December 2009 Issue 2

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

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Issue 2 - December 2009

Dr Maurice Titran

INTRODUCTION

NOFAS-UK would like to dedicate the second issue of the FETAL ALCOHOL FORUM to Dr Maurice Titran, who died 12th September 2009. Dr Titran was a paediatrician and a Fetal Alcohol pioneer in France.

Dr Titran's son, Benoit, was responsible for changing the law requiring pregnancy warning labels on alcohol in France. Benoit Titran has contributed an excerpt to the Forum from his book about French FASD History.

We also extend our appreciation to our Founding Patron, Celia Atkin and Lord Mitchell, who launched the FORUM in the House of Lords, London in January 2009, and to Elizabeth Mitchell for her editorial contribution.

In our second issue of the FETAL ALCOHOL FORUM, we are once again fortunate to have contributions from leading Fetal Alcohol Spectrum Disorder world experts.

We begin with original articles written for us by: Professors Moira and Martin Plant (United Kingdom), Professor Ed Riley (United States), Professor Denis Viljoen (South Africa), Dr Rod Densmore (Canada), and Dr Sherly Parackal (New Zealand).

The second section contains 59 abstracts with links to the latest FASD research, including a new study that has calculated the prevalence of FASD to be 3 to 5 in 100.

It is the hope of NOFAS-UK to inform, connect, build and strengthen the international FASD medical network.

Please pass the FETAL ALCOHOL FORUM on to colleagues, download the FORUM from our website (www.nofas-uk.org) or contact us if you would like to contribute an original article or refer us to new research. To join the FORUM network, click here.

Thank you for being part of the network that can help prevent FASD and improve the life outcomes for children and adults who live with Alcohol Related Brain Damage. We all need and can help each other.

Susan Fleisher
Executive Director
NOFAS-UK

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ORIGINAL ARTICLES BY FASD EXPERTS

January 2009

I. ALCOHOL AND WOMEN IN THE UNITED KINGDOM: WHAT'S HAPPENING?

Professor of alcohol studies[1] and Professor of addiction studies,[2] Alcohol and Health Research Unit, Centre for Public Health Research, University of the West of England, Blackberry Hill, Bristol BS16 1DD

Moira L. Plant[1] **Martin A. Plant[2]**

Drinking by women in the United Kingdom

There has been an enormous change in the drinking habits of women in the United Kingdom in recent years. This is highlighted by the remarkable fact that teenagers in England now drink twice as much as they did in 1990 (National Centre for Social Research/National Foundation for Educational Research, 2005). The United Kingdom is one of the very few countries in Western Europe where, until recently, per capita alcohol consumption and its allied problems have been rising (Alcohol Concern, in press).

Between 1995 and 2003, the proportion of UK girls aged 15 and 16 years who had 'binged' (five or more UK units, three times or more in the past 30 days) rose from 20% (1995) to 27% (1999), and had increased again to 29% by 2003 (Hibell et al., 1997, 2001, 2004; Plant et al., 2005). Further studies of adolescent and teenage drinking have shown similar results (National Centre for Social Research/National Foundation for Educational Research, 2005; Office for Standards in Education, Children's Services and Skills, 2007). Adolescent and teenage girls who drink heavily are especially likely to smoke tobacco and to use illicit drugs (Miller, 1997; Plant and Plant, 1992, Plant and Plant 2006). Many of these young women are now in their early twenties. There is no evidence that they have reduced their drinking. One particularly striking finding is that teenage girls (aged 15 and 16 years) in the British Isles (the UK, Ireland and the Isle of Man) were found in a 2003 survey to be more likely than their male counterparts to be binge drinkers. This was not the case in 32 other European countries that were surveyed (Hibell et al., 2004). Clearly something unusual has been happening to foster such heavy drinking amongst young women in the UK. This constitutes a major social change from the previous situation in which females invariably drunk much less than males. This may reflect a number of factors. These include new social attitudes, greater female social and economic empowerment, and alcohol marketing aimed at women.

Approximately 90% of women in Britain consume alcoholic drinks at least occasionally. Between 1980 and 2006 per capita alcohol consumption in the UK rose by 21% (British Beer and Pub Association, 2007). A survey of women in England, Scotland and Wales revealed that 8% of women aged 18–24 years had consumed at least 35 units of alcohol in the past week (Plant and Plant, 2001; Plant et al., 2002). This is defined as 'harmful drinking' by the Department of Health and the Home Office (2007). High levels of heavy drinking by young women have been noted by other investigators (Office for National Statistics, 2004, 2006; Scottish Government, 2007; Williamson et al., 2003). The General Household Survey indicates that between 1998 and 2004 the proportions of women aged 16 and older drinking more than six units on at least one day in the past week rose from 8 % to 9%. Over the period 2000 to 2002, 26% of women aged 16 to 24 years were drinking at this level. In 2004, this proportion was 24% (Office for National Statistics, op. cit.).

Rising health damage amongst UK women

Most of the commonplace alcohol-related harm amongst women is acute, and associated with accidents, arguments and assaults, including sexual assault or rape (Plant, 2008). Harm associated with childbearing includes miscarriage and prenatal alcohol exposure (Henriksen et al., 2004). Mortality rates for liver disease have risen steeply in the past decade, and the mean age at death has fallen significantly. The Office for National Statistics has recently reported that:

The alcohol-related death rate in the UK increased from 6.9 per 100,000 population in 1991 to 12.9 in 2005. The number of alcohol-related deaths has more than doubled from 4,144 in 1991 to 8,386 in 2005. (Office for National Statistics, 2006)

There has been an upsurge in the numbers of younger women developing and dying from alcohol-related liver disease (Donaldson, 2001; Gilmour, 2004). In addition, there has been a significant increase in young women being admitted to hospital in England for alcohol-related psychiatric problems (British Medical Association, 2008; Williams et al., 2005).

The implications for alcohol-related birth damage

Rising heavy drinking by young British women suggests that the number of babies with alcohol-related birth defects may be increasing. Evidence suggests that women will continue to drink at pre-pregnancy levels until the pregnancy is confirmed. This is particularly true if the pregnancy was unplanned (Tough et al., 2006). It has been noted that approximately 84% of UK mothers under the age of 20 years report their pregnancy was unplanned (Dex and Josh, 2005). Therefore, at the most vulnerable time for the foetus, the mother may still be drinking at pre-pregnancy level. However, the lack of recent UK evidence on alcohol-related birth defects leaves the scale of this problem unknown.

The UK has the highest rate of teenage pregnancy in Europe. Such pregnancies are especially commonplace amongst young women living in socially deprived areas (Office for National Statistics, 2005; Social Exclusion Unit, 1999; Youth Information, 2007). Teenagers and other young people who engage in unprotected sex are very likely to be heavy drinkers (Alcohol Concern, 2002; Bullock, 2001; Cooper, 2002; Karshin, 2002; Leigh and Miller, 1995; Leigh et al., 2007).

Conclusion

The recent increase in alcohol consumption and alcohol-related harm amongst UK women, in particular young women, has been substantial and alarming. It has also been very unusual by international standards. These negative trends have serious implications for both immediate and future health consequences associated with heavy drinking.

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November 2009

II. RAMBLINGS FROM A JET-LAGGED FASD RESEARCHER – "THE TIMES THEY ARE A CHANGING"

PROFESSOR EDWARD RILEY
Ph.D

So, I am sitting in yet another airport waiting for a flight, but at least this time I am heading home to San Diego. I just attended a meeting of the American Academy of Child and Adolescent Psychiatry, where Ken Jones, Tina Chambers, Kathy Sulik, Susan Rich and I presented a symposium on Fetal Alcohol Spectrum Disorders. For those of you who don't know them, Ken was the person who originally identified FAS in 1973 with David Smith at the University of Washington. Tina Chambers is an epidemiologist who is doing some very interesting work in Moscow and the Ukraine. Kathy Sulik is well known for her work on animal models of FAS, and demonstrated years ago that you could produce the face of FAS in a mouse with a single high dose binge exposure. Susan Rich is a psychiatrist in clinical practice with a long-standing interest in FASD, and it was Susan who put this meeting together.
I was somewhat sceptical on my way to the meeting, since our symposium was scheduled for the last day of the meeting at 7:30 am and, to be honest, child psychiatrists have never flocked to any symposium on FASD at which I have presented. Well, to my surprise we had a large audience of very interested, practicing and academic psychiatrists who had lots of excellent questions. Perhaps as Bob Dylan once said, indeed "the times they are a changing."

As some of you know, I have a long interest in international collaborations to study FASD, since I think we can learn the most by comparing the similarities and differences across populations. It allows us to build a large subject base and learn what cultural and environmental effects may mitigate or increase the consequences of prenatal alcohol exposure. Similarities between different populations argue that those effects are due to prenatal alcohol exposure, while differences can lead to insights about possible factors that moderate prenatal alcohol effects.
It has been an extremely busy year on the international research front, which is one reason I am sitting in yet another airport thinking about how the times have indeed changed. I attended my first international FASD meeting in 1990, which was planned and hosted by Consuelo Guerri in Valencia, Spain. At that meeting, Belgium, France, Spain, and the US were represented. In 1998, I organized a meeting in Missillac, France and this time we had several scientists representing France, Germany, Spain, Sweden, and the US. A few years later, Consuelo Guerri and I organized another international research meeting, and things improved tremendously, as we had representatives from numerous European countries, as well as from Russia, S. Africa, and the US. But international interest in FASD has grown dramatically in the last few years. I can remember when the position of many countries was to simply deny that FASD was a problem in their country – women didn't drink in some countries, they only drank with meals, their populations were too prosperous, too educated or whatever. In one of my first international research projects, we were advised to "stay under the radar" lest we upset some political applecart and have our project end. But countries have gone from no recommendations whatsoever about drinking during pregnancy, to taking steps to prevent FASD and recognizing the major public health implications that gestational alcohol exposure can have. The international approach to FASD can be seen by the attendance at the meetings put on in British Columbia in the past few years, which were the largest FASD meetings I have ever attended.

Late last year, I participated in a meeting between INSERM in France and the US National Institute on Alcohol Abuse and Alcoholism (NIAAA) in Paris. One of the areas for increased cooperation in terms of research between these two governments was FASD. In September of this year, I had the opportunity to visit several cities in Poland as part of a contingent from the US, and at the end of the trip an agreement was signed between the Ministry of Health of Poland and the NIAAA for collaborations in five areas of alcohol research, one of which was FASD. Following that trip, I went to

the excellent meeting put together by Hans Spohr in Berlin. While Hans, an outstanding clinician and scientist, has a long history in FASD work, I think he sometimes felt he was working in isolation and that little was being done to help his kids. But I got a different feeling this time, one of cooperation and collaboration between German clinicians and scientists and officials in public health. Shortly after that meeting, the French Senate convened to discuss how to deal with FASD and although I wasn't there, I heard that it went extremely well. Next month, I am heading to Seoul, South Korea, where Phil May and I have been invited to present at a meeting on FASD and to also participate in the first meeting of a new Asian society dealing with alcohol and drug abuse. I also have a new addition to my laboratory, Dr. So Hee Lee, courtesy of the S. Korean government, as they want to train their physicians and scientists about FASD.

Next year, the Europeans will be having the First European Conference on FASD entitled Fetal Alcohol Spectrum Disorders: Growing Awareness in Europe, which is being orchestrated by Diane Black. I am on the program committee and the number of scientists available from throughout Europe who will be speaking on topics ranging from basic molecular biology of FASD to clinical concerns is amazing. Certainly there is a growing awareness in Europe, but I think it is even broader. FASD is beginning to be understood as a major international public health problem in many parts of the world and I hear regularly from people in S. America and Australia, as well as Europe and Asia, about the need to study FASD in their own countries.

So, I am jet lagged (between the end of August and mid November, I will have flown through 70 different time zones), but I hope I have provided some insights into how research on an international level has grown in the last few years. It has grown from something that only a few people in a few countries were interested in, to one in which the number of investigators has increased significantly and perhaps more importantly to one in which governments are beginning to understand the major health issues involved in FASD and are willing to step in to do their part, both in advancing knowledge and in providing policy. I have been very fortunate that my interest in FASD has allowed me to visit so many places in the world (even if sometimes I feel like I never get to leave the hotel or meeting room), but I have been more fortunate to have been able to meet so many wonderful people, who have shown me remarkable kindness and goodwill. I just hope I have contributed a little to these "changing times."

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III. **FETAL ALCOHOL SPECTRUM DISORDER IN SOUTH AFRICA – A MAJOR HEALTH CHALLENGE**

PROFESSOR DENIS VILJOEN
CHAIRMAN, FOUNDATION FOR ALCOHOL RELATED RESEARCH, DIRECTOR OF THE CENTRE FOR GENETIC RESEARCH AND EDUCATION, UNIVERSITY OF STELLENBOSCH, CAPE TOWN

Fetal alcohol spectrum disorder (FASD) affects many children and spares few communities in the world from its ravages. It is claimed to be the most frequent cause of preventable mental handicap in the world and is devastating in its lifelong effects and serious sequelae in affected persons. In South Africa, the condition has been evaluated in several communities thought to be especially at risk and, from a population totalling 45 million, it has been estimated that 1 million persons have fetal alcohol syndrome (FAS) with a further 4 -5 million exhibiting signs of the lesser form of the condition, namely fetal alcohol spectrum disorder.

Since the seminal article by Jones and Smith published in the Lancet in 1973, more than 5000 medical manuscripts have been written worldwide. The Jones paper was preceded in 1969 by

Lemoine's manuscript on children born to alcoholic women in France. This latter article remained obscured in French Medical literature, as did ancient writings in the Bible and Torah, until research later revealed their collective wisdoms. FAS is the consequence of (usually) heavy alcohol ingestion by a mother on her unborn fetus. The diagnosis requires identification by a professional, well trained in dysmorphology a medical specialist who recognizes abnormal features in a person) following strict criteria laid down in the medical literature. FASD is more common and comprises less severe forms of the disorder which may occur 4 – 5 times more frequently than the more severe manifestations found in full FAS.

In South Africa, the Foundation for Alcohol Related Research was constituted in 1997 and has evaluated communities in 3 provinces for FAS/FASD. The need for this research followed a basic audit of clients investigated at Genetic Clinics undertaken at the only Paediatric Hospital functioning in the whole of Africa, namely the Red Cross War Memorial Children's Hospital in Cape Town. It was discovered that 1 to 10 children referred to these specialists clinics had the full stigmata of FAS. Much like NOFAS-UK, the FARR organization has since used every avenue to inform the general public, health planners and professionals, funding organizations and research groups regarding the dangers of FASD. FARR has been fairly successful in garnering support, especially from federally-funded research groups in the USA (National Institutes for Health (NIAAA) and Centres for Disease Control and Prevention (CDC)) in researching prevalence, risk factors, social conditions, behavioral pattern changes, genetic factors, speech and language deficits and many other associated with FASD. Unfortunately, as results shown below reveal, our governmental and local organizations have been slow to respond to the pandemic of FASD found in susceptible communities.

FARR's most telling research has emerged in publications revealing the very high prevalence's of FASD in several communities studied. (See Table I). In comparison to Developed Nations (USA, France and Sweden) the prevalence rates in our at-risk communities are mind-blowingly high. Unprecedented figures of 20 – 120/1000 school – entry children have been found in major studies in the Johannesburg area, in 2 towns in the Northern Cape Province and in a study repeated on three occasions in the Western Cape Province. These are the highest figures reported anywhere in the world. Due to the other disorders prevailing in our country (HIV/AIDS, Malaria, Tuberculosis and Malnutrition) health planners appear reluctant to part with significant resources to combat this pandemic.

TABLE 1 - International FAS Prevalence

Country	FAS Prevalence	
	per 1000	%
USA	1 - 3	0.1-0.3 %
France	1,2	0.1%
Sweden	1,33	0.1%
Certain sectors of American Indian Population	8	0.8%
South Africa Wellington (2002) De Aar (2002) Upington (2003) Johannesburg (2000)	 88 120 69 27	 8.8% 12% 6.9% 2.7%

Numerous non-governmental organizations (NGOs) have subsequently sprung up in South Africa to address FAS/FASD in susceptible communities. Public – minded individuals and organizations are to be commended for their enthusiasm in this regard, but as is often the case the disjointed, disorganized and unsustainable activities of such groups often wastefully exploit the few resources available to NGOs in South Africa.

Other findings from FARR's research have been the subject of some 40 manuscripts and congress presentations which have appeared in the international and local medical literature. These are briefly summarized below (for lack of space) and appear on our website. Significant findings have been:-

- Maternal risk factors associated with having a child with FASD, have been elucidated. These include binge drinking of large amounts of alcohol, poor socio-economic circumstances, cigarette smoking, low religiosity, limited education, single parenthood and maternal depression. (See May at al, American Journal of blic Health, July 2005, Vol 96, No 7, pages 1190 – 1199; Viljoen et al, Journal of Studies on Alcohol, January 2002, Vol 63, No1, pages 6 – 1) These risk factors will be different for every community.
- Photogrammetry has been employed for the rapid diagnosis of FASD and has been demonstrated to be remarkably specific and sensitive. The evaluation of several hundred children have been reported in many publications from FARR's collaborations with the Medical Imaging Unit at the University of Cape Town. (eg. Douglas et al, American Journal of Human Biology, 2003, vol 15, pages 573 – 578) Such methodologies may form the basis of surveillance in whole populations in future.
- Cognitive and Motor development in children with FASD has been evaluated amongst high – risk South African populations. Children have demonstrated deficiencies in several neurological domains including speech and hearing, performance, practical reasoning and eye/hand co-ordination. Surprisingly, locomotor subscales were relatively unaffected. (Adnams et al, Alcoholism: Clinical and Experimental Research, 2001, volume 25, pages 557 - 562).
- Genetic effects have been demonstrated in families spared FAS where polymorphisms of the ADH2 *2 molecule were significantly more common in controls than amongst FAS affected persons of mixed ancestry in the Western Cape Province of South Africa. This finding suggests that susceptibility in mothers and their offspring with FAS is increased because metabolism of alcohol is slower in these persons leading to higher blood alcohol concentrations compared with control subjects without FAS. These studies were the first reported in the literature to assist in understanding susceptibility to FAS amongst different populations. (See Alcoholism: Clinical and Experimental Research, 2001, Volume 25, pages 1719 – 1722)
- A new biomarker of fetal exposure to alcohol was reported in a study of meconuim (first stools passed by the baby after delivery) where ethyloleate concentrations were much higher in newborns exposed to recent alcohol use in their mother. This is a highly sensitive and specific indicator of maternal alcohol exposure during the latter stages of pregnancy. (See Bearer et al, Journal of Paediatrics, 2003, Volume 143, pages 463 – 469)
- The effects of prenatal alcohol exposure on infant visual acuity was demonstrated amongst children exposed to alcohol in a South African collaborative study. This was assessed by Teller Acuity Cards at 6 months post – delivery. The finding was consistent with clinical and annual evidence of alcohol related disruptions of the visual pathways. (See Carter et al, Journal of Paediatrics, 2005, Vol 147, pages 473 – 479).

Other research articles and contract with staff at the Foundation for Alcohol Related Research.
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IV. **FRANCE – FIRST IN EUROPE TO PUT PREGNANCY WARNING ON ALCOHOL**

BENOIT TITRAN

Fetal Alcohol Syndrome was identified by the French doctor, Paul Lemoine, in 1968. In 2004, Fetal Alcohol Spectrum Disorder (FASD) became a concern of the French Ministry of Health. In only a few short months, the problem moved from the medical arena to the judicial and political arena. This made the difference that would change a bit of French alcohol history.

Act 1: The Judicial Approach

In 2002, three mothers of children affected by FASD came to me, because I was a lawyer based in Lille. They explained that during their pregnancy nobody had warned them of the danger of drinking alcohol. They never imagined that they could do so much damage to their child. Though they knew that this condition was irreversible, they wanted other mothers to avoid inflicting the same harm on their children because of manifest ignorance.

What seemed clear was:
- the mothers were not wholly responsible due to their is a lack of information.
- doctors themselves had poor knowledge of this subject.

I looked at how to make this information available and compulsory. How could I pressure the government to act rather than to convict. The French system was completely different than the American judicial process. I wanted to avoid blame and find a way to make everyone accept their responsibility.

The main question was “Who” should give this information to women and “How could we do it? Should it be though the obstetricians or the GPs? But this would need more studying and training and there was insufficient time. It was necessary to find a more systematic way.

Alcohol is a basic product and therefore codified in legislation. Thus, it can be approached from a consumer angle. Consumers of alcohol have rights, and, they are entitled to be given correct and accurate information.

The law punishes *“whoever will, by some means or process, deceive the consumer about the risks inherent in the use of a product . . . or the precautions to be taken”*. This is what is called the *“offence of deceit of the goods”.* Since the rule applies to all basic products which are consumed, there is no reason to exclude alcohol from this rule.

As the lawyer, I argued that producers and distributors are under obligation to obey this rule. In fact the law already states that any danger in a product must be stated on the packaging for the attention of the consumer. If the producers do not observe this obligation, the government is obliged to enforce the law.

Thus, in the name of the three mothers who brought this matter to my attention, I brought it to the attention of the Court in Lille. It was clear that this would not resolve the problem, but I realised that the legal, and probably the public debate (via the media), would hopefully encourage the government to adopt a global approach.

Act 2: The Involvement of a Member of the Senate
Nothing would have been possible without the involvement of politicians. The second decisive ally in this affair was a Senator who did an enormous amount of work to inform Members of Parliament. During the discussions about the law of public health in 2004, the Senator, Anne-Marie Payet, raised an amendment on labeling in order to protect the fetus. It was first adopted and then rejected by the government, who was being pressurized by senators representing the wine lobby in the 19th January 2004 debate. They stressed the difficulties of the wine sector and how the economics of the industry would be affected. Nobody spoke about public health or pathology or suffering.

So the first reaction was that the amendment was unacceptable because it would have huge economic repercussions in a sector that is a major element of our culture. Emotions were running very high.

Another hobbyhorse of the representatives of the wine producers, was to try to differentiate between wine and spirits. However, the fetus does not differentiate between alcoholic drinks. But the wine sector won. This first legislative failure was extremely disappointing, but not useless, because it sharply addressed the medical profession, which would later provide support.

Act 3: A Third Protagonist, "the medical sector"
Some months earlier, gynecologists had been concerned by the action brought to Court. They were afraid that women would blame their doctors for not informing them about the risks of alcohol consumption during pregnancy. Although insufficient, the professionals realised that labeling could be useful and provide information about FASD. Thus, labeling would protect and facilitate the role of the doctors by limiting their liability and responsibility.

During its national Conference in November 2003, the National Gynecologist College urged the authorities to act to introduce labeling as quickly as possible.

