FASlink Fetal Alcohol Syndrome Information, Support & Communications Link

http://www.acbr.com/fas/

Fetal Alcohol Syndrome

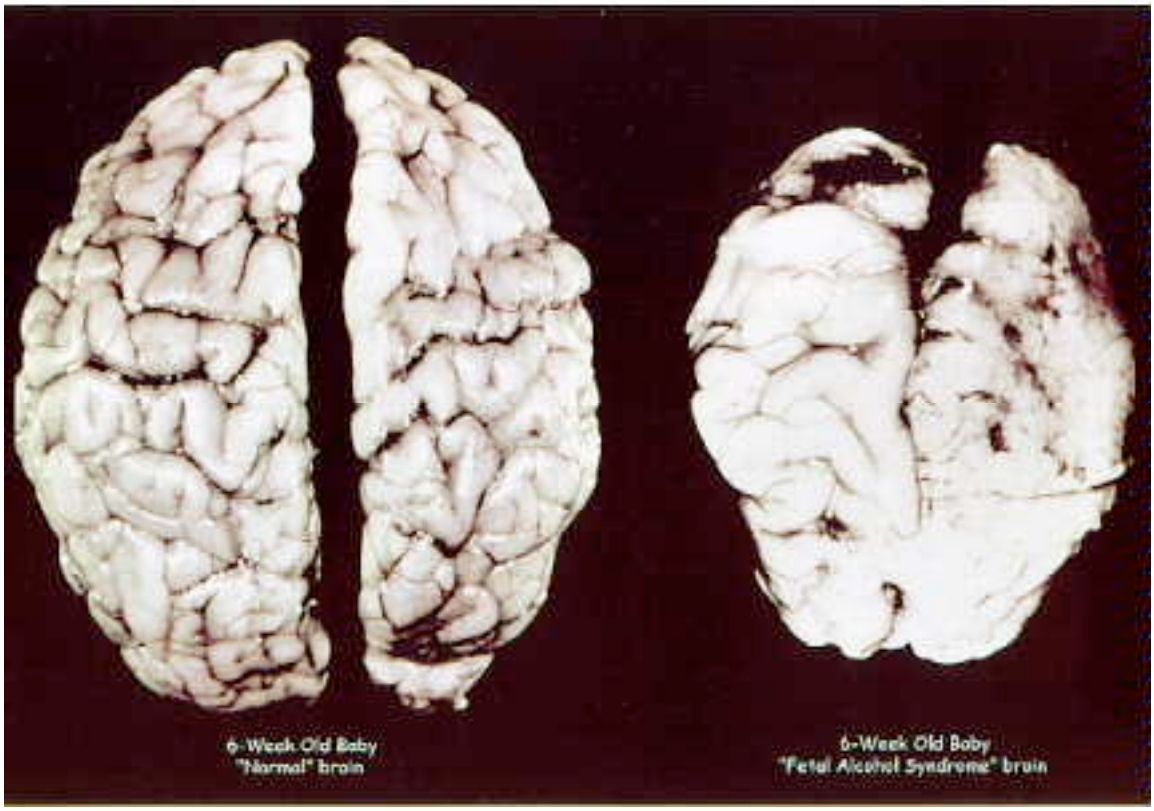
An individual's place, and success, in society is almost entirely determined by neurological functioning.

A neurologically injured child is unable to meet the expectations of parents, family, peers, school, career and can endure a lifetime of failures. The largest cause of neurological damage in children is prenatal exposure to alcohol. These children grow up to become adults. Often the neurological damage goes undiagnosed, but not unpunished.

They can become the forgotten kids - the children that have nearly invisible disabilities. They have their arms and legs, can see and hear, run, play, etc., but most have never been to a birthday party or a sleepover.. they are last to be chosen to play, and first to be blamed. Their illnesses aren't fatal, but a small part of their hearts and souls die with every rejection. Their behaviors may seem odd or unpredictable to themselves as much as society.

