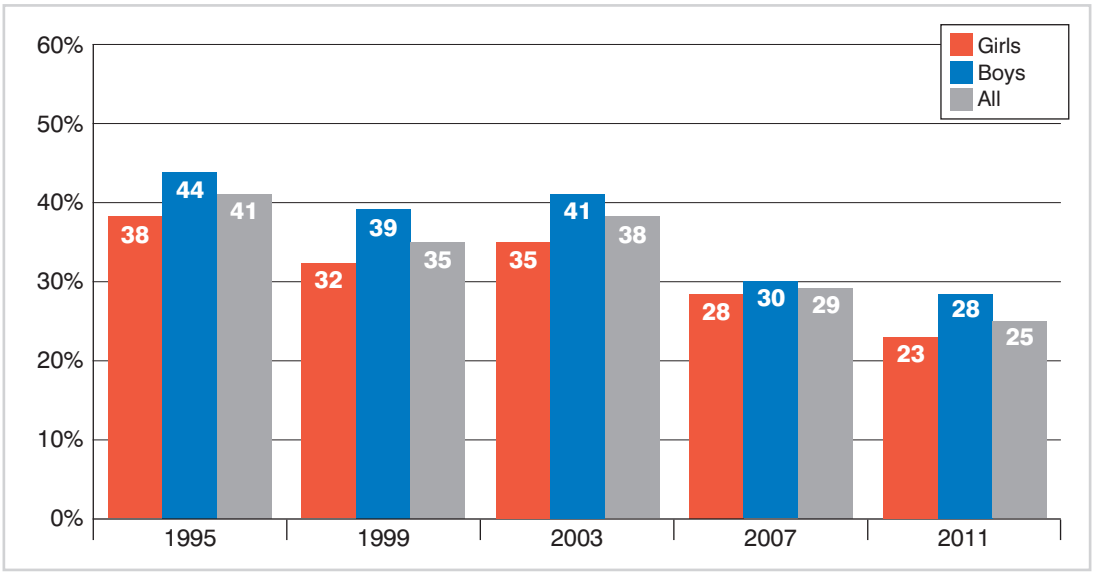
Just over a quarter (27%) of pupils reported having ever used any illicit drug in their lifetime[5] (see Graph 6). The proportion of pupils reporting lifetime use of any drug was higher among boys (29%) than girls (24%) (See Graph 6). Pupils reported higher proportions of cannabis use than any other drug for lifetime (25%), last 12 month (21%) and last 30 days use (13%) (see Graph 7 and 8). Cannabis use has also been reported as the most commonly used drug among this age group in national and European surveys (Fuller *et al*., 2011, Currie *et al.*, 2012). More boys reported lifetime, last 12 months and last 30 days cannabis use than girls (see Graph 7 and 8), and overall 42% of pupils reported that cannabis was 'fairly' to 'very' easy to obtain.

There has been a decrease in the proportion of 15-16 year old school pupils reporting having used illicit drugs during their lifetime since 1995 (see Graph 6). 42% of pupils reported having ever used an illicit drug in 1995, compared to 29% in 2007 and 27% in 2011. Similarly, the proportions of pupils reporting having used cannabis during their lifetime and during the last 30 days has decreased since 1995 (See Graph 7 and 8).