Though some in the industry were considering putting warning labels on alcohol containers, the Senator's amendment was rejected. Professionals reacted by issuing a press release questioning the legislators,.

Some weeks later, the National Medical Academy was asked to provide information for schools, apprenticeship colleges, companies and young people in general. Regarding labeling and the Senator's amendment, the Academy adopted the same position as the national College of Gynecologists-Obstetrics, confirming that *"information should appear on the packaging of alcoholic drinks*" with a reminder to the senators saying: *" alcoholic prenatal exposure presents a risk to the fetus which is common to all alcoholic drinks"* (beer, cider, spirit, champagne as well as wine).

Act 4: Another Ally, "the Prosecutor of Lille"
During this time and at my request, the prosecutor decided to make his contribution. In April 2004 he opened a preliminary inquiry on the basis of *"endangering the life of others, aggravated deceit on goods and involuntary wounds"*. He began interrogating of the sellers and purchasing managers of prominent alcohol companies.

It is very interesting to note that there were no complaints, even though it was only a prosecutor who decided to investigate on his own authority. This was extremely important because it suggested that society was prepared to consider a case of grave neglect, without targeting anybody in particular.

Act 5: The First Political Success: the adoption of part of "Payet's amendments"
During the summer of 2004, Senator Anne-Marie Payet organised two crucial meetings with experts on the topic of "Alcohol and Pregnancy": one within the Delegation of Women's Rights and the other within the Social Committee. A few days later, the law on public health returned for a second reading to the Senate. So, Senator Payet and I drafted five new amendments:

- regarding labeling

- prevention campaigns for FASD
- messages financed by the producers
- information for health professionals
- information in schools

After holding a hearing about these questions, the Social Committee promised to support four amendments out of five. Two days before the vote, in a very tense atmosphere, the senators friendly with the wine growers, let it be known that they would be prepared to support the Bill, if the one amendment concerning labeling was removed. Considering that labeling was not an end in itself, the Senator withdrew this Amendment. The Amendment relating to the financing of messages concerning FASD by the Industry, was also removed. Nevertheless the adoption of the other measures in July was a huge success.

Act 6: The magic effect in the media of the word "penal"
On the 3rd August, a press interview relating to the opening of the preliminary inquiry by the Prosecutor of Lille, was taken up by all the media (daily papers, radios and TV). No one anticipated the power of the word "penal". FASD moved from the status of "one among many health problems" to ***"implying a possible responsibility of political and economic important factors".***

The unpredictable media explosion that followed, took everybody by surprise! In a press conference the next day, the new Minister of Health announced his intention to label alcoholic drinks. He also announced a campaign of prevention via the pharmacists linked to the sale of pregnancy tests and the launch of a large epidemiological study. He intended to consult with the industry and the legal organisation ANPAA (Association Nationale de Prevention en Alcoologie et Addictologie) regarding the content of the warning label.

Act 7: Different reactions among the producers
The alcohol sector was split between the wine growers and the spirit producers. The spirit producers immediately announced that they accepted this labeling and reminded us that they have always promoted "no alcohol during pregnancy". This sector understood it had much to gain in terms of image.

However, this was not the case with the wine growers. They made a distinction between wine and spirits. They blamed the government for *"discouraging the consumption in the name of public health".*

The strategy had an adverse effect on customers. After a television documentary about Fetal Alcohol Spectrum Disorder, a wine grower in the South of France decided to label without waiting for it to become compulsory. He knew that in the USA labels on bottles carried compulsory warnings for pregnant women. His customers reacted positively and the press greeted his initiative favourably. However his independent act of labeling resulted in disagreements with his colleagues in the industry.

Act 8: The Perseverance of the Government
The Government and the national public insurance and the legal organisation ANPAA, lead a prevention campaign: "*Alcohol: your body remembers everything*".

In December 2004, most of the women's newspapers and magazines, ran a full page of information about the dangers of alcohol consumption when pregnant. This advert was printed on a pink background as a birth announcement, with the ingredients of a happy pregnancy: *"Nine months, eight kilos of strawberries, seven siestas a week, six mother's phone calls a day, five senses on alert, two litres of water a day, a happy event* ***and zero alcohol****".* The impact of the campaign on the country mobilised the media and local authorities.

Act 9: The last development and a beautiful victory
However, at the political level, there was still another bridge to cross. The commitment of the Ministry was not sufficient. The decision to label had to be approved by the Senate. In October 2004 Senator Payet used a parliamentary device to put her Amendment into another law relating to the

Rights of Handicapped Persons. In spite of several hostile reactions, the Amendment was adopted by 200 Members in favour and 84 against (In January there had been 228 against and 14 in favour).

To finally become law, the Amendment had to be ratified by Parliament in December. Then, one week before the hearing for the Amendment, the Committee of "Cultural and Social Affairs" of the Assembly decided to simply delete all text about FASD.

Eventually, after new consideration by opponents and emotional debates, the Amendment for labeling was adopted.

Finally in October 2006 warning labels on alcohol became compulsory and France became the first country in Europe to provide information on labels about the risks of alcohol consumption in pregnancy

Translated by Claude Riviere from the book "A sa Santé", Dr. Maurice Titran et Laure Gratias, Ed. Albin Michel 2005 and based on an interview given for the Magazine of Anpaa "Addictions" …. By Senator Anne-Marie Payet and Lawyer Benoît Titran.....

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V. GENETICS AND FASD

DR ROD DENSMORE
M.D., Adoptive Father

Heritability information gives us a deeper perspective on "Secondary Disabilities"

I am a parent of a child with Fetal Alcohol Spectrum Disorder (FASD) and a physician who cares for patients who have FASD.

Birth Mums and Birth Dads

Father's genetic contribution to their kids matter as much as Mother's genetic contributions do! Understanding both birthparents' inheritable traits can help caregivers plan preventative medicine interventions for their children. As a physician, these are the questions I'd wonder about:

Heritability of mental health problems can be high.
-Impulsivity has a heritability of over 50%
-Emotional liability is thought to have a heritability of close to 50%
-69% of the variance for expression of symptoms of *Borderline Personality Disorder* is accounted for by genetic influences [3]
-Heritability of *ADHD* may be as high as 76% [8]
-*Alcohol Dependence* has a 50-60% heritability [9]
-*Externalizing Behaviour* such as Conduct Disorder or Antisocial Personality Disorder associated with substance abuse can have a heritability of 80% [10]

Actually, in 2009 we can understand the genetics of *substance use* for men and women at a much deeper level. The National Institute on Alcohol Abuse and Alcoholism in the USA has funded the Collaborative Studies on the Genetics of Alcoholism (COGA) since 1989, with the goal of identifying the specific genes underlying this vulnerability. Alcohol use disorders have a 50-60% heritability and the genes that explain about half this heritable risk are identified thus far. If we can understand elements of this emerging field we can develop effective targeted public health preventative plans and understand how to interact more effectively with *all* kids, but especially those who have FASD.

Fathers' Roles

Animal studies have shown the offspring of alcohol exposed *fathers* to have a lowered birth weight, hyperactivity, lowered cognitive ability, reduced length, lower serum testosterone, smaller seminal vesicles, lower beta endorphin levels in the hypothalamus, more malformations, smaller eye globe size, increased incidence of microcephalus, more problems with muscle coordination and a higher likelihood of hydronephrosis. This list of findings is similar to what physicians find when they diagnose quite severely affected children with FASD. Epigenetic influences *from both birth mums and birth dads* result in a significant part of the pattern of prenatal injury we call FASD.

Gene by Environment Interaction

One gene can affect several different types of mental health issues. Different forms (called the "s" allele and the "l" allele) of the promotor part of the serotonin transporter gene **(5-HTT**) predispose to different mental health outcomes *in the presence of stressful or negative life events.* One or 2 copies of the short (s) allele increase both the incidence and severity and decrease the treatment responsiveness of depression. If you have one or 2 short alleles of this **5-HTT** polymorphism you are at increased risk of developing Depression or Post Traumatic Stress Disorder (PTSD) if you are exposed to stressful life events and if you have low levels of social support. [18] Furthermore, *various age* groups with the short allele show different adverse effects:

- increased drinking and drug use in *college students* exposed to negative life events
- increased amount of alcohol consumed and earlier start of drinking in maltreated *children*[18]

Before dozing off with this "medical-speak" consider Mary Schneider and her team's elegant work with these same gene polymorphisms in monkeys. If a monkey baby carried the short allele of this **5-HTT** gene *and if that baby's mum was exposed to alcohol during her pregnancy* the baby was much more likely to show irritability as a neonate, and increased activation of his/her HPA (Hypothalamus-Pituitary-Adrenal Cortex) stress axis when stressed compared with controls that were not exposed to alcohol or those with long ("l") alleles of the **5-HTT** *even if those* (with the long alleles) *were exposed to prenatal alcohol.* [19] In other words, the s allele conferred increased irritability to offspring of monkeys who (prenatally) consumed moderate amounts of alcohol. *This s allele is common; it is found in 25% (African) to 80% (Asian) of all of us.* So, does this one gene "cause" the irritability, difficulty feeding and hard to settle symptoms we call "regulatory disorders of infancy" often seen in FASD? ...Yes, ***in part - for some people****,* it does. Could this gene also "cause" irritability, "being set off by the least little thing", anxiety issues and other matters related to the overactivity and oversensitivity of the stress ("Fight or Flight" HPA axis)?... Yes, ***in part - for some people*****,** it could.

Genetics basics...or...why I say "*in part - for some people"*

We each have 46 chromosomes - ½ from our mum and ½ from our dad

- these chromosomes are arranged in 23 pairs
- each of us has 25,000 genes - scientists know where these genes are located
- a key question is "what turns these genes on and off?"
- most genes are inactive...women have *one* largely dormant X chromosome, a spleen cell does not usually make nerve growth factors, etc...TO A LARGE EXTENT THE QUESTION IS NOT WHETHER AN INDIVIDUAL HAS A SPECIFIC GENE---BUT IS WHETHER HE/SHE EXPRESSES THAT GENE OR NOT

The extent of genetic involvement in basic metabolic processes is astonishing. For example:

- oversensitivity to specific types of sensory input appears to have moderate genetic influence. In a study of toddler-aged twins auditory and especially tactile defensiveness had moderate heritability and was associated with anxiety and a fearful temperament. [20] This sensory profile seems to be more common in kids affected by FASD. Sensory overresponsivity (SOR) likely involves dopamine 2 receptor (D2R) binding in the straitum (caudate, putamen and globus pallidus). Genetic moduation of D2R, and dopamine transporter proteins would affect expression of SOR [21]
- when a new fact (for example 2+2=4) is learned this involves a process of glutamate release into a synapse → binding of the glutamate to an NMDA receptor → opening of the NMDA receptor which lets the secondary messenger, calcium, enter the post-synaptic cell → then calcium signals the DNA in the post-synaptic cell to synthesize proteins including more receptors! (thus increasing the

strength of transmission for the next similar glutamate release) Making new proteins when cells "learn" something new is called *long term potentiation.*[22]

- "Impulsivity" includes terms such as sensation seeking, risk-taking, novelty seeking, boredom susceptibility and unreliability-- impulsivity has been associated with risky behaviours including sexual risk taking and drug use. [23] Impulsivity has at least a 50% heritability [3] Impulsivity may relate to FASD. In a study of "sexual debut age 16 or younger" it was found genetics influenced disinhibitory traits: sensation seeking, impulsivity and non-conformity, 20-25**%** of the youth had been drinking when they had sex, and if youth showed more conduct disorder traits or drug use there was an increased chance of having *unprotected* sex and/or sex *in exchange* for drugs or money. [24]
- "Externalizing" disorders such as conduct disorder, antisocial personality disorder or substance abuse disorders appear to have a high heritability and are strongly associated with alcohol abuse [25]

- **Genes ARE NOT DESTINY**...that is why I am writing this chapter! ...kids of alcoholics have 2-4X higher risk of alcoholism but less than half become alcoholics.[9] Nevertheless there are 4 lines of evidence: adoption studies, twin studies, animal models and human genetics that all say there is a significant genetic component to alcohol use disorders. [9] If we can understand mechanisms that allow expression of potentially harmful genes we can target interventions to people at high risk.. Those who carry high risk genes *do not necessarily* develop even highly heritable diseases (e.g. even if your identical twin has Bipolar Disease you have a 30 % chance of *not* developing it)
- Re.: Addictions: you may have a lot of genetic predisposition but *if you are not exposed to a substance of abuse you will NOT develop an addiction to it .* This emphasizes the importance of *reducing access* to substances of abuse as an essential public health policy. A liquor store on every corner is ludicrous. Providing "free" drugs to addicts may reduce harm from dirty needles but will increase the extent of drug consumption and the public health burden from more drug damage (cirrhosis, alcohol-related road traffic accidents or violence when we consider alcohol [26]). Prof. Griffith Edwards, considering the link between increased availability, increased consumption and increased drinking problems in Eastern Europe in the 1990's says, " Where controls on price and access (to alcohol) already exist they should not be removed for political, ideological or trade reasons, with their public health significance ignored."[26]
- nicotine dependence has a 50-60% heritability and some genetic locations associated with nicotine addiction (a cluster of nicotine receptor genes on chromosome 15) also contribute an increased chance of alcohol use problems. Other nicotine receptor genes on chromosomes 9 and 11 are "candidate" genes that might predispose to impulsivity, cocaine dependence and nicotine dependence-- one cannot separate the effect of one factor on another i.e. the cocaine dependence might be via the impulsivity effect [27]
- not all that is *familial* is genetic e.g. choices and habits, "the way we do it in this family" plays a role in whether a risk taking behaviour will occur
- while genetics are a central factor in pathogenesis of many disorders " if someone self-administers enough of any substance (of abuse) he/she will "swamp" the effects of any genetic tendencies!"[27]

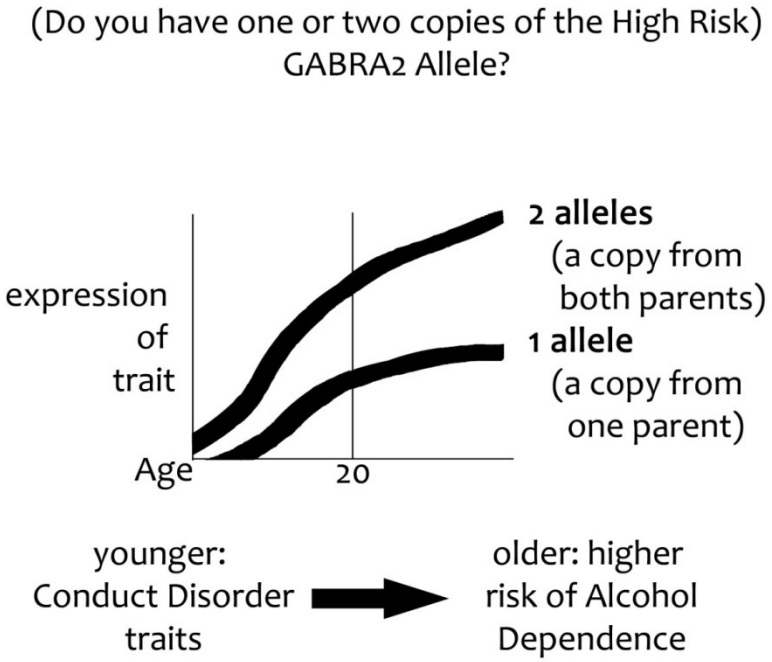
What actual genes seem to increase the risk of developing Alcohol Abuse?

-"Genes we already know predict a bit less than 50% of drug dependence so lots is unknown at this point" [27]

- Especially in complex conditions (such as personality traits, alcohol addiction, diabetes or hypertension) *many* genes influence an outcome---any *single* gene accounts for just a *small part* of an eventual outcome. Each gene may also affect some other important characteristics...and these might indirectly affect addictions (e.g. if you have the **GABRA2** gene the odds ratio of you developing alcoholism is 1.2 (an increased risk of 20%); if you have this same **GABRA2** gene the odds ratio of you *not* being married is 2.2! [27] Clearly **GABRA2** carries genetic codes that help govern reciprocity, anger, volatility, impulse control and other skills that are needed to maintain long-term relationships.
- Some genes influence enzymes that break down alcohol. For example, *Aldehyde Dehydrogenase* ***(ALDH2)*** is the gene that makes the liver enzyme that breaks down aldehyde which is a toxic breakdown product of alcohol. If aldehyde accumulates it causes flushing, nausea, lightheadedness and a fast heart rate. If you carry the gene for the inactive form of ALDH2 you will develop this flushing reaction when you drink-- it isn't "worth it"--you are at virtually no risk for alcohol

dependence if you are *homozygous* for this gene. (i.e. if you inherit the gene for the inactive form of ALDH2 from both your mum and your dad)

- Another way alcohol is broken down in the liver involves the *microsomal ethanol-oxidizing system (MEOS)*, which is located in the *smooth endoplasmic reticulum (SER)*. MEOS depends on *cytochrome P450 enzyme* systems which are also located on the SER. Chronic alcohol intake can induce a form of P450 called *CYP2E1* and that leads to an increased metabolic break down of alcohol, increased *alcohol tolerance* (which means more is needed to achieve intoxication), increased production of toxic breakdown products from alcohol or other drugs (such as Tylenol) which, together with increased reactive radicals, increase liver damage. [26] Genes govern the MEOS system but I have not seen any mention of these genes as risk factors for FASD or alcoholism in my review of medical literature.
- Little alcohol is broken down outside the liver but one spot where some metabolism occurs is the stomach. A form of the *Alcohol Dehydrogenase* (ADH) enzyme called *class IV or "sigma" ADH* is present in the stomach. Caucasian women and many Japanese people have reduced activity of this enzyme so there is less alcohol breakdown in their stomach and thus, higher blood alcohol levels. Women also have a smaller amount of body water and this also increases blood alcohol concentrations for a given intake versus men. Higher blood alcohol concentrations lead to more end organ damage for a given "dose" of alcohol. [26] Genes govern sigma ADH activity but I have not seen any information re this enzyme system in alcohol abuse literature.
- The Collaborative Study on Genetics of Alcoholism (COGA) is a quest to find variations of genes that increase the risk of alcoholism. COGA looks at family members of alcoholics in treatment. n= 14000! A single nucleotide polymorphism (SNP) is one change in one area of a gene. COGA looks at the entire genome of family members of alcoholics to see where there is increased incidence of certain SNP's ...these areas contain "candidate" genes for alcoholism:
- **GABA GENES:** the GABA A receptor is inhibitory-- it mediates anxiolysis, sedation, tolerance and dependence. As mentioned above, genes for the GABA A receptor, especially **GABRA2 increase** the risk of alcoholism. These are many SNPs on chromosome 11 (the 3' end of the gene not the 5' end) which are more common in relatives of alcoholics. If one groups SNPs from adjacent or very close areas together these "clusters" are called "halplotypes." Halplotypes are even a better way to predict areas of the genome associated with alcoholism. **GABRA2** does not influence variability of expression of drug dependence outcomes by being an area where there are amino acid sequence changes or by causing different proteins to be synthesized. **GABRA2** changes *how promotors and spicing variants work.* **GABRA2** is seen in populations who are both drug and alcohol dependent-- thus the "phenotype" (observable traits and behaviours) associated with this gene has drug and alcohol dependence, a lower age of onset of heavy drinking, a higher number of maximum drinks per episode of drinking (i.e. larger binges) and more profound withdrawal symptoms. They are also more likely to have co-morbid antisocial personality disorder, conduct disorder, major depressive disorder and "novelty seeking" traits. **GABRA2** increases the risk of alcoholism by 20% but increases the chance of not being married by 120% compared to "average" people in society. [9] Study of such extreme phenotypes might not help understand addictions in the majority...different genes might play a role in populations who drink more moderately. Below we see that expression of **GABRA2** is associated with conduct disorder symptoms in younger people and a severe pattern of alcohol abuse in older people. These tendencies are more profound for people who have more than one copy of the gene (homozygotes).
- **Comment:** Members of families affected by this type of genetic profile are at significant risk. If a family you are working with fits the profile: conduct disorder symptoms in the kids, antisocial tendencies in the adults, early-onset heavy drinking, large binges, profound withdrawal, marked "risk-taking" and comorbid depression please consider the possibility they could be dealing with GABRA2. Would you ask parents from such a family to take on more duties of special-needs interventions for their kids? Maybe ideas like "therapeutic daycare" *with transportation provided* might be more likely to succeed.



- **Dopamine 2 receptor genes (DRD2)** are not *proven* to play a role in ad€diction yet [28] but alcohol does increase dopamine release in the nucleus accumbens –hence its rewarding effect. There is reduced DRD2 receptor activity in the striatum in withdrawal. Also if you carry the A1 allele of DRD2 the chance of you learning to *avoid the negative consequences of withdrawal* are reduced! —and the *active* genes for this effect might be DRD2 A1 allele's *close neighbor* genes: **ANKK1, TTC12, NCAM1**
- **ANKK1** influences **TTC12** to change risk of heavy drinking and also these genes influence the risk of nicotine dependence dependence [28]
- **CRHR1** encodes for the corticotropin releasing hormone receptor 1 and there are 2 SNPs of this gene: rs242928 and rs1876831 that are associated with *binge drinking in adolescents* and *alcohol dependence* in adults [10]
- **hTAS2R16** gene on chromosome 7 encodes the "bitter" taste receptor**.** If this gene is expressed there is less sensitivity to bitter taste, a higher number of maximum drinks per 24 hrs and increased dependence [28]
- Low **Level of Response** to alcohol (how much can you "feel" alcohol's effect) is an important predictor of risk of alcoholism at all ages and with both sexes. There are many "candidate" genes that are associated with reduced level of response. For 13 yr olds: level of response is related to alcohol outcome but primarily through its effect on peer drinking behaviours. For 17 yr olds level of response operates *directly* and through mediators: *expectations* (what you think will happen if you drink) and *coping* (if you use alcohol to cope or have poor coping mechanisms) to predict alcohol outcome. Dr. Schuckit's level of response model predicts 78% of the variance of why a certain alcohol outcome is seen in this age group! The 17 yr old's 40 yr old parents' alcohol outcomes are not influenced by peers but level of response *directly* (and via *coping* and *expectations*) predicts half of the variance of why a certain alcohol outcome is seen. [29]
- **OPRM1** is the genetic locus for the mu opioid receptor. Mu and gamma opioid receptor stimulation by alcohol adds to positive reinforcement by dopamine release in the nucleus accumbens. Several SNPs of OPRM1 are associated with alcohol dependence. If drinkers have the *Asp*40 allele (vs. the *Asn*40 allele) Naltrexone (an opioid receptor blocker) helps avoid relapse into heavy drinking. [10] A SNP of OPRM1 called A118G (rs1799971) was seen in over half of adolescents that had early onset alcohol use problems (vs. 16% of their peers who had no alcohol use issues)—Naltrexone also helped reduce problems in this group (of adolescents) by reducing their increased sensitivity to the positive reinforcing effects of drink. [30]

- Variations in the nicotine receptor gene cluster on chromosome 15 called **CHRNA5, CHRNA3** and **CHRNA4** affect the risk of developing dependence to alcohol and nicotine. [10] We minimize the effect of nicotine at our peril for tobacco kills more people than alcohol-induced health problems do. Furthermore, nicotine decreases fetal growth. Varenicline (Chantix) is a partial nicotinic receptor agonist that may also help decrease alcohol consumption. Although I have not used this agent with pregnant mums unable to stop their smoking and drinking I wonder if in some very high risk situations it might be wise to use it given we know the very high teratogenic risk of tobacco and alcohol. This drug's information document says: "Teratogenic effects were not observed in animal studies; however, decreased fertility, decreased fetal weight, and increased auditory startle response were observed in the offspring. There are no adequate or well-controlled studies in pregnant women. [31] Pregnancy risk category "C" which means: Risk cannot be ruled out. Human studies are lacking and animal studies are either positive for fetal risk or lacking as well. However, potential benefits may justify the potential risk.