Fetal Alcohol Syndrome (**FAS**), Fetal Alcohol Effects (**FAE**), Alcohol Related Neurodevelopmental Disorders (**ARND**), Static Encephalopathy (alcohol exposed) (**SE**) or Alcohol Related Birth Defects (**ARBD**) are all names for a spectrum of disorders caused when a pregnant woman consumes alcohol.

More than 10% of children have been exposed to high levels of alcohol in utero. **All** will suffer varying degrees of effects, ranging from mild learning disabilities to major physical, mental and intellectual impairment. It takes very little alcohol to cause serious damage. Research has shown that even a single exposure to high levels of alcohol can cause significant brain damage in the infant.



Six week-old brains

Normal *FAS*

Alcohol is toxic at all concentrations. Alcohol damage to the fetus occurs over a wide continuum. Damage varies due to volume ingested, timing during pregnancy, peak blood alcohol levels, genetics and environmental factors.

FAS/E is a lifetime disability. It is not curable. A child does not "grow out of it". However, early diagnosis and intensive, and appropriate, intervention can make an enormous difference in the prognosis for the child. There is a small window of opportunity, up to about age 10 or 12, to achieve the greatest potential for an alcohol affected child. That period is when the greatest development of fixed neural pathways occurs. That is when alternative "coping" pathways are most easily built as "work-arounds" to damaged areas of the brain. Time is of the essence.

In utero alcohol damage can include:

Loss of intellectual functioning (IQ)	Mild to severe vision problems	Higher than normal to dangerously high pain tolerance
Severe loss of intellectual potential	Mental Retardation	Dyslexia
Serious maxilo-facial deformities	Dental abnormalities	Cleft palate
Immune system malfunctioning	Behavioral problems	Attention deficit disorders
ADD/ADHD	Extreme impulsiveness	Poor judgment
Little or no retained memory	Deafness	Little or no capacity for moral judgment
Little or no capacity for interpersonal empathy	Sociopathic behavior	Epilepsy
Tremors	Cerebral palsy	Renal (liver) failure
Asthma	Complex seizure disorder	Developmental speech and language disorder
Developmental delay	Height and weight deficiencies	Tight hamstrings
Cognitive perservations	Echolalia or stereotypical repetition of another's words or phrases	Autistic traits
Rigidity	Sleep disorder	Developmental coordination disorder
Adaptive esotropia (cross eyed)	Tourette's traits	Central auditory processing disorder
Night terrors	Precocious puberty	Social problems
Depression	Reactive outbursts	Suicide
Heart defects	Heart failure	Death

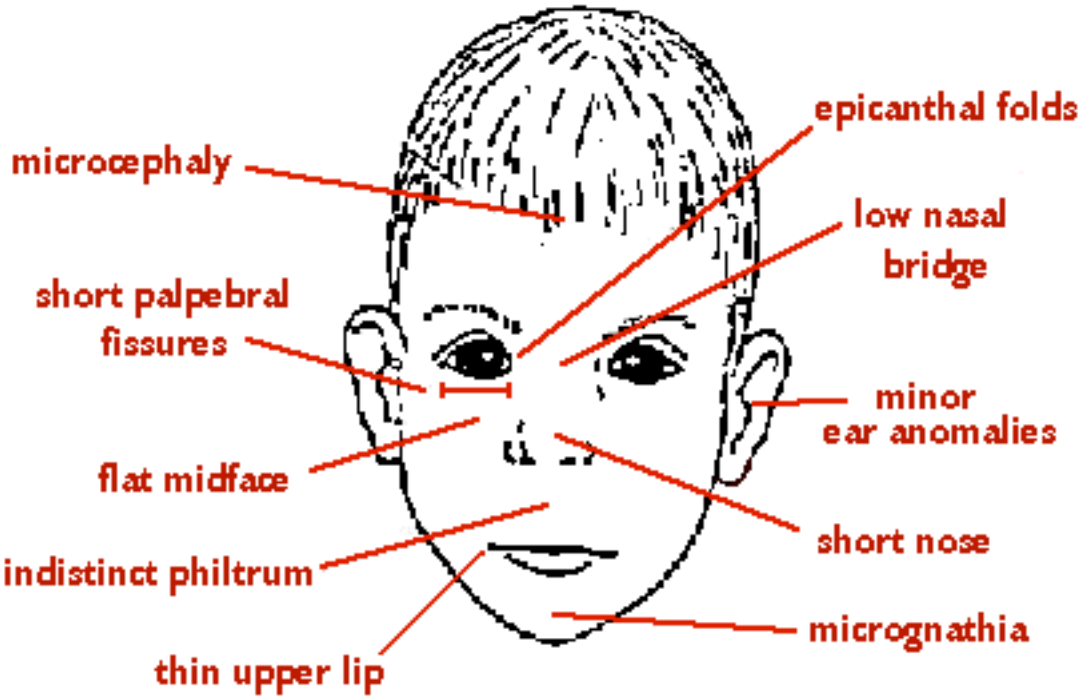
The brain's **Frontal Lobes** control judgement, inhibition, concentration, self-control, conscience, personality and emotional traits as well as cognition and memory, motor speech and movement skills.