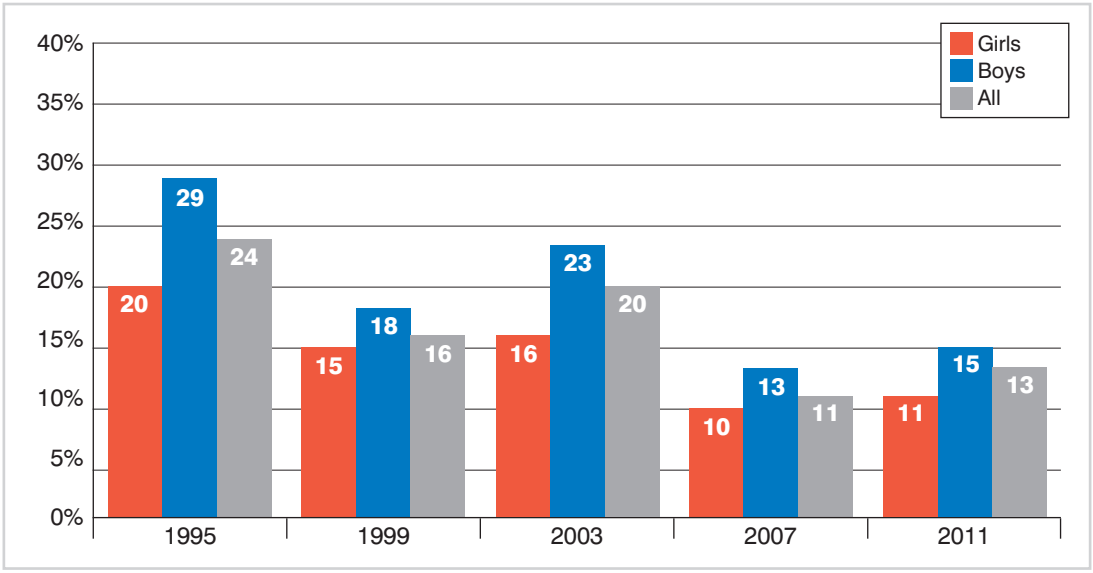
Graph 6: Lifetime use of any drug



Graph 7: Lifetime use of cannabis



Graph 8: Cannabis use in the last 30 days



Conclusions and issues to consider

A number of key variables have been included in this briefing to provide a summary of substance use prevalence and trends among 15-16 year old school pupils in the UK. There appears to have been a reduction in smoking among school pupils since the first phase of data collection in 1995. Girls continue to smoke more than boys. Similarly there has been a decrease in alcohol use since 1995. Critically however, patterns of heavy drinking (consuming more than 5 drinks in a single occasion) have not changed since 2003 and more girls than boys now report heavy drinking and drunkenness in the last 30 days. Lifetime use of illicit drugs has also decreased over time, but boys continue to report greater use of all types of drug use than girls

The 2011 ESPAD survey has provided important information about the extent of substance use among 15-16 year olds in the UK and how these have changed over the last 16 years. The survey provides intelligence to assist with the design and targeting of interventions that support healthy adolescent development, as well as for monitoring the impact of national policies. Comparison with wider European data provides insight as to whether the trends in substance use reported here are as a result of UK specific factors or reflect wider European changes. Homogenisation of international youth cultures, globalisation of addictive goods marketing, and the increasing importance of EU strategies designed to limit the harmful impact of substance use means that it is essential that UK data are considered alongside those of other countries. ESPAD is unique in that it is the only survey that provides comparative data on a wide range of substance use and risk behaviours at the European level. The continued participation of the UK in ESPAD is critical for providing basic intelligence on the substance use of UK youths compared to their European counterparts. However, quality intelligence on risk taking and related harms amongst UK school pupils is threatened by an increasingly busy curriculum, multiple requests for research in UK schools and a lack of national school research coordination.

References

Currie, C. *et al*., (2012) Social determinants of health and well-being among young people: Health Behaviour in School-Aged Children (HBSC) study: international report from the 2009/2010 survey. Copenhagen: World Health Organization Regional Office for Europe.

Fuller, E. *et al*., (2011) Smoking, Drinking and Drug Use among Young People in England in 2010. London: NHS Information centre for health and social care and Natcen.

Joosens, L., and Raw, M. (2010) The Tobacco Control Scale 2010 in Europe. Belgium: Association of European Cancer Leagues.

Notes

1. The Health Behaviour in School-aged Children (HBSC) survey is a cross-national study conducted in collaboration with the WHO Regional Office for Europe. The survey provides insight into the well-being and health behaviours (including substance use) of 11, 13 and 15 year olds across Europe. Data from the HBSC survey presented in this report are for 2009/2010 and are for 15 year olds only.

2. The UK government conducts a national annual survey to monitor smoking, drinking and drug use among secondary school pupils aged 11 to 15. Data from the 11-15 year old survey presented in this report are from 2010 and are for 15 year olds only.

3. The initial sample was randomly chosen from all schools in the UK and stratified in order to ensure schools from England, Wales, Scotland and Northern Ireland were included in numbers proportional to their population. If any school refused to participate another school was randomly chosen to take their place. The process was repeated until 74 schools had agreed to participate giving a low total school participation rate of 6%.

4. Drunkenness is self-defined.

5. ‘Any drug’ includes cannabis, amphetamine, cocaine, crack, ecstasy, LSD and heroin.

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