-Are there genes of resiliency that allow drug users to not progress to dependence? The National Institute on Drug Abuse (NIDA) consortium has much interest in these and research is ongoing.

Conclusions

Risk of problems from alcohol use is related to a number of **genes**-- we know about half of them thus far-- AND **choices** AND **environmental** influences. Inherited tendencies towards impulsivity, sensory sensitivities, and emotional dysregulation may affect choices about using an addicting substance. Level of response to alcohol is an important factor predicting risk of alcohol abuse at all ages and with both sexes. Different factors assume more importance for any given age group. Supportive attachments have been shown to positively influence genetic expression of parts of the HPA stress system. Genetic influences involve multiple genes; there is a small effect from each-- no 1 allele of 1 gene does it all! Traits and mental health problems, which may be risk factors for developing "Secondary Disabilities", have significant heritability.

Marc Schuckit points out, " Any *one* gene only explains **part** of the risk – and anyhow – who wants to change a gene? So identify genes that put people at high risk and make robust changes to the environment to help decrease expression of these high risk genes." Kieran O'Malley suggests there is nothing *special* about FASD—it should be treated with no more (**but also *no less***) seriousness and respect than any other significant health issue. [32] I believe Marc Schuckit's remarks about genetics and alcoholism also pertain to FASD: "We will never reach a point where we can say we can prevent alcoholism totally but the more we understand about the **risk factors**, the **genes** associated with them, and the **mechanisms** through which they work—for Depression, for Schizophrenia, for Cancer, for Heart Disease **and** for Alcoholism (and I will add for FASD)—the better we should be able to provide **early identification** and **prevention** of any of these disorders."

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November 2009

VI) ALCOHOL CONSUMPTION DURING PREGNANCY IN NEW ZEALAND: SUMMARY OF FINDINGS FROM THE FIRST NATIONAL BASELINE STUDY

Dr. Sherly Parackal, Research Fellow, Social and Community Health, School of Population Health, Faculty of Medical and Health Sciences, University of Auckland, Auckland, New Zealand.
Mathew Parackal, Lecturer, Department of Marketing, University of Otago, Dunedin, New Zealand.

FASD research is New Zealand is relatively young, primarily because health practitioners and policy makers largely under recognised this disorder. However, in recent years, recognition of FASD has increased and currently, reducing the prevalence of drinking among pregnant women and women planning a pregnancy is a key goal of the national drug policy. The Alcohol in Pregnancy study (APS), funded by the Alcohol Advisory Council and the Ministry of Health, New Zealand aimed to collect much needed information to aid in meeting the objectives of this goal of the national drug policy. 1

APS was an observational cross-sectional survey of a national sample of 1256 women, aged between 16 and 40 years. Suvey was implementd via telephone using a web-assisted telephone interviewing system (WATI). WATI is a unique cost-effective data collection system developed to produce stratified simple random samples from census area units. For APS, WATI was successful in producing a sample that was representative of the population on regional distribution, age, ethnicity, use of community service card (a surrogate for personal income), alcohol and tobacco use and pregnancy status.

Data collected by APS included 1) opinions on safety of alcohol consumption in pregnancy, 2) preference of a warning label as a source of information on the risks associated with alcohol consumption in pregnancy and 3) prevalence of alcohol consumption of women pregnant at the time of the survey and those who had a baby in the five years preceding the survey. Analysis of the data collected indicated that more than half of women surveyed were of the opinion that some alcohol was safe in pregnancy. A similar proportion gave a high rating for a warning label on alcohol containers as a source of information on risks associated with drinking in pregnancy. Just over half of currently pregnant women and those who had a baby in the last five years had drunk some alcohol in their pregnancy. The majority of women surveyed reported to have consumed some alcohol prior to realizing they were pregnant and then stopped. One in five women had binged during pregnancy, the majority having done so before they realised they were pregnant.

The pattern of alcohol consumption observed in the APS indicate the possibility of a prevalence rate of FASD similar to those established for other western countries. However, there is a dearth in studies that have established prevalence rates of FASD in New Zealand and urgent research attention is required to address this gap. Concurrently public health and policy initiatives should be implemented to reduce the prevalence of alcohol consumption among pregnant women and women planning a pregnancy in New Zealand.

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RESEARCH ABSTRACTS

PubMed, Br J Psychiatry. 2009 Oct;195(4):294-300.

1. MATERNAL TOBACCO, CANNABIS AND ALCOHOL USE DURING PREGNANCY AND RISK OF ADOLESCENT PSYCHOTIC SYMPTOMS IN OFFSPRING

Zammit S, Thomas K, Thompson A, Horwood J, Menezes P, Gunnell D, Hollis C, Wolke D, Lewis G, Harrison G.
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Abstract
Background: Adverse effects of maternal substance use during pregnancy on fetal development may increase risk of psychopathology. AIMS: To examine whether maternal use of tobacco, cannabis or alcohol during pregnancy increases risk of offspring psychotic symptoms.

Method: A longitudinal study of 6356 adolescents, age 12, who completed a semi-structured interview for psychotic symptoms in the Avon Longitudinal Study of Parents and Children (ALSPAC) birth cohort.

Results: Frequency of maternal tobacco use during pregnancy was associated with increased risk of suspected or definite psychotic symptoms (adjusted odds ratio 1.20, 95% CI 1.05-1.37, P = 0.007). Maternal alcohol use showed a non-linear association with psychotic symptoms, with this effect almost exclusively in the offspring of women drinking >21 units weekly. Maternal cannabis use was not associated with psychotic symptoms. Results for paternal smoking during pregnancy and maternal smoking post-pregnancy lend some support for a causal effect of tobacco exposure in utero on development of psychotic experiences.

Conclusions: These findings indicate that risk factors for development of non-clinical psychotic experiences may operate during early development. Future studies of how in utero exposure to tobacco affects cerebral development and function may lead to increased understanding of the pathogenesis of psychotic phenomena.

Link to the Article,
http://www.ncbi.nlm.nih.gov/pubmed/19794196?dopt=Abstract

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American Journal of Obstetrics & Gynecology, published online 21 October 2009

2. DO MULTIVITAMIN SUPPLEMENTS MODIFY THE RELATIONSHIP BETWEEN PRENATAL ALCOHOL INTAKE AND MISCARRIAGE?

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3 Division of Research, Kaiser Permanente, Oakland, CA

Objective
To determine whether multivitamin supplements modify the relationship between alcohol consumption during pregnancy and the risk of miscarriage.

Study Design
We used data from a population-based cohort study of pregnant women (n = 1061; response rate = 39%). Participants were asked about their alcohol consumption and vitamin intake during pregnancy.

Results
Among multivitamin nonusers, women who drank alcohol during their pregnancy were more likely to have a miscarriage compared with women who abstained (adjusted hazard ratio, 1.67; 95% confidence interval, 1.04–2.69). However, among multivitamin users, there was no difference in the risk of miscarriage between alcohol consumers and abstainers. Results suggest the volume of alcohol as well as the timing of multivitamin supplementation may also be important.

Conclusion
Our findings suggest that a woman of childbearing years might decrease her risk of miscarriage associated with alcohol intake by taking multivitamin supplements. However, our findings should be interpreted with caution and future research replicating these findings is necessary

Read Full Article,
http://www.ajog.org/article/S0002-9378(09)00845-X/abstract

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Nervenarzt. 2009 Oct 17. [Epub ahead of print]

3. ADDICTION ACROSS THE LIFESPAN

Mann K, Laucht M, Weyerer S.
ZI - Zentralinstitut für Seelische Gesundheit, Universität Heidelberg, Mannheim, Deutschland, sucht@zi-mannheim.de.

Abstract
Alcohol and nicotine are with us during most of our lifetime. About 4,000 children with fetal alcohol syndrome and another 20,000 children with fetal alcohol effects are born per year in Germany. Alcohol contributes to accidents and suicides especially in young people. It is particularly toxic for the developing brain.Germany is among the countries with a high consumption of alcohol and nicotine. Consequently substance-related diseases are highly prevalent. In the group of people aged 65 and older we expect a doubling of alcohol problems within the next 10 years. This will also lead to a sharp increase in alcohol-related dementias. Overall, treatment is effective especially if one considers the chronic relapsing nature of the disorder. Unfortunately, less than 10% of patients really receive specialist care. This segment needs to be expanded especially by psychiatrists and psychotherapists. Different prevention strategies are being applied but there is a reluctance to use a ban or curtail advertising and to raise taxes for a reduction in overall consumption.

Link to the Article,
http://www.ncbi.nlm.nih.gov/pubmed/19838663?dopt=Abstract

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J Am Acad Child Adolesc Psychiatry. 2009 Sep;48(9):884-93.

4. META-ANALYSIS: TREATMENT OF ATTENTION-DEFICIT/HYPERACTIVITY DISORDER IN CHILDREN WITH COMORBID TIC DISORDERS

Bloch MH, Panza KE, Landeros-Weisenberger A, Leckman JF
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Abstract

Objective:
The Food and Drug Administration currently requires the package inserts of most psychostimulant medications to list the presence of a tic disorder as a contraindication to their use. Approximately half of children with Tourette's syndrome experience comorbid attention-deficit/hyperactivity disorder

(ADHD). We sought to determine the relative efficacy of different medications in treating ADHD and tic symptoms in children with both Tourette's syndrome and ADHD.

Method:
We conducted a PubMed search to identify all double-blind, randomized, placebo-controlled trials examining the efficacy of medications in the treatment of ADHD in the children with comorbid tics. We used a random effects meta-analysis with standardized mean difference as our primary outcome to estimate the effect size of pharmaceutical agents in the treatment of ADHD symptoms and tics.

Results:
Our meta-analysis included nine studies involving 477 subjects. We assessed the efficacy of six medications-dextroamphetamine, methylphenidate, alpha-2 agonists (clonidine and guanfacine), desipramine, atomoxetine, and deprenyl. Methylphenidate, alpha-2 agonists, desipramine, and atomoxetine demonstrated efficacy in improving ADHD symptoms in children with comorbid tics. Alpha-2 agonists and atomoxetine significantly improved comorbid tic symptoms. Although there was evidence that supratherapeutic doses of dextroamphetamine worsens tics, there was no evidence that methylphenidate worsened tic severity in the short term.

Conclusions:
Methylphenidate seems to offer the greatest and most immediate improvement of ADHD symptoms and does not seem to worsen tic symptoms. Alpha-2 agonists offer the best combined improvement in both tic and ADHD symptoms. Atomoxetine and desipramine offer additional evidence-based treatments of ADHD in children with comorbid tics. Supratherapeutic doses of dextroamphetamine should be avoided.

Link to the Article,
http://www.ncbi.nlm.nih.gov/pubmed/19625978

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Alcohol. 2009 Sep;43(6):453-63.

5. BINGE ETHANOL EXPOSURE IN LATE GESTATION INDUCES ETHANOL AVERSION IN THE DAM BUT ENHANCES ETHANOL INTAKE IN THE OFFSPRING AND AFFECTS THEIR POSTNATAL LEARNING ABOUT ETHANOL

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Abstract

Previous studies show that exposure to 1 or 2g/kg of ethanol during the last days of gestation increases ethanol acceptance in infant rats. We tested whether prenatal exposure to 3g/kg, a relatively high ethanol dose, generates an aversion to ethanol in both the dam and offspring, and whether this prenatal experience affects the expression of learning derived from ethanol exposure postnatally. The answer was uncertain, because postnatal administration of a 3-g/kg ethanol dose induces an aversion to ethanol after postnatal day (PD) 10 but increases ethanol acceptance when administered during the first postnatal week. In the present study, pregnant rats received intragastric administrations of water or ethanol (3g/kg) on gestation days 17-20. On PDs 7-8 or 10-11, the offspring were administered water or ethanol (3g/kg). Intake of ethanol and water, locomotor activity in an open field, and ethanol odor preference were evaluated in the pups, whereas the mothers were evaluated in terms of ethanol intake. Results indicated an aversion to ethanol in dams that had been administered ethanol during gestation, despite a general increase in ethanol intake observed in their pups relative to controls. The prenatal ethanol exposure also potentiated the increase in ethanol intake observed after intoxication on PDs 7-8. Ethanol intoxication on PDs 10-11 reduced ethanol consumption; this ethanol aversion was still evident in infant rats exposed prenatally to ethanol despite their general increase in ethanol intake. No effects of prenatal ethanol exposure were

observed in terms of motor activity or odor preference. It is concluded that prenatal exposure to ethanol, even in a dose that induces ethanol aversion in the gestating dam, increases ethanol intake in infant rats and that this experience modulates age-related differences in subsequent postnatal learning about ethanol.

Link to the Article,
http://www.ncbi.nlm.nih.gov/pubmed/19801275?dopt=Abstract

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PubMed, Reprod Toxicol. 2009 Sep;28(2):262-9. Epub 2009 Apr 10.

6. DEVELOPMENTAL TOXICITY OF ETHANOL IN CHICK HEART IN OVO AND IN MICROMASS CULTURE CAN BE PREVENTED BY ADDITION OF VITAMIN C AND FOLIC ACID

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Centre for Integrated Systems Biology and Medicine, School of Biomedical Sciences, Queen's Medical Centre, University of Nottingham NG7 2UH, UK. mbxsm4@nottingham.ac.uk

Abstract
The teratogenic effects of ethanol include malformations of the cardiovascular system, which may be abrogated by multivitamin therapy. Chick cardiomyocytes in micromass culture were treated with ethanol alone or with supplementation with folate or vitamin C. Ethanol alone caused a loss of cell viability and differentiation (beating) whereas those cells treated in addition with vitamins were comparable to the control. Chick embryos were injected on day 3 of incubation with PBS, ethanol alone or with additional vitamin C or folic acid. On day 9 embryos were examined for viability, growth retardation and gross malformation and the hearts were processed for histology. Results showed that ethanol significantly decreased survival of embryos or caused growth retardation and gross malformation ($p<0.05$). Embryos incubated with addition of vitamin C or folic acid were comparable to the control. Data obtained in this study suggest that supplementation with vitamin C or folic acid during pregnancy may prevent defects in heart development brought about by ethanol.

Link to Article,
http://www.ncbi.nlm.nih.gov/pubmed/19473809?dopt=Abstract

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Oxford Journals, Alcohol and Alcoholism, Voulme 24, Number 6, published online on September 19, 2009

7. SEXUALLY DIMORPHIC EFFECTS OF ALCOHOL EXPOSURE DURING DEVELOPMENT ON THE PROCESSING OF SOCIAL CUES

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Abstract

Aims: The study used an animal model of fetal alcohol spectrum disorders (FASD) to investigate the impact of alcohol exposure during a period equivalent to all three trimesters in humans on social recognition memory. It was hypothesized that the effects on specific aspects of social recognition memory would be sexually dimorphic.

Methods: This study exposed rats to ethanol during both the prenatal and early postnatal periods. Two control groups included a group exposed to the administration procedures but not ethanol and a

non-treated group. At 90 days, all rats were tested repeatedly in a test of social recognition memory with a juvenile animal of the same sex. Experimental rats of both sexes were allowed to investigate an unknown juvenile for either 2, 3 or 5 min and then, after a delay of 30, 60, 120 and 180 min, were allowed to investigate the same juvenile for 5 min.

Results: Male rats investigated the juvenile for much longer than female rats. Ethanol-exposed male rats showed a deficit in recognition memory that was evident with longer delays when the initial investigation time was either 2- or 3-min long. In contrast, ethanol-exposed female rats showed a deficit in recognition memory only when the initial investigation period was of 2 min. Measurement of oxytocin receptor binding in the amygdala region indicated that ethanol exposure lowered oxytocin receptor binding in females but not males.

Conclusions: The results suggest that ethanol exposure during development caused a deficit in memory duration but not encoding in males and a deficit in encoding but not memory duration in females. The deficit in ethanol-exposed females may be related to changes in oxytocin receptors in the amygdala.

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http://alcalc.oxfordjournals.org/cgi/content/abstract/44/6/555

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Oxford Journal, Human Reproduction, published online on September 18, 2009

8. ALCOHOL BINGE DRINKING DURING PREGNANCY AND CRYPTORCHIDISM

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Abstract
Background: Recent studies have suggested gestational weeks 8–14 as a time window of particular importance to the intrauterine development of the male genitalia, and prenatal exposure to alcohol is under suspicion as a risk factor for cryptorchidism. We examined if prenatal exposure to alcohol, and especially binge drinking, during the suggested programming window is associated with an increased risk of cryptorchidism.

Methods: The authors used data on 41 268 live born singleton boys of mothers who were enrolled into the Danish National Birth Cohort in 1996–2002. During early childhood, 1598 cases of cryptorchidism were identified and 398 of these were orchiopexy verified. Maternal alcohol consumption including number and timing of binge drinking episodes was assessed in two computer-assisted telephone interviews around gestational weeks 17 and 32. Adjusted hazard ratios (HRs) of cryptorchidism were estimated by Cox regression.

Results: Average weekly alcohol consumption as well as frequency of binge drinking at any time during pregnancy was not associated with risk of cryptorchidism. Binge drinking in gestational weeks 7–15 was associated with a slightly increased risk of cryptorchidism with adjusted HRs between 1.03 and 1.66.

Conclusion: Prenatal exposure to alcohol—measured as average intake as well as frequency and timing of binge drinking—was not associated with cryptorchidism. Our findings, however, do not rule out that binge drinking during the suggested male programming window may increase the risk of cryptorchidism.

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PubMed, Dev Psychobiol. 2009 Sep 18

9. A TYPICAL FUNCTIONAL LATERALIZATION IN CHILDREN WITH FETAL ALCOHOL SYNDROME

Domellöf E, Rönnqvist L, Titran M, Esseily R, Fagard J
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Abstract
In order to explore effects of prenatal alcohol exposure on functional lateralization, item tasks measuring preferences of hand, foot, eye, and ear were administered to a sample of 23 children diagnosed with fetal alcohol syndrome (FAS) compared with typically developing (TD) children. In addition, a dichotic listening task was administered to a subsample of 11 children with FAS and a TD group of comparable age, sex and handedness. The children with FAS were characterized by increased nonright-handedness compared with TD children. No differences were evident for preferential use of foot, eye, or ear. Moreover, children with FAS displayed more right ear extinctions during dichotic listening relative to TD children, indicating a lack of right ear advantage. The results add to findings of decreased manual asymmetry and less left-lateralized speech perception in children with developmental disorders, and are further discussed in relation to the high incidence of callosal abnormalities in alcohol-exposed children. (c) 2009 Wiley Periodicals, Inc.

Link to the Article,
http://www.ncbi.nlm.nih.gov/pubmed/19768741?dopt=Abstract

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PubMed, Birth Defects Res A Clin Mol Teratol. 2009 Sep 17.

10. POLYMICROGYRIA IN FETAL ALCOHOL SYNDROME

Reinhardt K, Mohr A, Gärtner J, Spohr HL, Brockmann K
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Abstract

Background: Intrauterine exposure to alcohol may result in a distinct pattern of craniofacial abnormalities and central nervous system dysfunction, designated fetal alcohol syndrome (FAS). The spectrum of malformations of the brain associated with maternal alcohol abuse during pregnancy is much broader than the relatively uniform clinical phenotype of FAS. Among these malformations the most striking abnormalities involve the impairment of neuronal cell migration. However, polymicrogyria (PMG) has so far been reported only once in a human autopsy study of a child with FAS.

Case: A 16-year-old girl with confirmed maternal alcohol consumption during pregnancy and full phenotype of FAS presented after two generalized epileptic seizures for neurologic assessment. Cranial magnetic resonance imaging revealed bilateral PMG in the superior frontal gyrus with asymmetric distribution. History, clinical features, and genetic investigations provided no evidence for

any of the known genetic or acquired causes of PMG. Therefore, we propose that prenatal alcohol exposure is the cause of PMG in this patient rather than a mere coincidence.

Conclusion: Our observation represents only the second patient of PMG in FAS and confirms the phenotypic variability of cerebral malformations associated with maternal alcohol abuse during pregnancy. In patients with clinical features of FAS and neurologic deficits or seizures neuroimaging is recommended. Furthermore, FAS should be considered as a differential diagnosis for PMG. Birth Defects Research (Part A), 2009. (c) 2009 Wiley-Liss, Inc.

Link to Article,
http://www.ncbi.nlm.nih.gov/pubmed/19764076?dopt=Abstract

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Wiley InterScience, Alcoholism: Clinical and Experimental Research, Published Online: 9 Sep 2009

11. BOLD RESPONSE DURING SPATIAL WORKING MEMORY IN YOUTH WITH HEAVY PRENATAL ALCOHOL EXPOSURE

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From the San Diego State University/University of California (ADS, ADB, SLF), San Diego; Department of Psychiatry, University of California (SFT), San Diego; and Department of Psychology, Center for Behavioral Teratology, San Diego State University (SNM, EPR), San Diego, California.

Abstract

Background: Prenatal alcohol exposure has been consistently linked to neurocognitive deficits and structural brain abnormalities in affected individuals. Structural brain abnormalities observed in regions supporting spatial working memory (SWM) may contribute to observed deficits in visuospatial functioning in youth with fetal alcohol spectrum disorders (FASDs).

Methods: We used functional magnetic resonance imaging (fMRI) to assess the blood oxygen level dependent (BOLD) response in alcohol-exposed individuals during a SWM task. There were 22 young subjects (aged 10–18 years) with documented histories of heavy prenatal alcohol exposure (ALC, n = 10), and age- and sex-matched controls (CON, n = 12). Subjects performed a SWM task during fMRI that alternated between 2-back location matching (SWM) and simple attention (vigilance) conditions.

Results: Groups did not differ on task accuracy or reaction time to the SWM condition, although CON subjects had faster reaction times during the vigilance condition (617 millisecond vs. 684 millisecond, p = 0.03). Both groups showed similar overall patterns of activation to the SWM condition in expected regions encompassing bilateral dorsolateral prefrontal lobes and parietal areas. However, ALC subjects showed greater BOLD response to the demands of the SWM relative to the vigilance condition in frontal, insular, superior, and middle temporal, occipital, and subcortical regions. CON youth evidenced less increased brain activation to the SWM relative to the vigilance task in these areas ($p < 0.05$, clusters > 1,664 μl). These differences remained significant after including Full Scale IQ as a covariate. Similar qualitative results were obtained after subjects taking stimulant medication were excluded from the analysis.

Conclusions: In the context of equivalent performance to a SWM task, the current results suggest that widespread increases in BOLD response in youth with FASDs could either indicate decreased efficiency of relevant brain networks, or serve as a compensatory mechanism for deficiency at neural and/or cognitive levels. In context of existing fMRI evidence of heightened prefrontal activation in response to verbal working memory and inhibition demands, the present findings may indicate that frontal structures are taxed to a greater degree during cognitive demands in individuals with FASDs.

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http://www3.interscience.wiley.com/journal/122593914/abstract

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Wiley InterScience, Volume 15 Issue 3, Pages 176-192, Published Online 3rd September 2009

12. PREVALENCE AND EPIDEMIOLOGIC CHARACTERISTICS OF FASD FROM VARIOUS RESEARCH METHODS WITH AN EMPHASIS ON RECENT IN-SCHOOL STUDIES

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Funded by: National Institute on Alcohol Abuse and Alcoholism (NIAAA); Grant Number: RO1AA09440, UO1AA11685, RO1 AA015134 NIAAA pilot project (San Diego State University); Grant Number: AA014800, AA014828 NIH National Center on Minority Health and Health Disparities (NCMHD) Assessorato alla Sanita della Regione Lazio SITAC OULUS

Abstract

Researching the epidemiology and estimating the prevalence of fetal alcohol syndrome (FAS) and other fetal alcohol spectrum disorders (FASD) for mainstream populations anywhere in the world has presented a challenge to researchers. Three major approaches have been used in the past: surveillance and record review systems, clinic-based studies, and active case ascertainment methods. The literature on each of these methods is reviewed citing the strengths, weaknesses, prevalence results, and other practical considerations for each method. Previous conclusions about the prevalence of FAS and total FASD in the United States (US) population are summarized. Active approaches which provide clinical outreach, recruitment, and diagnostic services in specific populations have been demonstrated to produce the highest prevalence estimates. We then describe and review studies utilizing in-school screening and diagnosis, a special type of active case ascertainment. Selected results from a number of in-school studies in South Africa, Italy, and the US are highlighted. The particular focus of the review is on the nature of the data produced from in-school methods and the specific prevalence rates of FAS and total FASD which have emanated from them. We conclude that FAS and other FASD are more prevalent in school populations, and therefore the general population, than previously estimated. We believe that the prevalence of FAS in typical, mixed-racial, and mixed-socioeconomic populations of the US is at least 2 to 7 per 1,000. Regarding all levels of FASD, we estimate that the current prevalence of FASD in populations of younger school children may be as high as 2-5% in the US and some Western European countries. © 2009 Wiley-Liss, Inc. Dev Disabil Res Rev 2009; 15:176-192.

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Wiley InterScience, Developmental Disabilities Research Reviews
Volume 15, Issue 3, Pages 250-257 Published Online: 3rd September 2009

13. FETAL ALCOHOL SPECTRUM DISORDERS AND THE CRIMINAL JUSTICE SYSTEM

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Abstract
The life-long neurological impairments found in people with fetal alcohol spectrum disorders (FASDs), including learning disabilities, impulsivity, hyperactivity, social ineptness, and poor judgment, can increase susceptibility to victimization and involvement in the criminal justice system (CJS). Individuals with FASDs become involved in the CJS as complainants, witnesses, and accused. Their disabilities, resulting from the prenatal alcohol exposure, must be considered at all stages in the legal process. Adverse experiences, such as having a dysfunctional family background, mental health problems, and substance use disorders, are compounding factors. Experiencing physical, sexual, and emotional abuse also increases the risk that these individuals will become involved in the CJS. It is critical that everyone involved in the CJS receives education and training to understand FASD and the implications for the individual offender. A comprehensive medical-legal report, prepared by professionals experienced with FASD, can help judges and lawyers understand the complex interactions among brain damage, genetics and the environment. Corrections workers and probation officers need to comprehend the significance of FASD and how it affects the offender's abilities to understand and follow rules and probation orders. Caregivers and parents need to be involved whenever possible. Early recognition of the disabilities associated with FASDs may help reduce the over-representation of this group in the CJS. © 2009 Wiley-Liss, Inc. Dev Disabil Res Rev 2009;15:250-257.

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Wiley InterScience, Developmental Disabilities Research Reviews
Volume 15, Issue 3, Pages 167-169 Published Online: 3rd September 2009

14. FETAL ALCOHOL SPECTRUM DISORDERS: WHEN SCIENCE, MEDICINE, PUBLIC POLICY, AND LAWS COLLIDE

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Abstract

Historically, alcohol has been used for different purposes including as a part of religious observances, as a food, at times as a medicine and its well-known use as a beverage. Until relatively recently these purposes have not changed and have at times been at odds with one another, resulting in collisions among policies and practices in science, medicine, public policy and the law. One area in which this has been particularly true is that of fetal alcohol spectrum disorders (FASD) where the adverse consequences of consumed alcohol on children in the womb and after birth may have been observed since antiquity, but the actions taken based on such observations have been influenced as much by

the socio/cultural/political context of the times in which they were made as by evidence of harm. This article provides an overview of the inherent confusion when new scientific findings confront prevailing medical practice, the history involved in this confusion with respect to FASD, including public policy and legal issues that have arisen around alcohol and pregnancy, and the research and clinical challenges still being faced. Published 2009 Wiley-Liss, Inc. Dev Disabil Res Rev 2009;15:170-175.

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Wiley InterScience, Developmental Disabilities Research Reviews
Volume 15, Issue 3, Pages 193-199 Published Online: 3rd September 2009

15. PREVENTION OF FETAL ALCOHOL SPECTRUM DISORDERS

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The findings and conclusions in this report are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention.

Abstract

Alcohol use among women of childbearing age is a leading, preventable cause of birth defects and developmental disabilities in the United States. Although most women reduce their alcohol use upon pregnancy recognition, some women report drinking during pregnancy and others may continue to drink prior to realizing they are pregnant. These findings emphasize the need for effective prevention strategies for both pregnant and nonpregnant women who might be at risk for an alcohol-exposed pregnancy (AEP). This report reviews evidence supporting alcohol screening and brief intervention as an effective approach to reducing problem drinking and AEPs that can lead to fetal alcohol spectrum disorders. In addition, this article highlights a recent report of the National Task Force on Fetal Alcohol Syndrome and Fetal Alcohol Effect that describes effective interventions to reduce alcohol use and AEPs, and outlines recommendations on promoting and improving these strategies. Utilizing evidence-based alcohol screening tools and brief counseling for women at risk for an AEP and other effective population-based strategies can help achieve future alcohol-free pregnancies. © 2009 Wiley-Liss, Inc. Dev Disabil Res Rev 2009;15:193-199.

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Wiley InterScience, Developmental Disabilities Research Reviews
Volume 15, Issue 3, Pages 200-208 Published Online: 3rd September 2009

16. ANIMAL MODELS OF FETAL ALCOHOL SPECTRUM DISORDERS: IMPACT OF THE SOCIAL ENVIRONMENT

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Abstract

Animal models of fetal alcohol spectrum disorder (FASD) have been used to demonstrate the specificity of alcohol's teratogenic effects and some of the underlying changes in the central nervous system (CNS) and, more recently, to explore ways to ameliorate the effects of alcohol. The main point of this review is to highlight research findings from the animal literature which point to the impact of the social context or social behavior on the effect(s) of alcohol exposure during development, and also to point to research questions about the social environment and effects of prenatal alcohol exposure that remain to be answered. Alcohol exposure during early development alters maternal responding to the exposed pup in a variety of ways and the alteration in maternal responding could alter later stress responsivity and adult maternal and social behavior of the exposed offspring. Environmental enrichment and voluntary exercise have been shown to ameliorate some of alcohol's impact during development, but the roles of enhanced social interactions in the case of enrichment and of social housing during voluntary exercise need to be more fully delineated. Similarly, the role of social context across the lifespan, such as social housing, social experiences, and contact with siblings, needs further study. Because of findings that alcohol during development alters DNA methylation patterns and that there are alterations in the maternal care of the alcohol-exposed offspring, epigenetic effects and their relationship to social behavior in animal models of FASD are likely to become a fruitful area of research. Because of the simpler social behavior and the short lifespan of rodents, animal models of FASD can be useful in determining how the social context impacts the effects of alcohol exposure during development. © 2009 Wiley-Liss, Inc. Dev Disabil Res Rev 2009;15:200-208.

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Wiley InterScience, Developmental Disabilities Research Reviews
Volume 15, Issue 3, Pages 209-217 Published Online: 3rd September 2009

17. NEUROIMAGING AND FETAL ALCOHOL SPECTRUM DISORDERS

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Funded by: National Institute on Alcohol Abuse and Alcoholism; Grant Number: R01 AA010820, R01 AA010417, U01 AA014834, T32 AA013525

Abstract

The detrimental effects of prenatal alcohol exposure on the developing brain include structural brain anomalies as well as cognitive and behavioral deficits. Initial neuroimaging studies of fetal alcohol spectrum disorders (FASD) using magnetic resonance imaging (MRI) confirmed previous autopsy reports of overall reduction in brain volume and central nervous system (CNS) disorganization, with specific structural abnormalities of the corpus callosum, cerebellum, caudate, and hippocampus.

Advances in neuroimaging techniques have allowed detection of regional increases in cortical thickness and gray matter volume along with decreased volume and disorganization of white matter in individuals with FASD. In addition, functional imaging studies have found functional and neurochemical differences in those prenatally exposed to alcohol. Behavioral alterations noted in individuals with FASD are consistent with the findings noted in the brain imaging studies. Continued neuroimaging studies are needed to further advance understanding of the neuroteratogenic effects of alcohol. © 2009 Wiley-Liss, Inc. Dev Disabil Res Rev 2009;15:209-217.

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Wiley InterScience, Developmental Disabilities Research Reviews
Volume 15, Issue 3, Pages 218-224 Published Online: 3rd September 2009

18. NEUROCOGNITIVE PROFILE IN CHILDREN WITH FETAL ALCOHOL SPECTRUM DISORDERS

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Funded by: NIH; Grant Number: 1P20 AA017068 NIAAA

Abstract
The question of whether children with fetal alcohol spectrum disorders (FASD) exhibit a unique neurocognitive profile has received considerable attention over the past three decades. The identification of a syndrome-specific neurocognitive profile would aid in diagnosing prenatally exposed children with cognitive deficits who do not exhibit clinically discernable physical anomalies. The current review of the literature, therefore, focuses on the studies of higher-order cognitive skills in children with FASDs with a view towards delineating a pattern of cognitive functioning. Researchers have documented that children with FASDs show diminished intellectual functioning, with average IQ scores falling within the borderline to low average ranges. Slow information processing and disturbances of attention have been observed from infancy through adulthood in individuals with FASDs. Clinical and experimental reports on individuals with FASD have documented marked deficits in executive functioning, particularly in tasks that involve holding and manipulating information in working memory. Studies examining specific domains of cognitive functioning such as language, visual perception, memory and learning, social functioning, and number processing in individuals with FASDs have revealed performance decrements associated with increased task complexity. The above findings converge on the conclusion that children with FASDs have a generalized deficit in the processing and integration of information. We recommend the study of developmental trajectories of both elementary and higher-order functions in future research on FASD to elucidate the development of this cognitive profile. © 2009 Wiley-Liss, Inc. Dev Disabil Res Rev 2009;15:218-224.
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Wiley InterScience, Developmental Disabilities Research Reviews
Volume 15, Issue 3, Pages 225-234 Published Online: 3rd September 2009

19. PSYCHIATRIC CONDITIONS ASSOCIATED WITH PRENATAL ALCOHOL EXPOSURE

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The contents do not necessarily represent the positions or policies of the Centers for Disease Control and Prevention and endorsement by the Federal Government should not be assumed.
Funded by: Centers for Disease Control and Prevention; Grant Number: UDD000041

Abstract

Since the identification of fetal alcohol syndrome (FAS) over 35 years ago, mounting evidence about the impact of maternal alcohol consumption during pregnancy has prompted increased attention to the link between prenatal alcohol exposure (PAE) and a constellation of developmental disabilities that are characterized by physical, cognitive, and behavioral impairments. These disabilities include a continuum of developmental disorders known as fetal alcohol spectrum disorders (FASDs). Longitudinal studies suggest that individuals with FASDs are at a greatly increased risk for adverse long-term outcomes, including mental health problems and poor social adjustment. This review summarizes the existing literature on mental health outcomes for individuals with PAE across the lifespan, including findings in infancy and early childhood, middle childhood, and adolescence and early adulthood. Research on the psychiatric disabilities suffered by individuals with FASDs throughout development highlights the need for training of mental health professionals in the identification and the provision of specific treatments to address the unique features of this developmental disability since early identification and treatment have been demonstrated to be protective against more serious secondary disabilities. It is hoped that with greater awareness of the mental health problems experienced by individuals with FASDs, these individuals can receive appropriate and early treatment resulting in more adaptive and rewarding lives. © 2009 Wiley-Liss, Inc. Dev Disabil Res Rev 2009;15:225-234.

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Wiley InterScience, Developmental Disabilities Research Reviews
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20. FAMILY MATTERS: FETAL ALCOHOL SPECTRUM DISORDERS AND THE FAMILY

Heather Carmichael Olson [1 2] *, Rosalind Oti [2], Julie Gelo [3], Sharon Beck [3]
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Funded by: Centers for Disease Control and Prevention; Grant Number: U01 0000DD038-04

Abstract

Information about family matters is vital to developing targeted interventions, reducing placement disruption, and enhancing outcome in fetal alcohol spectrum disorders (FASD). The quality of the caregiving environment and family function are associated with long-term outcome in natural history study of individuals with FASD. This article integrates multiple information sources to better understand the role of family factors in the outcome of individuals with FASD, and how the family is affected by raising a child with this lifelong condition. A brief description of the useful informal literature is brought together with a review of the surprisingly limited body of systematic research findings on FASD and caregiver/family function, and new data describing children with FASD and characteristics of their caregivers. Directions for future data-gathering and intervention development emerge from combining what is already known with an exploration of what can be learned from a highly targeted review of family-related data in the wide-ranging, general literature on developmental disabilities, and use of a proposed conceptual framework that joins a developmental systems perspective with a family systems approach. © 2009 Wiley-Liss, Inc. Dev Disabil Res Rev 2009;15:235-249.

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http://www3.interscience.wiley.com/journal/122588282/abstract

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Wiley InterScience, Developmental Disabilities Research Reviews
Volume 15, Issue 3, Pages 258-267 Published Online: 3rd September 2009

21. INTERVENTION FOR INDIVIDUALS WITH FETAL ALCOHOL SPECTRUM DISORDERS: TREATMENT APPROACHES AND CASE MANAGEMENT

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The contents do not necessarily represent the positions or policies of the Centers for Disease Control and Prevention and endorsement by the Federal Government should not be assumed.
Funded by: Centers for Disease Control and Prevention; Grant Number: U84/CCU925033-01

Abstract

Exposure to alcohol in utero is considered to be the leading cause of developmental disabilities of known etiology. The most severe consequence of such exposure, fetal alcohol syndrome (FAS), is characterized by a distinct constellation of characteristic facial anomalies, growth retardation, and central nervous system (CNS) dysfunction. Some individuals with prenatal alcohol exposure (PAE) do not meet the full criteria for FAS, but instead are diagnosed with partial FAS, alcohol related neurodevelopmental disorder (ARND), or alcohol related birth defects (ARBD). The entire continuum of effects from PAE is increasingly being referred to under the umbrella term of fetal alcohol spectrum disorders (FASDs). An extensive body of research has documented major cognitive, behavioral, adaptive, social, and emotional impairments among individuals with FASDs. Although FAS was identified in the U.S. over 35 years ago, the development, evaluation, and dissemination of evidence-based interventions for individuals with FASDs have lagged behind significantly. Encouragingly, however, in recent years there has been a marked increase in efforts to design and test interventions to remediate the impairments associated with prenatal alcohol exposure. This article will review treatment needs and considerations for individuals with FASDs and their families, current empirically tested treatment approaches, case management issues, and suggestions for future directions in research on the treatment of FASDs. © 2009 Wiley-Liss, Inc. Dev Disabil Res Rev 2009;15:258-267.

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http://www3.interscience.wiley.com/journal/122588279/abstract

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Journal of Neurochemistry. 110(3):779-790, August 2009

22. ETHANOL INHIBITS L1 CELL ADHESION MOLECULE TYROSINE PHOSPHORYLATION AND DEPHOSPHORYLATION AND ACTIVATION OF PP60SRC

Yeaney, Natalie K; He, Min; Tang, Ningfeng; Malouf, Alfred T.; O'Riordan, Mary Ann; Lemmon, Vance; Bearer, Cynthia F.

Abstract
Fetal alcohol syndrome is a leading cause of mental retardation. The neuropathology found in patients with fetal alcohol syndrome overlaps with those with mutations in the gene for cell adhesion molecule (L1). We have previously shown that L1-mediated neurite outgrowth and L1 activation of extracellular receptor kinases 1/2 are inhibited at low concentrations of ethanol. One possible mechanism for this effect is through disruption of a tyrosine-based sorting signal, Y(1176)RSLE, on the cytoplasmic domain of L1. Our goal was to determine if ethanol inhibited the sorting signal or its phosphorylation state. Using cerebellar granule neurons and dorsal root ganglion neurons, we found that ethanol had no effect on L1 distribution to the growth cone or its ability to be expressed on the cell surface as determined by confocal microscopy. In cerebellar granule neurons, clustering of L1 resulted in increased dephosphorylation of Y(1176), increased L1 tyrosine phosphorylation, and an increase in the activation of pp60src as measured by immunoblot. All changes were inhibited by 25 mM ethanol. Using PP2 to inhibit pp60src activation resulted in inhibition of increases in L1 tyrosine and extracellular receptor kinases 1/2 phosphorylation, and Y(1176) dephosphorylation. We conclude that ethanol disrupts L1 trafficking/signaling following its expression on the surface of the growth cone, and prior to its activation of pp60src.

Read Full Article
http://pt.wkhealth.com/pt/re/jneu/abstract.00005064-200908000-00001.htm

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Journal of Neurochemistry. 110(3):976-989, August 2009.

23. PARADOXICAL EFFECT OF ETHANOL ON POTASSIUM CHANNEL CURRENTS AND CELL SURVIVAL IN CEREBELLAR GRANULE NEURONS.

Lefebvre, Thomas; Gonzalez, Bruno J.; Vaudry, David ; Desrues, Laurence ; Falluel-Morel, Antony; Aubert, Nicolas; Fournier, Alain; Tonon, Marie-Christine; Vaudry, Hubert; Castel, Helene

Abstract
Transient exposure to ethanol (EtOH) results in a massive neurodegeneration in the developing brain leading to behavioral and cognitive deficits observed in fetal alcohol syndrome. There is now compelling evidence that K+ channels play an important role in the control of programmed cell death. The aim of the present work was to investigate the involvement of K+ channels in the EtOH-induced cerebellar granule cell death and/or survival. At low and high concentrations, EtOH evoked membrane depolarization and hyperpolarization, respectively. Bath perfusion of EtOH (10 mM) depressed the IA (transient K+ current) potassium current whereas EtOH (400 mM) provoked a marked potentiation of the specific IK (delayed rectifier K+ current) current. Pipette dialysis with GTP[gamma]S or GDP[beta]S did not modify the effects of EtOH (400 mM) on both membrane potential and IK current. In contrast, the reversible depolarization and slowly recovering inhibition of IA induced by EtOH (10 mM) became irreversible in the presence of GTP[gamma]S. EtOH (400 mM) induced prodeath responses whereas EtOH (10 mM) and K+ channel blockers promoted cell survival. Altogether, these results indicate that in cerebellar granule cells, EtOH mediates a dual effect on K+ currents partly involved in the control of granule cell death.

Read Full Article
http://pt.wkhealth.com/pt/re/jneu/abstract.00005064-200908000-00020.htm

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Alcoholism Clinical and Experimental Research, Volume 33, Issue 8, Pages 1346-1354, August 2009

24. ETHANOL ATTENUATES SPATIAL MEMORY DEFICITS AND INCREASES MGLU1A RECEPTOR EXPRESSION IN THE HIPPOCAMPUS OF RATS EXPOSED TO PRENATAL STRESS

Vincent Van Waes, Mihaela Enache, Annarita Zuena, Jérôme Mairesse, Ferdinando Nicoletti, Elisabeth Vinner, Michel Lhermitte, Stefania Maccari, and Muriel Darnaudéry
From the NEUROSTRESS EA 4347, "Université Lille Nord de France" (VVW, ME, AZ, JM, SM, MD), 59655 Villeneuve d'Ascq, France; Department of Human Physio & Pharmacology, Sapienza University of Roma (AZ, JM, FN, SM), 00185 Roma, Italy; Neuromed (I.R.C.C.S.), 86079 Venafro (FN), Italy; and Laboratory of Toxicology & Genopathy UPRES EA 2679, "Université Lille Nord de France" (EV, ML) and Calmette Hospital, 59037 Lille, France.

Abstract

Background: Although it is generally believed that chronic ethanol consumption impairs learning and memory, results obtained in experimental animals are not univocal, and there are conditions in which ethanol paradoxically improves cognitive functions. In the present work, we investigated the effects of prenatal stress and of chronic ethanol exposure during adulthood on spatial memory in rats.

Methods: Rats were subjected to a prenatal stress delivered as 3 daily 45-minute sections of restraint stress to the mothers during the last 10 days of pregnancy (PRS rats). After 7 months of ethanol exposure (ethanol 10%, oral intake), memory performances were evaluated in a spatial discrimination test in control and PRS male rats. Then, the oxidative damages and the expression of metabotropic glutamate (mGlu) receptors were assessed in their hippocampus.

Results: Chronic ethanol exposure resulted in a reduced performance in a spatial recognition task in control animals. Unexpectedly, however, the same treatment attenuated spatial memory deficits in rats that had been subjected to prenatal stress. This paradigm of ethanol administration did not produce detectable signs of oxidative damage in the hippocampus in either unstressed or PRS rats. Interestingly, ethanol intake resulted in differential effects in the expression of mGlu receptor subtypes implicated in mechanisms of learning and memory. In control rats, ethanol intake reduced mGlu2/3 and mGlu5 receptor levels in the hippocampus; in PRS rats, which exhibited a constitutive reduction in the levels of these mGlu receptor subtypes, ethanol increased the expression of mGlu1a receptors but did not change the expression of mGlu2/3 or mGlu5 receptors.
Conclusion: Our findings support the idea that stress-related events occurring before birth have long-lasting effects on brain function and behavior, and suggest that the impact of ethanol on cognition is not only dose- and duration-dependent, but also critically influenced by early life experiences.
Read Full Article,
http://www3.interscience.wiley.com/journal/122371195/abstract

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Hum Mol Genet. 2009 Nov 15;18(22):4457-66. Epub 2009 Aug 17.

25. A NON-SYNONYMOUS VARIANT IN ADH1B IS STRONGLY ASSOCIATED WITH PRENATAL ALCOHOL USE IN A EUROPEAN SAMPLE OF PREGNANT WOMEN.

Zuccolo L, Fitz-Simon N, Gray R, Ring SM, Sayal K, Smith GD, Lewis SJ.
Department of Social Medicine, University of Bristol, Bristol, UK. l.zuccolo@bristol.ac.uk

Abstract

Pregnant women are advised to abstain from alcohol despite insufficient evidence on the fetal consequences of moderate prenatal alcohol use. Mendelian randomization could help distinguish causal effects from artifacts due to residual confounding and measurement errors; however, polymorphisms reliably associated with alcohol phenotypes are needed. We aimed to test whether alcohol dehydrogenase (ADH) gene variants were associated with alcohol use before and during pregnancy. Ten variants in four ADH genes were genotyped in women from South-West England. Phenotypes of interest were quantity and patterns of alcohol consumption before and during pregnancy, including quitting alcohol following pregnancy recognition. We tested single-locus associations between genotypes and phenotypes with regression models. We used Bayesian models (multi-locus) to take account of linkage disequilibrium and reanalyzed the data with further exclusions following two conservative definitions of 'white ethnicity' based on the woman's reported parental ethnicity or a set of ancestry-informative genetic markers. Single-locus analyses on 7410 women of white/European background showed strong associations for rs1229984 (ADH1B). Rare allele carriers consumed less alcohol before pregnancy [odds ratio (OR) = 0.69; 95% confidence interval (CI): 0.56-0.86, P = 0.001], were less likely to have 'binged' during pregnancy (OR = 0.55, 95% CI: 0.38-0.78, P = 0.0009), and more likely to have abstained in the first trimester of gestation (adjusted OR = 1.42, 95% CI: 1.12-1.80, P = 0.004). Multi-locus models confirmed these results. Sensitivity analyses did not suggest the presence of residual population stratification. We confirmed the established association of rs1229984 with reduced alcohol consumption over the life-course, contributing new evidence of an effect before and during pregnancy.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2766294/?tool=pubmed

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Arch Pediatr. 2009 Oct;16(10):1364-73. Epub 2009 Aug 15.

26. ALCOHOL AND PREGNANCY - FRANCE

[Article in French]
Seror E, Chapelon E, Bué M, Garnier-Lengliné H, Lebeaux-Legras C, Loudenot A, Lejeune C.
Service d'hématologie pédiatrique, hôpital Saint-Louis, Assistance publique-Hôpitaux de Paris, 1 avenue Claude-Vellefaux, Paris cedex 10, France. seror.je@laposte.net

Abstract
Alcohol consumption during pregnancy is a major cause of mental retardation in Western countries. Fetal alcohol syndrome (FAS) is mainly characterized by pre- and postnatal stunted growth, neurocognitive disorders, and facial dysmorphism. It compromises the intellectual and behavioral prognosis of the child. Prevention tools exist, through better information of health professionals, for optimal care of high-risk women before, during, and after pregnancy, which would decrease the incidence of SAF in the future.

Link to the Article,
http://www.ncbi.nlm.nih.gov/pubmed/19683904?dopt=Abstract

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NCBI – PubMed - 2009 Aug 14;4(8):e6643

27. PHOSPHODIESTERASE INHIBITION INCREASES CREB PHOSPHORYLATION AND RESTORES ORIENTATION SELECTIVITY IN A MODEL OF FETAL ALCOHOL SPECTRUM DISORDERS

Krahe TE, Wang W, Medina AE.
Department of Anatomy and Neurobiology, Virginia Commonwealth University Medical Center, Richmond, Virginia, USA

Abstract
Background: Fetal alcohol spectrum disorders (FASD) are the leading cause of mental retardation in the western world and children with FASD present altered somatosensory, auditory and visual processing. There is growing evidence that some of these sensory processing problems may be related to altered cortical maps caused by impaired developmental neuronal plasticity.

Methodology/Principal Findings: Here we show that the primary visual cortex of ferrets exposed to alcohol during the third trimester equivalent of human gestation have decreased CREB phosphorylation and poor orientation selectivity revealed by western blotting, optical imaging of intrinsic signals and single-unit extracellular recording techniques. Treating animals several days after the period of alcohol exposure with a phosphodiesterase type 1 inhibitor (Vinpocetine) increased CREB phosphorylation and restored orientation selectivity columns and neuronal orientation tuning.

Conclusions/Significance: These findings suggest that CREB function is important for the maturation of orientation selectivity and that plasticity enhancement by vinpocetine may play a role in the treatment of sensory problems in FASD.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2721629/

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Alcoholism: Clinical and Experimental Research; Volume 33 Issue 11, Pages 1945 - 1955
Published Online: 10 Aug 2009

28. ETHANOL INHIBITS MUSCARINIC RECEPTOR-INDUCED AXONAL GROWTH IN RAT HIPPOCAMPAL NEURONS

Kathryn L. VanDeMark*, Marina Guizzetti*, Gennaro Giordano, and Lucio G. Costa
From the Department of Environmental and Occupational Health Sciences (KLV, MG, GG, LGC), University of Washington, Seattle, Washington; and Department of Human Anatomy, Pharmacology and Forensic Sciences (LGC), University of Parma Medical School, Parma, Italy.
Correspondence to Reprint requests: Marina Guizzetti, Department of Environmental and Occupational Health Sciences, University of Washington, 4225 Roosevelt Way NE, Suite 100, Seattle, WA 98105; Fax: 206-685-4696; E-mail: marinag@u.washington.edu
*These authors contributed equally to this work.

Abstract
Background: In utero alcohol exposure can lead to fetal alcohol spectrum (FAS) disorders characterized by cognitive and behavioral deficits. In vivo and in vitro studies have shown that ethanol alters neuronal development. One mechanism through which ethanol has been shown to exert its effects is the perturbation of activated signaling cascades. The cholinergic agonist carbachol has been shown to induce axonal outgrowth through intracellular calcium mobilization, protein kinase C (PKC) activation, and ERK1/2 phosphorylation. This study investigated the effect of ethanol on the differentiation of rat hippocampal pyramidal neurons induced by carbachol as a possible mechanism involved in the developmental neurotoxicity of ethanol.

Methods: Prenatal rat hippocampal pyramidal neurons were treated with ethanol (50 to 75 mM) in the presence or absence of carbachol for 24 hours. Neurite outgrowth was assessed spectrophotometrically; axonal length was measured in neurons fixed and immunolabeled with the neuron-specific βIII tubulin antibody; cytotoxicity was analyzed using the thiazolyl blue tetrazolium bromide assay. The effect of ethanol on carbachol-stimulated intracellular calcium mobilization was assessed utilizing the fluorescent calcium probe, Fluo-3AM. The PepTag® assay for nonradioactive detection of PKC from Promega was used to measure PKC activity, and ERK1/2 activation was determined by densitometric analysis of Western blots probed for phospo-ERK1/2.

Results: Ethanol treatment (50 to 75 mM) caused an inhibition of carbachol-induced axonal growth, without affecting neuronal viability. Neuron treatment for 15 minutes with ethanol did not inhibit the

carbachol-stimulated rise in intracellular calcium, while inhibiting PKC activity at the highest tested concentration and ERK1/2 phosphorylation at both the concentrations used in this study. On the other hand, neuron treatment for 24 hours with ethanol significantly inhibited carbachol-induced increase in intracellular calcium.

Conclusions: Ethanol inhibited carbachol-induced neurite outgrowth by inhibiting PKC and ERK1/2 activation. These effects may be, in part, responsible for some of the cognitive deficits associated with in utero alcohol exposure.

Read Full Article,
http://www3.interscience.wiley.com/journal/122542545/abstract

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Alcoholism Clinical and Experimental Research, Volume 33, Number 7, July 2009 , pp. 1238-1245(8)

29. ETHANOL TERATOGENESIS IN FIVE INBRED STRAINS OF MICE

Downing, Chris; Balderrama-Durbin, Christina; Broncucia, Hali; Gilliam, David; Johnson, Thomas E.

Abstract:
Background: Previous studies have demonstrated individual differences in susceptibility to the detrimental effects of prenatal ethanol exposure. Many factors, including genetic differences, have been shown to play a role in susceptibility and resistance, but few studies have investigated the range of genetic variation in rodent models.

Methods: We examined ethanol teratogenesis in 5 inbred strains of mice: C57BL/6J (B6), Inbred Short-Sleep, C3H/Ibg, A/Ibg, and 129S6/SvEvTac (129). Pregnant dams were intubated with either 5.8 g/kg ethanol (E) or an isocaloric amount of maltose-dextrin (MD) on day 9 of pregnancy. Dams were sacrificed on day 18 and fetuses were weighed, sexed, and examined for gross morphological malformations. Every other fetus within a litter was then either placed in Bouin's fixative for subsequent soft-tissue analyses or eviscerated and placed in ethanol for subsequent skeletal analyses.

Results: B6 mice exposed to ethanol in utero had fetal weight deficits and digit, kidney, brain ventricle, and vertebral malformations. In contrast, 129 mice showed no teratogenesis. The remaining strains showed varying degrees of teratogenesis.

Conclusions: Differences among inbred strains demonstrate genetic variation in the teratogenic effects of ethanol. Identifying susceptible and resistant strains allows future studies to elucidate the genetic architecture underlying prenatal alcohol phenotypes.
Read Full Article,
http://www.ingentaconnect.com/content/bsc/acer/2009/00000033/00000007/art00018

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Can J Clin Pharmacol. 2009 Summer;16(2):e370-80. Epub 2009 Jul 26

30. THE RELATION BETWEEN THEORY OF MIND AND EXECUTIVE FUNCTIONS IN CHILDREN WITH FETAL ALCOHOL SPECTRUM DISORDERS

Rasmussen C, Wyper K, Talwar V.
University of Alberta, Edmonton, Canada. carmen@ualberta.ca

Abstract
Background: Children with Fetal Alcohol Spectrum Disorders (FASD) are faced with a range of physical, cognitive, behavioral, and/or learning deficits, as well as poor executive functioning and social skills. Theory of mind (ToM) is the ability to understand that one's own perspective may differ

from the perspective of another individual. ToM develops around age 4 and is correlated with performance on executive functioning tasks.

Objective: The goals of this study were to examine ToM performance in young children with FASD, how age was related to ToM performance, and whether ToM abilities were related to underlying executive function difficulties.

Method: Fifty-three children (aged 4 to 8 years) participated: 25 children with FASD and 28 control children. All children were tested on measures of ToM, executive functioning, and receptive vocabulary.

Results: More children in the FASD group (44%) failed one or both ToM measures than in the control group (25%). Older children with FASD performed worse on ToM than younger children, but this was not the case for the control group. For the FASD group, ToM performance was correlated with a measure of inhibition, but for the control group, ToM was correlated with visual-spatial working memory.

Conclusions: Children with FASD have difficulty on ToM tasks, and this difficulty may be related to underlying deficits in inhibition.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/19638654

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Best Pract Res Clin Obstet Gynaecol. 2009 Jul 24.

31. NEONATAL MANAGEMENT AND LONG-TERM SEQUELAE

Halliday HL.
Perinatal Medicine, Royal Maternity Hospital, and Department of Child Health, Queen's University Belfast, Belfast, Northern Ireland.

Abstract
Intrauterine or fetal growth restriction is best defined by using customised birth weight percentiles based upon the growth potential for an individual infant. Growth restriction in utero may be classified as asymmetric or symmetric depending upon the duration of the process. Asymmetric growth restriction is caused by placental insufficiency, maternal hypertensive conditions, long-standing maternal diabetes, smoking, living at altitude or multiple gestation. Symmetric growth restriction may be due to congenital infections, chromosomal or other abnormalities, fetal alcohol syndrome, low socioeconomic status or be constitutional. The underlying cause of growth restriction often predicts the potential adverse effects on the foetus and newborn and later effects in childhood and adulthood. With placental insufficiency, there may be chronic or acute on chronic fetal hypoxia with birth asphyxia and hypothermia, neonatal hypoglycaemia, polycythaemia and coagulopathy. Management is directed at prevention or early treatment of these conditions. In contrast, symmetrically growth-restricted infants should be examined carefully to look for congenital infections and malformations that may need specific interventions. Infants with constitutional short stature generally do not need any specific management. Feeding of growth-restricted infants is important to overcome deficiencies incurred in utero. Most infants show catch-up growth although about 10% do not. Those with excessive catch-up growth may be at greatest risk of developing insulin resistance in adulthood leading to diabetes, obesity and heart disease. The so-called fetal origins of disease may actually have a postnatal onset related more to excessive weight gain in infancy. There is still controversy over the indications for growth hormone treatment in growth-restricted infants who remain of short stature in early childhood. Intrauterine growth restriction is also associated with a five- to seven-fold increased risk of cerebral palsy probably due to chronic placental insufficiency.

Link to the Article,
http://www.ncbi.nlm.nih.gov/pubmed/19632899?dopt=Abstract

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Unbound Medline - Alcohol Clin Exp Res 2009 Jul 23.

32. MICROSTRUCTURAL CORPUS CALLOSUM ANOMALIES IN CHILDREN WITH PRENATAL ALCOHOL EXPOSURE: AN EXTENSION OF PREVIOUS DIFFUSION TENSOR IMAGING FINDINGS

Wozniak JR, Muetzel RL, Mueller BA, McGee CL, Freerks MA, Ward EE, Nelson ML, Chang PN, Lim KO

Abstract

Background: Several studies have now shown corpus callosum abnormalities using diffusion tensor imaging (DTI) in children with fetal alcohol spectrum disorders (FASD) in comparison with nonexposed controls. The data suggest that posterior regions of the callosum may be disproportionately affected. The current study builds on previous efforts, including our own work, and moves beyond midline corpus callosum to probe major inter-hemispheric white matter pathways with an improved DTI tractographic method. This study also expands on our prior work by evaluating a larger sample and by incorporating children with a broader range of clinical effects including full-criteria fetal alcohol syndrome (FAS).

Methods: Participants included 33 children with FASD (8 FAS, 23 partial FAS, 2 static encephalopathy) and 19 nonexposed controls between the ages of 10 and 17 years. Participants underwent DTI scans and intelligence testing. Groups (FASD vs. controls) were compared on fractional anisotropy (FA) and mean diffusivity (MD) in 6 white matter tracts projected through the corpus callosum. Exploratory analyses were also conducted examining the relationships between DTI measures in the corpus callosum and measures of intellectual functioning and facial dysmorphology.

Results: In comparison with the control group, the FASD group had significantly lower FA in 3 posterior tracts of the corpus callosum: the posterior mid-body, the isthmus, and the splenium. A trend-level finding also suggested lower FA in the genu. Measures of white matter integrity and cognition were correlated and suggest some regional specificity, in that only posterior regions of the corpus callosum were associated with visual-perceptual skills. Correlations between measures of facial dysmorphology and posterior regions of the corpus callosum were nonsignificant.

Conclusions: Consistent with previous DTI studies, these results suggest that microstructural posterior corpus callosum abnormalities are present in children with prenatal alcohol exposure and cognitive impairment. These abnormalities are clinically relevant because they are associated with cognitive deficits and appear to provide evidence of abnormalities associated with prenatal alcohol exposure independent of dysmorphic features. As such, they may yield important diagnostic and prognostic information not provided by the traditional facial characteristics.

Read Full Article
http://www.unboundmedicine.com/medline/ebm/record/19645729/abstract/Microstructural_Corpus_Callosum_Anomalies_in_Children_With_Prenatal_Alcohol_Exposure:_An_Extension_of_Previous_Diffusion_Tensor_Imaging_Findings_

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ScienceDirect - Available online 21 June 2009.

33. A DUAL-FOCUS MOTIVATIONAL INTERVENTION TO REDUCE THE RISK OF ALCOHOL-EXPOSED PREGNANCY

Mary M. Velasquez[1], Karen S. Ingersoll[b], Mark B. Sobell[c], R. Louise Floyd[d], Linda Carter Sobell[c] and Kirk von Sternberg[1]
1University of Texas at Austin

bUniversity of Virginia
cNova Southeastern University
dCenters for Disease Control and Prevention

Abstract
Project CHOICES developed an integrated behavioral intervention for prevention of prenatal alcohol exposure in women at high risk for alcohol-exposed pregnancies. Settings included primary care, university-hospital based obstetrical/gynecology practices, an urban jail, substance abuse treatment settings, and a media-recruited sample in three large cities. The intervention was based on motivational interviewing and targeted both adoption of effective contraception and reduction of alcohol use. Treatment included 4 manual-guided sessions delivered by mental health clinicians and 1 contraceptive counselling session delivered by a family planning clinician. This paper describes the rationale for treatment; the use of motivational interviewing and the transtheoretical model for a dual-focused approach to behavior change; the development of the Project CHOICES intervention; development of the study protocol and treatment manual; and selection, training, supervision, and monitoring of study counselors. Implications for future applications of the intervention are discussed.

Read Full Article
Go to this website - http://dx.doi.org
Enter this code - doi:10.1016/j.cbpra.2009.02.004

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Alcohol Clin Exp Res. 2009 Oct;33(10):1656-70. Epub 2009 Jul 15

34. SOCIAL COGNITIVE AND EMOTION PROCESSING ABILITIES OF CHILDREN WITH FETAL ALCOHOL SPECTRUM DISORDERS: A COMPARISON WITH ATTENTION DEFICIT HYPERACTIVITY DISORDER

Greenbaum RL, Stevens SA, Nash K, Koren G, Rovet J.

Children's Mental Health Team, Surrey Place Centre, Toronto, Ontario, Canada.

Abstract

Background: Although children with Fetal Alcohol Spectrum Disorders (FASDs) are at high risk of attention deficit hyperactivity disorder (ADHD), direct comparisons show distinct cognitive phenotypes in the 2 diagnoses. However, these groups have not been directly compared for social problems or social cognition, nor has social cognition been directly examined in FASDs.

Objectives: To compare FASDs and ADHD groups on social cognition tasks and determine whether deficient social cognition and emotion processing predict behavioral problems and social skills.

Methods: Studied were 33 children with FASDs, 30 with ADHD, and 34 normal controls (NC). All received tasks of social cognition and emotion processing. Parents and teachers rated children on measures of completed questionnaires assessing child's behavioral problems and social skills using the Child Behavior Checklist, Teacher Report Form, and Social Skills Rating Scale. Children received 3 subtests from the Saltzman-Benaiah and Lalonde (2007) Theory of Mind Task as a measure of social cognition and 4 subtests from the Minnesota Test of Affective Processing (Lai et al., 1991) to assess emotion processing.

Results: Parents and teachers reported more behavior problems and poorer social skills in children in FASD and ADHD than NC groups. FASDs demonstrated significantly weaker social cognition and facial emotion processing ability than ADHD and NC groups. Regression analyses identified social cognition as a significant predictor of behavior problems and emotion processing as a significant predictor of social skills.

Conclusions: Children with FASDs show a distinct behavioral profile from children with ADHD. Difficulties in social cognition and emotion processing in children with FASDs may contribute to their high incidence of social behavioral problems.

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http://www.ncbi.nlm.nih.gov/pubmed/19624575

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Unbound Medline - Alcohol Clin Exp Res 2009 Jul 1

35. MAGNETIC RESONANCE IMAGING OUTCOMES FROM A COMPREHENSIVE MAGNETIC RESONANCE STUDY OF CHILDREN WITH FETAL ALCOHOL SPECTRUM DISORDERS

Astley SJ, Aylward EH, Olson HC, Kerns K, Brooks A, Coggins TE, Davies J, Dorn S, Gendler B, Jirikowic T, Kraegel P, Maravilla K, Richards T

Abstract

Background: Magnetic resonance (MR) technology offers noninvasive methods for in vivo assessment of neuroabnormalities.

Methods: A comprehensive neuropsychological/psychiatric battery, coupled with MR imaging, (MRI), MR spectroscopy (MRS), and functional MRI (fMRI) assessments, were administered to children with fetal alcohol spectrum disorders (FASD) to determine if global and/or focal abnormalities could be identified, and distinguish diagnostic subclassifications across the spectrum. The 4 study groups included: (i) fetal alcohol syndrome (FAS)/partial FAS (PFAS); (ii) static encephalopathy/alcohol exposed (SE/AE); (iii) neurobehavioral disorder/alcohol exposed (ND/AE) as diagnosed with the FASD 4-Digit Code; and (iv) healthy peers with no prenatal alcohol exposure. Presented here are the MRI assessments that were used to compare the sizes of brain regions between the 4 groups. The neuropsychological/behavioral, MRS, and fMRI outcomes are reported separately.

Results: Progressing across the 4 study groups from Controls to ND/AE to SE/AE to FAS/PFAS, the mean absolute size of the total brain, frontal lobe, caudate, putamen, hippocampus, cerebellar vermis, and corpus callosum length decreased incrementally and significantly. The FAS/PFAS group (the only group with the 4-Digit FAS facial phenotype) had disproportionately smaller frontal lobes relative to all other groups. The FAS/PFAS and SE/AE groups [the 2 groups with the most severe central nervous system (CNS) dysfunction] had disproportionately smaller caudate regions relative to the ND/AE and Control groups. The prevalence of subjects in the FAS/PFAS, SE/AE, and ND/AE groups that had 1 or more brain regions, 2 or more SDs below the mean size observed in the Control group was 78, 58, and 43%, respectively. Significant correlations were observed between size of brain regions and level of prenatal alcohol exposure, magnitude of FAS facial phenotype, and level of CNS dysfunction.

Conclusions: Magnetic resonance imaging provided further validation that ND/AE, SE/AE, and FAS/PFAS as defined by the FASD 4-Digit Code are 3 clinically distinct and increasingly more affected diagnostic subclassifications under the umbrella of FASD. Neurostructural abnormalities are present across the spectrum. MRI could importantly augment diagnosis of conditions under the umbrella of FASD, once population-based norms for structural development of the human brain are established.

Read Full Article,
http://www.unboundmedicine.com/medline/ebm/record/19572986/abstract

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QSpace at Queen's University, June 2009

36. THE PREVALENCE OF EPILEPSY AND SEIZURES IN SUBJECTS WITH FETAL ALCOHOL SPECTRUM DISORDERS

Stephanie Helen Bell

Abstract

FASD is the umbrella term that describes the range of adverse developmental outcomes that occur in offspring as a consequence of maternal drinking during pregnancy. FASD has been associated with a large number of co-morbidities, including neurological disorders such as epilepsy. Epilepsy occurs in 0.6% of the population in Canada. The aim of this study was to evaluate the prevalence of epilepsy or seizure disorders in people who have been diagnosed with Fetal Alcohol Syndrome (FAS), partial Fetal Alcohol Syndrome (pFAS) or Alcohol Related Neurodevelopmental Disorder (ARND).

Methods:
A retrospective chart review was conducted on all active charts (N=1063) at St. Michael's Hospital (Toronto) and Glenrose Rehabilitation Hospital (Edmonton) FASD clinics. A total of 425 subjects between the ages of 2 to 49 were included in the analysis. The relationship between FASD diagnosis and other risk factors for co-occurrence of epilepsy and seizures (e.g. extent of exposure to alcohol and other drugs, type of birth, maternal history, and trauma) in subjects with FASD was also examined. Chi-square tests and multivariate multinomial logistic regression were used.

Results:
Twenty-five (5.9%) individuals with FASD had a confirmed diagnosis of epilepsy, and 50 (11.8%) had at least one documented seizure episode, yielding an overall prevalence of 17.7% with a history of seizures in this population. Those with epilepsy or seizures were two times ii i (Odds Ratio=2.27, 95% Confidence Interval=1.14-4.51, $p<0.05$) more likely to have an unnatural birth and those with epilepsy were three times (OR=3.41, 95% CI 1.11-10.5, $p<0.05$) more likely to have had an unnatural type of birth (breech, caesarean, forceps or vacuum) than those subjects with no history of seizures. None of the other risk factors examined were associated with a greater prevalence of epilepsy or seizures in subjects with FASD. These results indicate a remarkably high prevalence of epilepsy/seizures in the FASD population of two specialized FASD clinics compared with the general population.

Read Full Article
https://qspace.library.queensu.ca/dspace/bitstream/1974/1998/1/Bell_Stephanie_H_200906_MSc.pdf
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Journal of Neurochemistry, Volume 109, Number 5, June 2009 , pp. 1311-1323(13)

37. PRENATAL ETHANOL EXPOSURE PERSISTENTLY IMPAIRS NMDA RECEPTOR-DEPENDENT ACTIVATION OF EXTRACELLULAR SIGNAL-REGULATED KINASE IN THE MOUSE DENTATE GYRUS

Samudio-Ruiz, Sabrina L.; Allan, Andrea M.; Valenzuela, Carlos Fernando; Perrone-Bizzozero, Nora I.; Caldwell, Kevin K.

Abstract:
The dentate gyrus (DG) is the central input region to the hippocampus and is known to play an important role in learning and memory. Previous studies have shown that prenatal alcohol is associated with hippocampal-dependent learning deficits and a decreased ability to elicit long-term potentiation (LTP) in the DG in adult animals. Given that activation of the extracellular signal-regulated kinase 1/2 (ERK1/2) signaling cascade by NMDA receptors is required for various forms of learning and memory, as well as LTP, in hippocampal regions, including the DG, we hypothesized that fetal alcohol-exposed adult animals would have deficits in hippocampal NMDA receptor-dependent ERK1/2 activation. We used immunoblotting and immunohistochemistry techniques to detect NMDA-stimulated ERK1/2 activation in acute hippocampal slices prepared from adult fetal

alcohol-exposed mice. We present the first evidence linking prenatal alcohol exposure to deficits in NMDA receptor-dependent ERK1/2 activation specifically in the DG of adult offspring. This deficit may account for the LTP deficits previously observed in the DG, as well as the life-long cognitive deficits, associated with prenatal alcohol exposure.

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http://www.ingentaconnect.com/content/bsc/jnc/2009/00000109/00000005/art00011

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ScienceDirect, NeuroScience and Biobehavioral Reviews

38. PRENATAL ALCOHOL EXPOSURE: FETAL PROGRAMMING AND LATER LIFE VULNERABILITY TO STRESS, DEPRESSION AND ANXIETY DISORDERS

Kim G.C. Hellemansa[a b], Joanna H. Sliwowska[a], Pamela Verma[a] and Joanne Weinberg[a], ,
aDepartment of Cellular and Physiological Sciences, University of British Columbia, 2350 Health Sciences Mall, Vancouver, British Columbia, Canada V6T 1Z3
bDepartment of Psychology, Carleton University, 1125 Colonel By Drive, Ottawa, Ontario, Canada K1S 5B6

Abstract
Children with fetal alcohol spectrum disorder (FASD) exhibit cognitive, neuropsychological and behavioral problems, and numerous secondary disabilities including depression and anxiety disorders. Dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis is common in depression/anxiety, reflected primarily in increased HPA tone or activity. Prenatal alcohol exposure (PAE) increases HPA tone and results in HPA dysregulation throughout life, paralleling many of the HPA changes in depression/anxiety. We review data demonstrating altered HPA function and increased depression/anxiety in FASD. In the context of the stress-diathesis model, we discuss the hypothesis that fetal programming of the HPA axis by PAE alters neuroadaptive mechanisms that mediate the stress response, thus sensitizing the organism to stressors encountered later in life, and mediating, at least partly, the increased vulnerability to depression/anxiety disorders. Furthermore, we present evidence demonstrating sex-specific alterations in both hormonal and behavioral responsiveness to tasks measuring depressive- and anxiety-like behaviors in PAE offspring. Overall, the research suggests that the stress-diathesis model provides a powerful approach for elucidating mechanisms underlying the increased vulnerability to mental illness among individuals with FASD, and developing appropriate treatments for these individuals. Dr. Seymour Levine's seminal work on the long-term consequences of early life experiences formed a framework for the development of the research described in this review.

Read Full Article
Go to this website - http://dx.doi.org
Enter this code - doi:10.1016/j.neubiorev.2009.06.004

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Alcohol, Volume 43, Issue 4, Pages 333-339, June 2009

39. PROCEEDINGS OF THE 2008 ANNUAL MEETING OF THE FETAL ALCOHOL SPECTRUM DISORDERS STUDY GROUP

Jennifer D. Thomas[a], Feng C. Zhou[b], Cynthia J.M. Kane[c]

Abstract
The annual meeting of the Fetal Alcohol Spectrum Disorders Study Group (FASDSG) was held on June 28, 2008 in Washington DC, as a satellite to the Research Society on Alcoholism meeting. The FASDSG membership includes clinical, basic, and social scientists who meet to discuss recent advances and issues in FASD research. The main theme of the meeting was “Factors that Influence

Brain and Behavioral Development: Implications for Prevention and Intervention." Two keynote speakers, Dr. Stephen Suomi and Dr. Carl Keen addressed how early environment and nutrition may influence outcome after prenatal alcohol exposure. The final keynote speaker, Kathy Mitchell, addressed issues regarding the relationship between scientists and the families with children with FASD. Members of the FASDSG provided updates on new findings through brief (FASt) data reports and national agency representatives provided updates of activities and funding priorities. Presentations were also made by recipients of the Student Research Merit award and Rosett award.

Read Full Article,
http://www.alcoholjournal.org/article/PIIS0741832909000603/fulltext

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Alcohol Clin Exp Res. 2009 Jun;33(6):1075-88. Epub 2009 Mar 23.

40. EFFECTS OF PRENATAL ETHANOL EXPOSURE ON HYPOTHALAMIC-PITUITARY-ADRENAL FUNCTION ACROSS THE ESTROUS CYCLE

Ni Lan, Fiona Yamashita, Alison G. Halpert, Joanna H. Sliwowska, Victor Viau, and Joanne Weinberg
From the Department of Cellular and Physiological Sciences, University of British Columbia (NL, FY, AGH, JHS, VV, JW), Vancouver, British Columbia, Canada; and Department of Anatomy, College of Basic Medical Sciences, China Medical University (NL), Shenyang, Liaoning, China.

Abstract
Background: Rats prenatally exposed to ethanol (E) typically show increased hypothalamic-pituitary-adrenal (HPA) responses to stressors in adulthood. Importantly, prenatal ethanol may differentially alter stress responsiveness in male and female offspring, suggesting a role for the gonadal hormones in mediating the effects of ethanol on HPA activity. We investigated the role of ethanol-induced changes in hypothalamic-pituitary-gonadal (HPG) activity in the differential HPA regulation observed in E compared to control females across the estrous cycle.

Methods: Peripheral hormones and changes in central neuropeptide mRNA levels were measured across the estrous cycle in adult female offspring from E, pair-fed (PF) and ad libitum-fed control (C) dams.

Results: Ethanol females showed normal estrous cyclicity (vaginal smears) but delayed sexual maturation (vaginal opening). Both HPG and HPA activity were differentially altered in E (and in some cases, PF) compared to control females as a function of estrous cycle stage. In relation to HPG activity, E and PF females had higher basal and stress estradiol (E2) levels in proestrus compared to other phases of the cycle, and decreased GnRH mRNA levels compared to C females in diestrus. Further, E females had greater variation in LH than PF and C females across the cycle, and in proestrus, only E females showed a significant LH increase following stress. In relation to HPA activity, both basal and stress CORT levels and overall ACTH levels were greater in E than in C females in proestrus. Furthermore, AVP mRNA levels were increased overall in E compared to PF and C females.

Conclusions: These data demonstrate ethanol-induced changes in both HPG and HPA activity that are estrous phase-specific, and support the possibility that changes in HPA activity in E females may reflect differential sensitivity to ovarian steroids. E females appear to have an increased HPA sensitivity to E2, and a possible shift toward AVP regulation of HPA activity. That PF were similar to E females on some measures suggests that nutritional effects of diet or food restriction played a role in mediating at least some of the changes observed.

Read Full Article,
http://www3.interscience.wiley.com/journal/122270981/abstract

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C J Clin Pharmacol Vol 16 (2) Summer 2009:e370-e375; June 24, 2009

41. ETHYLGLUCURONIDE AND ETHYLSULFATE IN MECONIUM TO ASSESS GESTATIONAL ETHANOL EXPOSURE: PRELIMINARY RESULTS IN TWO MEDITERRANEAN COHORTS

Original Research

Simona Pichini, Luca Morini, Emilia Marchei, Ilaria Palmi, Maria Concetta Rotolo, Federica Vagnarelli, Oscar Garcia-Algar, Oriol Vall, Piergiorgio Zuccaro

Abstract

Background: In recent years, fatty acid ethyl esters (FAEEs) in meconium emerged as reliable, direct biological markers for establishing gestational ethanol exposure. Among the minor nonoxidative products of ethanol metabolism, there are ethyl glucuronide (EtG) and ethyl sulfate (EtS).

Objectives: The aim of the study was to analyse meconium specimens from two different Mediterranean cohorts to check for the presence of EtG and EtS, and to investigate the eventual correlation between meconium FAEEs and these two metabolites and their possible application as direct biomarkers of gestational ethanol exposure.

Methods: FAEEs, EtG and EtS were quantified by liquid chromatography tandem mass spectrometry in meconium samples obtained from the Neonatal Intensive Care Unit of Arcispedale Santa Maria Nuova, Reggio Emilia, Italy (N= 96) and from the Pediatric Service of the Hospital del Mar in Barcelona, Spain (N=81).

Results: EtG was present in more than 80% meconium samples while EtS only in 50% specimens Although the samples from Spain and Italy originated from similar socio-demographic cohort, EtG values in the Barcelona samples (median value: 101.5 ng/g) were statistically higher than those from Reggio Emilia ones (median value: 15.6 ng/g). In the Barcelona cohort, EtG values could differentiate between samples with FAEEs below and those equal or above 2 nmol/g - the cut-off used to differentiate heavy maternal ethanol consumption during pregnancy from occasional or no use.

Conclusion: For the first time the presence of EtG and EtS in meconium has been proven, with EtG concentration likely to discriminate heavy maternal ethanol consumption during pregnancy disclosed by FAEEs concentration in this matrix. Further investigations are needed to verify the use of these two ethanol metabolites as alternative biomarkers of chronic in utero exposure to ethanol.

Read Full Article,
http://www.cjcp.ca/pubmed.php?articleId=205

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The Journal of Immunology, 2009, 182, 7803 -7808

42. FETAL EXPOSURE TO ETHANOL HAS LONG-TERM EFFECTS ON THE SEVERITY OF INFLUENZA VIRUS INFECTIONS

Jodi McGill, David K. Meyerholz, Michelle Edsen-Moore, Betty Young, Ruth A. Coleman, Annette J. Schlueter, Thomas J. Waldschmidt, Robert T. Cook and Kevin L. Legge

Abstract

Alcohol use by pregnant women is a significant public health issue despite well-described risks to the fetus including physical and intellectual growth retardation and malformations. Although clinical studies are limited, they suggest that in utero alcohol exposure also results in significant immune deficiencies in naive neonates. However, little is known about fetal alcohol exposure (FAE) effects on adult infections. Therefore, to determine the long-term effects of FAE on disease susceptibility and the adult immune system, we infected FAE adult mice with influenza virus. In this study, we demonstrate that mice exposed to ethanol during gestation and nursing exhibit enhanced disease

severity as well as increased and sustained pulmonary viral titers following influenza virus infection. Secondary exposure to alcohol as an adult further exacerbates these effects. Moreover, we demonstrate that FAE mice have impaired adaptive immune responses, including decreased numbers of virus-specific pulmonary CD8 T cells, a decreased size and frequency of pulmonary B cell foci, and reduced production of influenza-specific Ab following influenza infection. Together, our results suggest that FAE induces significant and long-term defects in immunity and susceptibility to influenza virus infection and that FAE individuals could be at increased risk for severe and fatal respiratory infections.

Read Full Article
http://www.jimmunol.org/cgi/reprint/182/12/7803?maxtoshow=&HITS=10&hits=10&RESULTFORMAT=1&title=FETAL+EXPOSURE+TO+ETHANOL&andorexacttitle=and&andorexacttitleabs=and&andorexactfulltext=and&searchid=1&FIRSTINDEX=0&sortspec=relevance&resourcetype=HWCIT

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Unbound Medline - Alcohol Clin Exp Res 2009 Jun 10.

43. PRENATAL ALCOHOL EXPOSURE AND INTERHEMISPHERIC TRANSFER OF TACTILE INFORMATION: DETROIT AND CAPE TOWN FINDINGS

Dodge NC, Jacobson JL, Molteno CD, Meintjes EM, Bangalore S, Diwadkar V, Hoyme EH, Robinson LK, Khaole N, Avison MJ, Jacobson SW

Abstract
Background: Previous research has demonstrated that heavy prenatal alcohol exposure affects the size and shape of the corpus callosum (CC) and compromises interhemispheric transfer of information. The aim of this study was to confirm the previous reports of poorer performance on a finger localization test (FLT) of interhemispheric transfer in a cohort of heavily exposed children and to extend these findings to a cohort of moderately exposed young adults.

Methods: In Study 1, the FLT was administered to 40 heavily exposed and 23 nonexposed children from the Cape Coloured community of Cape Town, South Africa, who were evaluated for fetal alcohol syndrome (FAS) dysmorphology and growth. Anatomical images of the CC were obtained using structural MRI on a subset of these children. In Study 2, the FLT was administered to a cohort of 85 moderate-to-heavily exposed young adults participating in a 19-year follow-up assessment of the Detroit Prenatal Alcohol Exposure cohort, whose alcohol exposure had been ascertained prospectively during gestation.

Results: In Study 1, children with FAS showed more transfer-related errors than controls after adjustment for confounding, and increased transfer-related errors were associated with volume reductions in the isthmus and splenium of the CC. In Study 2, transfer-related errors were associated with quantity of alcohol consumed per occasion during pregnancy. More errors were made if the mother reported binge drinking (>/=5 standard drinks) during pregnancy than if she drank regularly (M >/= 1 drink/day) without binge drinking.

Conclusions: These findings confirm a previous report of impaired interhemispheric transfer of tactile information in children heavily exposed to alcohol in utero and extend these findings to show that these deficits are also seen in more moderately exposed individuals, particularly those exposed to binge-like pregnancy drinking.

Read Full Article
http://www.unboundmedicine.com/medline/ebm/record/19519722/abstract/Prenatal_Alcohol_Exposure_and_Interhemispheric_Transfer_of_Tactile_Information:_Detroit_and_Cape_Town_Findings

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Alcoholism: Clinical and Experimental Research
Volume 33 Issue 9, Published Online: 10 Jun 2009

44. EFFECT OF ALCOHOL CONSUMPTION ON CPG METHYLATION IN THE DIFFERENTIALLY METHYLATED REGIONS OF H19 AND IG-DMR IN MALE GAMETES—IMPLICATIONS FOR FETAL ALCOHOL SPECTRUM DISORDERS

Lillian A. Ouko, Katpaham Shantikumar, Jaysen Knezovich, Philip Haycock, Desmond J. Schnugh, and Michèle Ramsay
From the Division of Human Genetics, National Health Laboratory Service and School of Pathology, University of the Witwatersrand, Johannesburg, South Africa.

Abstract
Background: Exposure to alcohol in utero is the main attributable cause of fetal alcohol spectrum disorders (FASD) which in its most severe form is characterized by irreversible behavioral and cognitive disability. Paternal preconception drinking is not considered to be a significant risk factor, even though animal studies have demonstrated that chronic paternal alcohol consumption has a detrimental effect on the physical and mental development of offspring even in the absence of in utero alcohol exposure. It has been documented that alcohol can reduce the levels and activity of DNA methyltransferases resulting in DNA hypomethylation and that reduced methyltransferase activity can cause activation of normally silenced genes. The aim of this study was to establish a link between alcohol use in men and hypomethylation of paternally imprinted loci in sperm DNA in genomic regions critical for embryonic development, thus providing a mechanism for paternal effects in the aetiology of FASD.

Methods: Sperm DNA from male volunteers was bisulfite treated and the methylation patterns of 2 differentially methylated regions (DMRs), H19 and IG-DMR, analyzed following sequencing of individual clones. The methylation patterns were correlated with the alcohol consumption levels of the volunteer males.

Results: There was a pattern of increased demethylation with alcohol consumption at the 2 imprinted loci with a significant difference observed at the IG-DMR between the nondrinking and heavy alcohol consuming groups. Greater inter-individual variation in average methylation was observed at the H19 DMR and individual clones were more extensively demethylated than those of the IG-DMR. CpG site #4 in the IG-DMR was preferentially demethylated among all individuals and along with the H19 DMR CpG site #7 located within the CTCF binding site 6 showed significant demethylation in the alcohol consuming groups compared with the control group.

Conclusion: This study demonstrates a correlation between chronic alcohol use and demethylation of normally hypermethylated imprinted regions in sperm DNA. We hypothesize that, should these epigenetic changes in imprinted genes be transmitted through fertilization, they would alter the critical gene expression dosages required for normal prenatal development resulting in offspring with features of FASD.

Read Full Article,
http://www3.interscience.wiley.com/journal/122455044/abstract

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BMC Public Health. 2009 Jun 5;9:175.

45. PREGNANT WOMEN'S ATTITUDES TOWARDS ALCOHOL CONSUMPTION

Raymond N, Beer C, Glazebrook C, Sayal K.
Division of Psychiatry, School of Community Health Sciences, University of Nottingham, Nottingham, UK. mzyznr@nottingham.ac.uk

Abstract

Background: There is uncertainty as to whether there is a safe threshold for drinking alcohol during pregnancy. We explored pregnant women's attitudes towards drinking alcohol in pregnancy and their attitudes towards sources of information about drinking in pregnancy following recent changes in UK government guidance.

Methods: A qualitative study involving individual, semi-structured interviews with 20 pregnant women recruited from community organisations in the UK. Interview transcripts were analysed qualitatively using thematic analysis.

Results: Most women found information and advice about safe levels of drinking in pregnancy confusing and lacking in evidence and detail. Although most women considered that there were risks involved with drinking in pregnancy and these perceptions influenced their behaviour, only six women reported abstinence. Women reported being influenced by advice from family and friends and their experiences of previous pregnancies. Many had received no individual advice from general practitioners or midwives relating to drinking during pregnancy.

Conclusion: Pregnant women wished to take responsibility for their own health and make choices based on informed advice. In order to do so, they require clear and consistent advice about safe levels of drinking from policy makers and health professionals. This is an important issue as women might drink socially during their pregnancy.

Read Full Article,
http://www.biomedcentral.com/1471-2458/9/175

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BMC Pediatrics, Published 25th May 2009

46. SYSTEMATIC REVIEW OF INTERVENTIONS FOR CHILDREN WITH FETAL ALCOHOL SPECTRUM DISORDERS

Elizabeth Peadon[1,2,3] , Biarta Rhys-Jones [1] , Carol Bower[4] and Elizabeth J Elliott[1,2,3]
1 Discipline of Paediatrics and Child Health, University of Sydney, Australia
2 Australian Paediatric Surveillance Unit, Sydney, Australia
3 The Children's Hospital at Westmead, Sydney, Australia
4 Telethon Institute for Child Health Research, Centre for Child Health Research, University of Western Australia, Perth, Australia

Abstract
Background: Children with Fetal Alcohol Spectrum Disorders (FASD) may have significant neurobehavioural problems persisting into adulthood. Early diagnosis may decrease the risk of adverse life outcomes. However, little is known about effective interventions for children with FASD. Our aim is to conduct a systematic review of the literature to identify and evaluate the evidence for pharmacological and non-pharmacological interventions for children with FASD.

Methods: We did an electronic search of the Cochrane Library, MEDLINE, EMBASE, PsychINFO, CINAHL and ERIC for clinical studies (Randomized controlled trials (RCT), quasi RCT, controlled trials and pre- and post-intervention studies) which evaluated pharmacological, behavioural, speech therapy, occupational therapy, physiotherapy, psychosocial and educational interventions and early intervention programs. Participants were aged under 18 years with a diagnosis of a FASD. Selection of studies for inclusion and assessment of study quality was undertaken independently by two reviewers. Meta-analysis was not possible due to diversity in the interventions and outcome measures.

Results: Twelve studies met the inclusion criteria. Methodological weaknesses were common, including small sample sizes; inadequate study design and short term follow up. Pharmacological interventions, evaluated in two studies (both RCT) showed some benefit from stimulant medications. Educational and learning strategies (three RCT) were evaluated in seven studies. There was some

evidence to suggest that virtual reality training, cognitive control therapy, language and literacy therapy, mathematics intervention and rehearsal training for memory may be beneficial strategies. Three studies evaluating social communication and behavioural strategies (two RCT) suggested that social skills training may improve social skills and behaviour at home and Attention Process Training may improve attention.

Conclusion: There is limited good quality evidence for specific interventions for managing FASD, however seven randomized controlled trials that address specific functional deficits of children with FASD are underway or recently completed.

Read Full Article,
http://www.biomedcentral.com/1471-2431/9/35/abstract

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Unbound Medline - Birth Defects Res A Clin Mol Teratol 2009 May 18

47. THE EFFECTS OF PRENATAL ALCOHOL EXPOSURE ON THE MORPHOLOGICAL CHARACTERISTICS OF SPINAL MOTONEURONS

David P, Subramaniam K

Abstract
Background: Clinical studies and research in animals have established that alcohol consumption during pregnancy produces irreversible developmental anomalies. Deficits in fine motor performance are often noted in infants diagnosed with fetal alcohol syndrome. However, the effects of alcohol on the spinal motoneurons have not been examined. In this study, the morphometric alterations in spinal motoneurons were assessed as a result of prenatal alcohol exposure.

Methods: Pregnant Sprague Dawley rats were administered with 1.0 ml of 20% ethyl alcohol per 100 gm body weight via intraperitoneal injections, and unexposed rats served as controls. Rats were perfused through the left cardiac ventricle and a complete laminectomy was performed. Spinal cord sections from the L4-5 segments were cut serially and stained for cresyl fast violet. Sections were also subjected to TUNEL assay for detection of apoptosis. Observations were made between 1 and 4 weeks after birth.

Results: Morphologic characteristics of motoneurons in the alcohol-exposed group of rats were altered. Counts and measurements revealed significant reduction in number and size of alcohol-exposed spinal motoneurons at all time points studied.

Conclusions: Prenatal exposure to alcohol showed cytotoxic effects whereby it adversely affected both motoneuron growth and differentiation in utero. Birth Defects Research (Part A) 2009. (c) 2009 Wiley-Liss, Inc.

Read full Article,
http://www.unboundmedicine.com/medline/ebm/record/19452514/abstract/The_effects_of_prenatal_alcohol_exposure_on_the_morphological_characteristics_of_spinal_motoneurons_

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ScienceDirect - Addictive Behaviors Volume 34, Issue 9, September 2009, Pages 714-716

48. A QUICK DRINKING SCREEN FOR IDENTIFYING WOMEN AT RISK FOR AN ALCOHOL-EXPOSED PREGNANCY

Mariam Duma, Linda Carter Sobell[a], Mark B. Sobell[a], Nicholas Heinecke[a], Andrew Voluse[a] and Kenneth Johnson[b]
aCenter for Psychological Studies, Nova Southeastern University, Ft. Lauderdale, Florida, USA

bCollege of Osteopathic Medicine, Health Professions Division, Nova Southeastern University, Ft. Lauderdale, Florida, USA

Abstract
Two previous studies comparing the Quick Drinking Screen (QDS) with the Timeline Followback (TLFB) found that these two instruments yielded similar reports of alcohol use for clinical and nonclinical populations of problem drinkers. The current study evaluated the correspondence between these two drinking measures with women at risk of an Alcohol-Exposed Pregnancy (AEP). Participants were 355 women who voluntarily participated in a research study during 2005 through 2007 designed to prevent AEPs. All women were screened by phone for eligibility using the QDS and approximately 2 weeks later completed a 3-month TLFB by mail. Results of this study, analyzed in 2008, paralleled previous studies showing that the QDS and the TLFB, two very different drinking measures, collected similar aggregate drinking data for women who drink heavily and are at risk of an AEP. Correspondence between the two drinking measures met acceptable levels of reliability. The present study found that the QDS has demonstrated efficacy for screening women whose level of alcohol use puts them at risk for an AEP. Although the QDS does not yield detailed drinking information, it could be used when it is not possible or necessary to gather daily drinking data.

Read Full Article
Go to this website - http://dx.doi.org
Enter this code - doi:10.1016/j.addbeh.2009.04.001

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PubMed, Funct Neurol. 2009 Apr-Jun;24(2):77-81.

49. WOMEN, ALCOHOL AND THE ENVIRONMENT: AN UPDATE AND PERSPECTIVES IN NEUROSCIENCE

Mancinelli R, Vitali M, Ceccanti M.
National Center for Chemical Substances, Istituto Superiore di Sanità, Rome, Italy.

Abstract
This paper highlights gender peculiarities in the neuroscience of alcohol effects and draws attention to emerging problems due to simultaneous exposure to alcohol and environmental factors. All the available gender studies on alcohol show greater severity of alcohol-related damage, including brain damage, in females compared with males. The differences are due to physiological peculiarities that make women more vulnerable to the effects of alcohol. Today the trend to start consuming alcohol at a younger age, together with the growing number of women drinking excessively, is increasing the alcohol-related risks to women's health and justifying the need for better, gender-based studies of alcohol use and abuse. A further aspect to consider in this context is the risk of the occurrence of foetal alcohol spectrum disorders and foetal alcohol syndrome in the offspring of women who drink during pregnancy. Several lines of evidence indicate that prenatal ethanol exposure can influence cell proliferation and differentiation in the central nervous system, causing severe neurotoxicity and permanent birth defects.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pubmed/19775534?dopt=Abstract

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Pediatrics Vol. 123 No. 3 March 2009

50. CHARACTERISTICS OF CHILDREN WHOSE SIBLINGS HAVE FETAL ALCOHOL SYNDROME OR INCOMPLETE FETAL ALCOHOL SYNDROME

Valborg L. Kvigne, MBA[a], Gary R. Leonardson, PhD[b], Joseph Borzelleca, MD, MPH[c], Martha Neff-Smith, PhD, MPH, RN, CS, FAAN[d] and Thomas K. Welty, MD, MPH[a]
a Aberdeen Area Indian Health Service, Aberdeen, South Dakota

b Mountain Plains Research, Dillon, Montana
c Department of Obstetrics and Gynecology, Virginia Commonwealth University, Richmond, Virginia
d Global Consultants, Gordonsville, Virginia

Abstract
Objective. To describe the clinical features of American Indian children born just before and just after a sibling with fetal alcohol syndrome or incomplete fetal alcohol syndrome.

Methods. Two retrospective case-control studies were conducted of Northern Plains American Indian children with fetal alcohol syndrome or incomplete fetal alcohol syndrome identified from 1981 to 1993 by using International Classification of Diseases, Ninth Revision, Clinical Modification code 760.71.

Results. Compared with the controls, the 39 siblings born just before children with fetal alcohol syndrome (study 1) and 30 siblings born just before children with incomplete fetal alcohol syndrome (study 2) had more facial dysmorphology (23.1% and 16.7%, respectively), growth delay (38.5% and 10.0%), and central nervous system impairment (48.7% and 33.3%). The 20 siblings born just after children with fetal alcohol syndrome (study 1) and 22 siblings born just after children with incomplete fetal alcohol syndrome (study 2) had more facial dysmorphology (20.0% and 9.1%, respectively), growth delay (45.0% and 22.7%), and central nervous system impairment (50.0% and 31.8%) than the control siblings.

Conclusions. The "before" siblings had characteristics of fetal alcohol syndrome that could have predicted that the next child was at risk for fetal alcohol syndrome. The "after" siblings had better outcomes than the previous siblings with fetal alcohol syndrome, a finding that was associated with a decrease in maternal alcohol consumption during the after-sibling pregnancy.

Link to the article
http://pediatrics.aappublications.org/cgi/content/abstract/123/3/e526

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Wiley InterScience, Human Brain Mapping
Volume 30 Issue 10, Pages 3265 – 3274, Published Online: 10 Mar 2009

51. VOXELWISE AND SKELETON-BASED REGION OF INTEREST ANALYSIS OF FETAL ALCOHOL SYNDROME AND FETAL ALCOHOL SPECTRUM DISORDERS IN YOUNG ADULTS

Longchuan Li [1], Claire D. Coles [2] *, Mary Ellen Lynch [2], Xiaoping Hu [1]
1Biomedical Imaging Technology Center, Department of Biomedical Engineering, Emory University/Georgia Institute of Technology, Atlanta, Georgia
2Department of Psychiatry and Behavioral Sciences, Emory University School of Medicine, Atlanta, Georgia

Abstract
Though fetal alcohol syndrome (FAS) and fetal alcohol spectrum disorders are among the most common developmental disorders, their understanding is incomplete. Diffusion tensor imaging (DTI), which is sensitive to microstructural organization in white matter, may provide a relevant measure in this population demonstrating incompletely characterized white matter pathology. In this study, tract-based spatial statistics (TBSS) routine and a skeleton-based region of interest analyses were employed to detect differences in DTI-derived metrics between young adults who were alcohol exposed and an unexposed control group. Participants include 28 with dysmorphic features associated with FAS, 29 who were prenatally exposed but do not show physical effects, and 25 with the same low socioeconomic status but unexposed. The TBSS analysis revealed a statistically significant decrease in fractional anisotropy at the isthmus of the corpus callosum and its connected callosal fibers in dysmorphic individuals relative to controls (clusterwise $P_{FWE} < 0.05$). This finding was consistent with that of the follow-up skeleton-based region of interest analysis ($F(2,79) = 3.256$, p

= 0.044). In addition, the patterns in axial and radial diffusivity changes suggest that demyelination may be associated with the degraded white matter integrity observed in the dysmorphic group. Hum Brain Mapp, 2009. © 2009 Wiley-Liss, Inc.

Read Full Article,
http://www3.interscience.wiley.com/journal/122246128/abstract

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Wiley InterScience, Human Brain Mapping; Volume 30 Issue 10, Pages 3200 - 3208
Published Online: 4 Mar 2009

52. ALTERED FRONTAL-PARIETAL FUNCTIONING DURING VERBAL WORKING MEMORY IN CHILDREN AND ADOLESCENTS WITH HEAVY PRENATAL ALCOHOL EXPOSURE

Elizabeth D. O'Hare [1 2] *, Lisa H. Lu [1 3], Suzanne M. Houston [1], Susan Y. Bookheimer [4], Sarah N. Mattson [5], Mary J. O'Connor [4], Elizabeth R. Sowell [1]
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Abstract
This study evaluated the neural basis of verbal working memory (WM) function in a group of 20 children and adolescents with fetal alcohol spectrum disorders (FASDs) and 20 typically developing comparison participants using functional magnetic resonance imaging (fMRI). Both groups showed prominent activation in the frontal-parietal-cerebellar network known to be important for verbal WM. Despite equivalent behavioral performance between groups, alcohol-exposed individuals showed increased activation relative to typically developing individuals in left dorsal frontal and left inferior parietal cortices, and bilateral posterior temporal regions during verbal WM. These effects remained even when group differences on IQ were statistically controlled. This pattern of increased activation coupled with equivalent behavioral performance between groups suggests that individuals with FASD recruit a more extensive network of brain regions during verbal WM relative to typically developing individuals. These findings may suggest that frontal-parietal processing during verbal WM is less efficient in alcohol-exposed individuals. Hum Brain Mapp 2009. © 2009 Wiley-Liss, Inc

Read Full Article,
http://www3.interscience.wiley.com/journal/122236382/abstract

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Wiley InterScience, Journal of Child Psychology and Psychiat
ry; Volume 50 Issue 9, Pages 1073 – 1083; Published Online – 27th Feb 2009

53. IS PRENATAL ALCOHOL EXPOSURE RELATED TO INATTENTION AND HYPERACTIVITY SYMPTOMS IN CHILDREN?

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11 Biocenter Oulu, University of Oulu, Finland
12 MRC Social Genetic Developmental Psychiatry Centre, Institute of Psychiatry, King's College, London, UK

Abstract
Background: Studies concerning whether exposure to low levels of maternal alcohol consumption during fetal development is related to child inattention and hyperactivity symptoms have shown conflicting results. We examine the contribution of covariates related to social adversity to resolve some inconsistencies in the extant research by conducting parallel analyses of three cohorts with varying alcohol consumption and attitudes towards alcohol use.

Methods: We compare three population-based pregnancy–offspring cohorts within the Nordic Network on ADHD from Denmark and Finland. Prenatal data were gathered via self-report during pregnancy and birth outcomes were abstracted from medical charts. A total of 21,678 reports concerning inattention and hyperactivity symptoms in children were available from the Strengths and Difficulties Questionnaire or the Rutter Scale completed by parents and/or teachers.

Results: Drinking patterns differed cross-nationally. Women who had at least some social adversity (young, low education, or being single) were more likely to drink than those better off in the Finnish cohort, but the opposite was true for the Danish cohorts. Prenatal alcohol exposure was not related to risk for a high inattention-hyperactivity symptom score in children across cohorts after adjustment for covariates. In contrast, maternal smoking and social adversity during pregnancy were independently and consistently associated with an increase in risk of child symptoms.

Conclusions: Low doses of alcohol consumption during pregnancy were not related to child inattention/hyperactivity symptoms once social adversity and smoking were taken into account.

Read Full Article,
http://www3.interscience.wiley.com/journal/122221504/abstract?CRETRY=1&SRETRY=0

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Behav Brain Funct. 2009; 5: 3. Published online 2009 January 15

54. THE CONSEQUENCE OF FETAL ETHANOL EXPOSURE AND ADOLESCENT ODOR RE-EXPOSURE ON THE RESPONSE TO ETHANOL ODOR IN ADOLESCENT AND ADULT RATS

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Abstract

Background
An epidemiologic predictive relationship exists between fetal ethanol exposure and the likelihood for adolescent use. Further, an inverse relationship exists between the age of first experience and the probability of adult abuse. Whether and how the combined effects of prenatal and adolescent ethanol experiences contribute to this progressive pattern remains unknown. Fetal ethanol exposure directly changes the odor attributes of ethanol important for both ethanol odor preference behavior and ethanol flavor perception. These effects persist only to adolescence. Here we tested whether

adolescent ethanol odor re-exposure: (Experiment 1) augments the fetal effect on the adolescent behavioral response to ethanol odor; and/or (Experiment 2) perpetuates previously observed adolescent behavioral and neurophysiological responses into adulthood.

Methods
Pregnant rats received either an ethanol or control liquid diet. Progeny (observers) experienced ethanol odor in adolescence via social interaction with a peer (demonstrators) that received an intragastric infusion of either 1.5 g/kg ethanol or water. Social interactions were scored for the frequency that observers followed their demonstrator. Whole-body plethysmography evaluated the unconditioned behavioral response of observers to ethanol odor in adolescence (P37) or adulthood (P90). The olfactory epithelium of adults was also examined for its neural response to five odorants, including ethanol.

Results
Experiment 1: Relative to fetal or adolescent exposure alone, adolescent re-exposure enhanced the behavioral response to ethanol odor in P37 animals. Compared to animals with no ethanol experience, rats receiving a single experience (fetal or adolescent) show an enhanced, yet equivalent, ethanol odor response. Fetal ethanol experience also increased olfactory-guided following of an intoxicated peer. Experiment 2: Combined exposure yielded persistence of the behavioral effects only in adult females. We found no evidence for persistence of neurophysiological effects in either sex.

Conclusion
Fetal ethanol exposure influences adolescent re-exposure, in part, by promoting interactions with intoxicated peers. Re-exposure subsequently enhances ethanol odor responsivity during a key developmental transition point for emergent abuse patterns. While persistence of behavioral effects occurred in females, the level of re-exposure necessary to uniformly yield persistence in both sexes remains unknown. Nonetheless, these results highlight an important relationship between fetal and adolescent experiences that appears essential to the progressive pattern of developing ethanol abuse.

Read Full Article,
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2639612/

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Alcoholism: Clinical and Experimental Research, Volume 33, Issue 4, Pages 601-609
Published Online 12th January 2009

55. LONG-TERM BEHAVIORAL CHANGES IN RESPONSE TO EARLY DEVELOPMENTAL EXPOSURE TO ETHANOL IN ZEBRAFISH

Yohaan Fernandes and Robert Gerlai
From the Department of Psychology, University of Toronto at Mississauga, Mississauga, ON, Canada.

Abstract
Background: Zebrafish is becoming an important research tool for the analysis of brain function and behavior. It has been proposed to model human alcoholism as well as fetal alcohol syndrome. Previous studies investigating the consequences of exposure to ethanol during early development of zebrafish employed robust dosing regimens (high ethanol concentration and long exposure) that may model a rare situation in the human clinic. These studies found major structural abnormalities developing in the exposed fish.

Methods: Here we hope to avoid such gross changes and administer only low doses of ethanol (0.00, 0.25, 0.50, 0.75, 1.00 vol/vol %) at 24-hour postfertilization and for only a short period of time (for 2 hours). We analyze the behavior of exposed fish at adult stage using computerized stimulus presentation and automated videotracking response quantification.

Results: Despite the short ethanol exposure period and the modest concentrations, significant behavioral alterations were found: fish exposed to higher doses of ethanol swam at an increased distance from a computer-animated zebrafish shoal while their activity levels did not change.

Conclusions: Although the interpretation of and the mechanisms underlying this finding will require further investigation, the results suggest that zebrafish will be an appropriate model organism for the analysis of the effects of moderate to mild prenatal ethanol exposure.

Read Full Article,
http://www3.interscience.wiley.com/journal/121637008/abstract

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Alcoholism: Clinical and Experimental Research, Volume 33, Issue 4, Pages 634-644
Published Online 12th January 2009

56. A METRIC OF MATERNAL PRENATAL RISK DRINKING PREDICTS NEUROBEHAVIORAL OUTCOMES IN PRESCHOOL CHILDREN

Lisa M. Chiodo, James Janisse, Virginia Delaney-Black, Robert J. Sokol, and John H. Hannigan
From the Carman and Ann Adams Department of Pediatrics (LMC, VDB), Family Medicine & Public Health Sciences (JJ), Obstetrics & Gynecology (RJS, JHH), Psychology (JHH), and the C.S. Mott Center for Human Growth & Development (RJS, JHH), School of Medicine, Wayne State University, Detroit, Michigan.

Abstract
Background: Fetal Alcohol Spectrum Disorders (FASDs), including Fetal Alcohol Syndrome, continue to be high-incidence developmental disorders. Detection of patterns of maternal drinking that place fetuses at risk for these disorders is critical to diagnosis, treatment, and prevention, but is challenging and often insufficient during pregnancy. Various screens and measures have been used to identify maternal risk drinking but their ability to predict child outcome has been inconsistent. This study hypothesized that a metric of fetal "at-risk" alcohol exposure (ARAE) derived from several indicators of maternal self-reported drinking would predict alcohol-related neurobehavioral dysfunctions in children better than individual measures of maternal alcohol consumption alone.

Methods: Self-reported peri-conceptional and repeated maternal drinking during pregnancy were assessed with semi-structured interviews and standard screens, i.e., the CAGE, T-ACE, and MAST, in a prospective sample of 75 African-American mothers. Drinking volumes per beverage type were converted to standard quantity and frequency measures. From these individual measures and screening instruments, a simple dichotomous index of prenatal ARAE was defined and used to predict neurobehavioral outcomes in the 4- to 5-year-old offspring of these women. Study outcomes included IQ, attention, memory, visual-motor integration, fine motor skill, and behavior. Statistical analyses controlled for demographic and other potential confounders.

Results: The current "at-risk" drinking metric identified over 62% of the mothers as drinking at risk levels—23% more than the selection criterion identified—and outperformed all individual quantity and frequency consumption measures, including averages of weekly alcohol use and "binge" alcohol exposures (assessed as intake per drinking occasion), as well as an estimate of the Maternal Substance Abuse Checklist (Coles et al., 2000), in predicting prenatal alcohol-related cognitive and behavioral dysfunction in 4- to 5-year-old children.

Conclusions: A metric reflecting multiple indices of "at-risk" maternal alcohol drinking in pregnancy had greater utility in predicting various prenatal alcohol-related neurobehavioral dysfunction and deficits in children compared to individual measures of maternal self-reported alcohol consumption or a previous maternal substance abuse index. Assessing fetal risk drinking in pregnant women was improved by including multiple indicators of both alcohol consumption and alcohol-related consequences and, if appropriate practical applications are devised, may facilitate intervention by

health care workers during pregnancy and potentially reduce the incidence or severity of FASDs.

Read Full Article,
http://www3.interscience.wiley.com/journal/121637010/abstract

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Alcoholism: Clinical and Experimental Research, Volume 33, Issue 3 Pages 514-521
Published Online 22nd Dec 2008

57. CHARACTERIZATION OF WHITE MATTER MICROSTRUCTURE IN FETAL ALCOHOL SPECTRUM DISORDERS

Susanna L. Fryer, Brian C. Schweinsburg, Olivia A. Bjorkquist, Lawrence R. Frank, Sarah N. Mattson, Andrea D. Spadoni, and Edward P. Riley
From the San Diego Joint Doctoral Program in Clinical Psychology, San Diego State University/University of California (SLF, ADS); Department of Psychiatry, University of California (BCS); VA San Diego Healthcare System (BCS, LRF); Department of Psychology and the Center for Behavioral Teratology, San Diego State University (OAB, SNM, EPR); Department of Radiology, University of California San Diego (LRF), San Diego, California.

Abstract
Background: Exposure to alcohol during gestation is associated with CNS alterations, cognitive deficits, and behavior problems. This study investigated microstructural aspects of putative white matter abnormalities following prenatal alcohol exposure.

Methods: Diffusion tensor imaging was used to assess white matter microstructure in 27 youth (age range: 8 to 18 years) with (n = 15) and without (n = 12) histories of heavy prenatal alcohol exposure. Voxelwise analyses, corrected for multiple comparisons, compared fractional anisotropy (FA) and mean diffusivity (MD) between groups, throughout the cerebrum.

Results: Prenatal alcohol exposure was associated with low FA in multiple cerebral areas, including the body of the corpus callosum and white matter innervating bilateral medial frontal and occipital lobes. Fewer between-group differences in MD were observed.

Conclusions: These data provide an account of cerebral white matter microstructural integrity in fetal alcohol spectrum disorders and support extant literature showing that white matter is a target of alcohol teratogenesis. The white matter anomalies characterized in this study may relate to the neurobehavioral sequelae associated with gestational alcohol exposure, especially in areas of executive dysfunction and visual processing deficits.

Read Full Article,
http://www3.interscience.wiley.com/journal/121582656/abstract

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The Open Families Study Journal, 5th June 2008

58. REWARDS OF PARENTING A CHILD WITH A FETAL ALCOHOL SPECTRUM DISORDER

Jason D. Brown, Susan Rodger, Natalie George, David St. Arnault, Jennifer Sintzel

Abstract:
There is a considerable amount of research on the challenges associated with parenting children who have disabilities, and little that focuses on positive aspects. The purpose of the study was to describe the rewards of parents of children with a fetal alcohol spectrum disorder (FASD). Nineteen birth, foster or adoptive parents were asked to answer the following question: “What are the rewards of parenting a child with a FASD?” The data were analyzed using multidimensional scaling and cluster

analysis. Four clusters resulted. Parents indicated that they saw the children's effort, growth, and accomplishment in a variety of domains as encouraging. Parents also reported feeling appreciated by the children. Results suggest that rewarding parenting experiences with children who have alcohol-related disabilities are multiple, diverse and, when compared to the literature, largely consistent. The results lend credibility to the existing literature on the rewards of parenting children with developmental disabilities, and FASD in particular.

Read Full Article,
http://www.bentham-open.org/pages/b_viewarticle.php

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IFAU, Working Paper 2008:4

59. DOES A PINT A DAY AFFECT YOUR CHILD'S PAY? THE EFFECT OF PRENATAL ALCOHOL EXPOSURE ON ADULT OUTCOMES

J Peter Nilsson

Abstract
This paper utilizes a Swedish alcohol policy experiment conducted in the late 1960s to identify the impact of prenatal alcohol exposure on educational attainments and labor market outcomes. The experiment started in November 1967 and was prematurely discontinued in July 1968 due to a sharp increase in alcohol consumption in the experimental regions, particularly among youths. Using a difference-in-difference-in-differences strategy we find that around age 30 the cohort in utero during the experiment have substantially reduced educational attainments, lower earnings and higher welfare dependency rates compared to the surrounding cohorts. The results indicate that investments in early-life health may have far reaching effects on economic outcomes later in life.

Read Full Article,
http://www.ifau.se/upload/pdf/se/2008/wp08-04.pdf

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PRESS ARTICLES

Rutgers, The State University of New Jersey, Media Relations

A) ALCOHOL, PREGNANCY AND BRAIN CELL DEATH

Dipak Sarkar recently received a $3.5 million MERIT Award from the National Institutes of Health (NIH) to continue researching the damaging effects of alcohol on the nervous systems of the unborn.

The MERIT (Method to Extend Research In Time) Award will extend NIH support another 10 years for one of Sarkar's research grants, now in its 13th year. Sarkar has five active grants that support the work of 16 research assistants, including post-doctoral students, graduate students, undergraduates, and a senior scientist, who collaborate on his research projects. Sarkar jokingly says he needs five grants "just to feed these people."

"Alcohol consumption during pregnancy is a significant public health problem and may result in a wide range of adverse outcomes for the child," Sarkar says. "Many Fetal Alcohol Syndrome patients have problems coping with stress; they have learning disabilities, infections, and increased susceptibility to diseases."

These problems stem from the alcohol-induced destruction of neurons in the part of the brain known as the hypothalamus. These beta-endorphin neurons produce the endorphin hormone and are particularly vulnerable during the early development of the fetus.

Sarkar is a professor in the Department of Animal Science at the School of Environmental and Biological Sciences, director of the Endocrine Research Program, and a faculty member of the Center for Alcohol Studies. His interest in alcohol research began in 1990 when he serendipitously observed the neuron-killing effect of a small dose of alcohol while working on neuronal development.

Sarkar's research has shown that a seemingly irreversible reduction in the number and function of beta-endorphin neurons results in a permanent impairment of stress and immune system functions throughout life. While the body often displays the ability to recover from damage or disease, this does not seem to come into play with the loss of beta-endorphin neurons.

Sarkar says that preliminary data on the reduced function of beta-endorphin neurons is pointing toward "epigenetic" changes as a causal factor – changes in biochemistry that inhibit the genes responsible for these particular neurons. The genes themselves become abnormal and, while they may be producing some cells, the cells do not produce endorphin.

"One thing we cannot reverse is the death of these cells, but maybe we can reverse those epigenic alterations that are ultimately responsible for their demise," Sarkar says.

His continuing research is aimed at discovering the molecular mechanism involved in alcohol's toxic action on beta-endorphin neurons. A clear understanding of the underlying mechanism could offer a starting point from which to develop pharmaceuticals for fetal alcohol patients in the future.

Beta-endorphin neurons are also known as opioids because, like opium-based narcotics, their hormone products have the ability to reduce pain and increase a sense of well-being. Their loss would consequently have an opposite effect, reducing the ability to manage stress.

Consistent with this condition but unrelated to fetal alcohol exposure, there is substantial evidence that people with depression, schizophrenia, and other psychological disorders also have lower numbers of opioids, Sarkar says.

These neurons also have connections with the lymphatic system, which is engaged in transporting immune cells to and from the lymph nodes and can stimulate an immune response. Again, a reduction in the number of opioids can lessen the immune response and decrease the body's ability to fight infection and disease.

Beyond stress and immune function, the opioid system is also very much involved in metabolism. Sarkar notes that researchers are finding substantial evidence that an altered opioid system is involved in the metabolic changes leading to diabetes as well as obesity.

Link to the Article,
http://news.rutgers.edu/medrel/research/alcohol-and-the-deat-20090826

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American Academy of Sleep Medicine, 08/01/2009

B) POOR SLEEP IN CHILDREN MAY HAVE PRENATAL ORIGINS

Westchester, Ill. —A study in the Aug.1 issue of the journal SLEEP found that alcohol consumption during pregnancy and small body size at birth predict poorer sleep and higher risk of sleep disturbances in 8-year-old children born at term. Findings are clinically significant, as poor sleep and sleep disturbances in children are associated with obesity, depressive symptoms, attention deficit hyperactivity disorder, and poor neurobehavioral functioning.

Results indicate that children exposed prenatally to alcohol were 2.5 times more likely to have a short sleep duration of 7.7 hours or less and 3.6 times more likely to have a low sleep efficiency of 77.2 percent or less across all nights, independent of body size at birth and current maternal alcohol use. Smaller body size at birth also was associated with poorer sleep and with a higher risk for clinically significant sleep disturbances among children born at term. More specifically, lower weight and shorter length at birth were associated with lower sleep efficiency, and a lower ponderal index (an indicator of fetal growth status) was associated with the presence of sleep disturbances. In addition, children with short sleep duration were more likely to have been born via Caesarean section than were children sleeping longer (23.1 percent versus 8.4 percent respectively).

According to principal investigator Katri Räikkönen, PhD, in the department of psychology at the University of Helsinki, Finland, even low levels of weekly prenatal exposure to alcohol have adverse effects on sleep quantity and quality during childhood.

“The results were in accordance with the fetal origins of health and disease hypothesis and the many studies that have shown that adverse fetal environment may have lifelong influences on health and behavior,” said Räikkönen. “However, this is among the few studies that have reported associations between birth variables and sleep quality and quantity among an otherwise healthy population of children.”

The epidemiologic cohort study obtained data from 289 children born at term (from 37 to 42 weeks of gestation) between March and November 1998. Sleep duration and sleep efficiency (actual sleep time divided by the time in bed) were measured objectively by actigraphy at 8 years of age for an average of 7.1 days. Parents completed the Sleep Disturbance Scale for Children to report sleep problems and sleep disorder symptoms such as bedtime resistance and sleep disordered breathing.

Results show that the odds for low sleep efficiency increased by 70 percent for every standard deviation decrease in weight at birth and by more than 200 percent for every decrease in length. For every standard deviation decrease in ponderal index at birth, the risk of parent-reported sleep disorders increased by 40 percent. Associations were not confounded by sex, gestational length, prenatal and perinatal complications, body mass index (BMI) at eight years of age, asthma, allergies or parental socioeconomic status.

The authors report that small body size at birth may function as a crude marker of disturbances in the fetal environment, and it is associated with prematurity, intrauterine growth retardation, prenatal alcohol exposure and poorer sleep quality in children and young adults. Results demonstrate that

among children born healthy and at full-term, a linear relationship exists between smaller body size at birth and poorer sleep quality eight years from birth.

SLEEP is the official journal of the Associated Professional Sleep Societies, LLC (APSS), a joint venture of the American Academy of Sleep Medicine and the Sleep Research Society.The APSS publishes original findings in areas pertaining to sleep and circadian rhythms. SLEEP, a peer-reviewed scientific and medical journal, publishes 12 regular issues and 1 issue comprised of the abstracts presented at the SLEEP Meeting of the APSS.

For a copy of the study, “Prenatal Origins of Poor Sleep in Children,” or to arrange an interview with the study’s author, please contact Kelly Wagner, AASM public relations coordinator, at (708) 492-0930, ext. 9331, or kwagner@aasmnet.org.

Link to the Article,
http://www.aasmnet.org/Articles.aspx?id=1380

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e! Science News, Published: Wednesday, June 10, 2009

C) SPECIFIC GENETIC CAUSE OF FETAL ALCOHOL-RELATED DEVELOPMENTAL DISORDERS FOUND

Alcohol consumption by pregnant women hinders brain development in their children by interfering with the genetic processes that control thyroid hormone levels in the fetal brain, a new animal study found. Results will be presented Wednesday at The Endocrine Society's 91st Annual Meeting in Washington, D.C. Fetal alcohol exposure—even from moderate drinking during pregnancy—can cause neurodevelopmental disorders, such as emotional behavioral disorders and deficits in learning, memory and speech. There is currently no treatment for these problems, said the author who will present the study results, Laura Sittig, a student at Northwestern University Feinberg School of Medicine.

Past animal research shows that some of these lasting cognitive impairments occur because alcohol consumption during pregnancy decreases the level of maternal thyroid hormones and, therefore, fetal thyroid hormones.

"Specific concentrations of thyroid hormones must be available in the fetal brain to support normal neurological development," Sittig said. One of the enzymes that control thyroid hormone levels in the fetal brain is the iodothyronine deiodinase type III, or Dio3, she explained. Sittig and her colleagues hypothesized that alcohol exposure in the womb leads to cognitive impairments by inducing epigenetic alterations—changes to DNA that do not alter the actual DNA sequence—of developmental genes like Dio3 in the fetal brain. To investigate this hypothesis, they used rats to model moderate alcohol consumption during pregnancy.

The study, which was funded by the National Institutes of Health, demonstrated that fetal alcohol exposure disrupts the epigenetic "imprinting" of Dio3. In this process, Dio3 normally originates from the father's gene, while the maternal gene is silenced by epigenetic control. But alcohol exposure changes the paternal-maternal dosage of Dio3, which increases the amount of the enzyme present in specific brain regions of the fetus, the authors found.
This increase, in turn, reduces the availability of vital thyroid hormones in the parts of the brain that control learning, memory and emotional behaviors.

"In light of our current finding, we can begin testing specific dietary supplements that could reverse the epigenetic alterations that disrupt the regulation of Dio3," Sittig said. "When given to the mother or newborn, this might correct the imprinting deficits induced by alcohol." "This is a promising avenue to improve the prognosis of alcohol-related neurodevelopmental disorders, for which we currently have no intervention strategy," she said.

Link to Article,
http://esciencenews.com/articles/2009/06/10/specific.genetic.cause.fetal.alcohol.related.developmental.disorders.found

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The Ring, University of Victoria's community newspaper, March 2009-11-08

D) A CLEAR LINK BETWEEN EXERCISE AND BRAIN HEALTH HOLDS PROMISE FOR THE TREATMENT OF NEUROLOGICAL DISORDERS

Sheila Potter

In case you needed one, here's another good reason to exercise-it can make you smarter.

University of Victoria neuroscientist Dr. Brian Christie was one of the first researchers to discover that exercise stimulates the growth of brain cells in the hippocampus, an area of the brain involved with learning and memory.

The finding debunked the long-held belief that our brains aren't able to produce new cells-known as neurons-as we age.

"We now know that new neurons are produced continually throughout our lives and that this process can be ramped up or dampened by our lifestyles," says Christie. "In other words, the better we take care of our brains, the better they function."

Christie studies the biological mechanisms in the brain that are activated by exercise. A deeper understanding of these mechanisms may ultimately result in new approaches to establishing, maintaining and even enhancing brain cells and their connections as we age.

The applications of Christie's research are astonishingly broad. Exercise seems to reduce the impact of any stress on the brain, whether the stress comes from a hard day at work or from such neurological disorders as Alzheimer's disease, autism, stroke or fetal alcohol spectrum disorder (FASD).

FASD refers to a spectrum of disorders associated with poor learning, attention, memory and behavioural problems.
"FASD is a tricky problem, because a lot of women don't realize that they are pregnant in the early stages and can consume alcohol unwittingly, and they may not be aware of the toxic effects of alcohol on the developing fetus," says Christie. "The bottom line is that no amount of alcohol is safe when you're pregnant."

The link between FASD and exercise first occurred to Christie at a medical conference. "The presenter was describing how children with FASD have fewer neurons in their hippocampus, and that these neurons are less branched," he says. "This is the diametric opposite of the positive effects of exercise. It was a definite 'aha' moment."

Using sophisticated microscopy and protein chemistry techniques, Christie and his team have demonstrated that exercise promotes the growth of new neurons in FASD brains, and that these neurons are better able to communicate with each other.

In fact, Christie was surprised by how big a difference exercise makes for FASD compared to other brain disorders he has studied. He believes daily exercise should be a key treatment for FASD, guessing that an hour a day, continuous or broken up, might be enough.

Christie notes that FASD can be very difficult to diagnose and children showing symptoms are often misdiagnosed with attention-deficit hyperactivity disorder (ADHD). These kids are typically

discouraged from running around for fear they will get overexcited-clearly a bad strategy given his findings.

Christie and his team are now looking at the effects of different amounts of alcohol at various stages of pregnancy. They're also investigating sex differences-it's possible that testosterone makes developing brains more susceptible to alcohol damage, making FASD worse in boys.

Christie's research is supported by the Canadian Institutes of Health Research, the Natural Sciences and Engineering Research Council and the Michael Smith Foundation for Health Research.

Link to the Article,
http://ring.uvic.ca/09mar05/excercise.html

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CDC, May 22, 2009 / 58(19); 529-532

E) ALCOHOL USE AMONG PREGNANT AND NONPREGNANT WOMEN OF CHILDBEARING AGE --- UNITED STATES, 1991—2005

CH Denny, PhD, J Tsai, MD, RL Floyd, DSN, PP Green, MSPH, Div of Birth Defects and Developmental Disabilities, National Center on Birth Defects and Developmental Disabilities, CDC.

Alcohol consumption during pregnancy is a risk factor for poor birth outcomes, including fetal alcohol syndrome, birth defects, and low birth weight (1). In the United States, the prevalence of fetal alcohol syndrome is estimated at 0.5--2.0 cases per 1,000 births, but other fetal alcohol spectrum disorders (FASDs)* are believed to occur approximately three times as often as fetal alcohol syndrome (2). The 2005 U.S. Surgeon General's advisory on alcohol use in pregnancy, advises women who are pregnant or considering becoming pregnant to abstain from using alcohol (2). Binge drinking is particularly harmful to fetal brain development (2,3). Healthy People 2010 objectives include increasing the percentage of pregnant women who report abstinence from alcohol use to 95% and increasing the percentage who report abstinence from binge drinking to 100% (4). To examine the prevalence of any alcohol use and binge drinking among pregnant women and nonpregnant women of childbearing age in the United States and to characterize the women with these alcohol use behaviors, CDC analyzed 1991--2005 data from Behavioral Risk Factor Surveillance System (BRFSS) surveys. The findings indicated that the prevalence of any alcohol use and binge drinking among pregnant and nonpregnant women of childbearing age did not change substantially from 1991 to 2005. During 2001--2005, the highest percentages of pregnant women reporting any alcohol use were aged 35--44 years (17.7%), college graduates (14.4%), employed (13.7%), and unmarried (13.4%). Health-care providers should ask women of childbearing age about alcohol use routinely, inform them of the risks from drinking alcohol while pregnant, and advise them not to drink alcohol while pregnant or if they might become pregnant (2,5).

BRFSS conducts state-based, random-digit--dialed telephone surveys of the noninstitutionalized U.S. civilian population aged ≥18 years, collecting data on health conditions and health risk behaviors. For this report, CDC analyzed BRFSS data from 1991 to 2005 from all 50 states and the District of Columbia for women aged 18--44 years. The median response rate among states, based on Council of American Survey and Research Organizations (CASRO) guidelines, ranged from 71.4% in 1993 to 51.1% in 2005. This report focuses on two drinking behaviors: any use, defined as having at least one drink of any alcoholic beverage in the past 30 days, and binge drinking, defined as having five or more drinks on at least one occasion in the past 30 days.† The wording of the question regarding any alcohol use was changed in 1993, 2001, and 2005,§ the wording of the question regarding binge drinking was changed in 1993 and 2001.¶ BRFSS questionnaires are available at http://www.cdc.gov/brfss/questionnaires/questionnaires.htm.

Percentage estimates and 95% confidence intervals were calculated each year for the two drinking behaviors among pregnant and nonpregnant women. Logistic regression was used to examine the association of age, race/ethnicity, education, employment, and marital status with the two drinking

behaviors for pregnant and nonpregnant women with the behaviors as the dependent variables and sociodemographic characteristics as the independent variables in the models. Adjusted odds ratios (AORs) were calculated to describe significant differences by characteristic category. Data from 2001--2005 were aggregated to provide stable estimates to assess the association of these characteristics with the drinking behaviors. Data were weighted to state population estimates and aggregated to represent a nationwide estimate.

Of the 533,506 women aged 18--44 years surveyed during 1991--2005, 22,027 (4.1%) reported being pregnant at the time of the interview. The prevalence of any alcohol use and binge drinking among pregnant and nonpregnant women from 1991 to 2005 did not change substantially over time (Figure). The average annual percentage of any alcohol use among pregnant women was 12.2% (range: 10.2%--16.2%), of binge drinking among pregnant women was 1.9% (range: 0.7%--2.9%), of any alcohol use among nonpregnant women was 53.7% (range: 51.6%--56.3%), and of binge drinking among nonpregnant women was 12.1% (range: 10.8%--13.7%).

Of the 329,975 women aged 18--44 years surveyed during 2001--2005, 13,820 (4.2%) reported being pregnant at the time of the interview. Among pregnant women, 17.7% of those aged 35--44 years reported any alcohol use, compared with 8.6% of pregnant women aged 18--24 years (AOR = 2.3). Greater percentages of pregnant women with at least some college education (11.2%), or a college degree or more (14.4%), reported alcohol use than pregnant women with a high school diploma or less (8.5%) (AORs = 1.4 and 1.9, respectively). A greater percentage of employed pregnant women (13.7%) reported alcohol use than unemployed pregnant women (8.3%, AOR = 1.5), and a greater percentage of unmarried pregnant women (13.4%) reported alcohol use than married pregnant women (10.2%, AOR = 2.2) (Table). In addition, a greater percentage of employed pregnant women (2.3%) reported binge drinking, compared with unemployed pregnant women (1.3%, AOR = 1.8), and a greater percentage of unmarried pregnant women (3.6%) reported binge drinking than married pregnant women (1.1%, AOR = 4.4).

Read Full Article,
http://www.cdc.gov/Mmwr/preview/mmwrhtml/mm5819a4.htm

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Special Education Law Blog, 27th October 2008

F) VITAMIN MAY REDUCE THE EFFECTS OF FETAL ALCOHOL

Amanda Windom

Prenatal damage to infants by drinking alcohol while pregnant is a growing problem. Estimates vary from, for every 1000 live births in the U.S. 1-2 infants are born with fetal alcohol syndrome and 3-5 are born with fetal alcohol effects (a less severe diagnosis) to fetal alcohol spectrum disorders occuring in 1 out of every 100 live births. Despite increased warnings that drinking alcohol while pregnant is damaging to the developing fetus statistics show that more than 50% of pregnant women between the ages of 15-44 reported drinking while pregnant. As a result, scientists are now looking for ways to treat fetal alcohol effects after birth.

Science Daily reports that research conducted at San Diego University found that giving the vitamin choline to effected infants may help reduce some of the negative effects. The study, headed by Jennifer Thomas, PhD, involved 170 rats and demonstrated that giving choline to pups who were exposed to alcohol significantly reduced overactivity and learning deficits. Motor coordination deficits were not affected by the vitamin and it is important to note that giving choline is not going to fix all the problems associated with fetal alcohol effects, women must continue to be cautioned not to drink while pregnant says Thomas. Preliminary results show that the positive effects of choline are long lasting and more research must be done to discover how late in development the vitamin can be given and still be effective.

Choline is a chemical that is similar to B-vitamins, but not classified as one. It is essential for brain

development in both fetus' and infants and may even help prevent memory loss associated with aging. Choline has been shown to protect the liver from certain types of damage and may even reverse damage that has already occured. It may also help reduce cholesterol and protect against certain cancers, among other things. Choline can be obtained through some foods such as beef, cauliflower, and peanut butter and it is important not to get more than the recommended doses. (Click on the link above for a chart showing recommended amount by age and sex.)

Other studies have also demonstrated the positive effects of choline on prenatal development in rats, and Thomas and her colleagues hope to conduct clinical studies of choline on infants affected by prenatal alcohol exposure. While preliminary results look good, remember to always check with your child's doctor before giving them any vitamin supplements.

Link to the Article,
http://specialedlaw.blogs.com/home/2008/10/vitamin-may-red.html

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Calgary Herald, October 24th 2008

G) ALBERTA GOVERNMENT ANNOUNCES TREATMENT BEDS FOR YOUNG WOMEN

CALGARY- A Calgary addictions centre hopes to replace the crutch of alcohol and drugs with the embrace of healing, with eight new residential treatment beds announced today for young women.
Clients routinely wait six to eight weeks for spaces at Aventa, but an official with the province's lone women's-only addictions treatment facility said the centre has always strived to find beds immediately for pregnant women gripped by substance abuse, and at risk of harming themselves and giving birth to children with severe Fetal Alcohol Spectrum Disorder.

"They may not prevent it 100 per cent, but they may prevent the degree of FASD, and that's very important," said Mara Thorvalson, Aventa's clinical director.

The Alberta government announced today the opening of 20 new treatment beds for young Albertans — the eight at Aventa, and five for males and seven for female inpatients in the Edmonton area. Health Minister Ron Liepert said 20 new spaces may not sound like much, but the $1.5-million investment will help hundreds of young men and women, often staying in rehab for three-month stints or shorter ones.

These are the first of 100 beds for addictions treatment promised last year by the Stelmach Tories, in response to a task force on community safety.

The province is on track to help fund the rest of them by 2010, Justice Minister Alison Redford told reporters.
It's part of the government's renewed efforts to tackle the root causes of crime and keep teens and young adults out of the justice system, so "we're doiong a much better job than assuming everyone needs to go to jail," Redford said.

Aventa's new youth-only treatment beds opened last month, the first major expansion since it opened its 36-bed facility in the Mission neighbourhood four years ago.

Link to the Article,
http://www.canada.com/calgaryherald/news/story.html?id=5968c674-7caf-4424-8a7e-c96343d2e448

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