The **Left** Hemisphere deals with language based memory - **logical interpretation** of language, mathematics, abstraction and reasoning, facts and rules (such as safety and social).

The **Right** Hemisphere deals with **holistic functioning -** processing of images, sound, touch, for a "holistic" picture. Memory here is visual, auditory and spatial. So, the Left side is logic, facts, rules. The Right side is sensory input and reactive.

The **Corpus Callosum** connects right and left sides to allow communication between the hemispheres. The Right side senses input, checks with the Left side to see if there are rules to deal with this pattern of input, integrates the stored information and reacts in a modified way. Damage to any of these systems causes very poor, inappropriate response. For example, if the Corpus Callosum cannot access the appropriate information, quickly enough (or at all), then reaction to stimulae will be completely spontaneous, impulsive, based solely on instinct, (if any). Alcohol seriously damages the physical structures, "wiring" and brain chemistry.

FAS (Fetal Alcohol Syndrome) individuals may have a distinctive physical appearance and lower IQs, but have lower crime and addiction rates than FAE individuals as they get earlier diagnosis and can be better protected by society and their parents.



While FAE (Fetal Alcohol Effects) individuals may lack the outward physical appearance of alcohol damage, and generally have higher IQ's, the internal damage to the brain and other organs can be just as serious as full FAS. IQ measures convergent fact based thinking. Life skills require divergent adaptive thinking that in FAE individuals will be substantially lower than their IQ. However, because FAE individuals "look normal" they are expected to perform normally. These issues lead to secondary disabilities. Primary disabilities are those the child is born with. Secondary

disabilities are those that develop as a result of failure to properly deal with the primary disabilities.

"**The girls get knocked up and the boys get locked up."** They are followers, easily misled, with little or no appreciation of consequences. Without intervention, many ride the justice system merry-go-round or become **"homeless street people"**. They are required to compete in society but have been denied the tools to do so.

Of FAE individuals between the ages of 12 and 51:

- 95% will have mental health problems;
- 60% will have "disrupted school experience";
- 60% will experience trouble with the law;
- 55% will be confined in prison, drug or alcohol treatment centre or mental institution;
- 52% will exhibit inappropriate sexual behaviour.

Of FAE individuals between 21 and 51:

- more than 50% of males and 70% of females will have alcohol and drug problems;
- 82% will not be able to live independently;
- 70% will have problems with employment
-

http://depts.washington.edu/fadu/

Early diagnosis can help prevent secondary disabilities such as mental health problems, dropping out of school, trouble with the law and substance abuse. **After diagnosis, parents often find that their ability to cope with the child's behavior changes dramatically when they understand that the problems are most likely based on organic brain damage, rather than the child's choice to be inattentive or uncooperative.**

http://depts.washington.edu/fasdpn/whatisfasdpn.html

Costs of FAS/E

On average, each FAS/E individual costs the taxpayer more than **$3 million** in his or her lifetime (health problems, special education, psychotherapy and counseling, welfare, crime, and the justice system).

More than 60% of prisoners are likely affected by alcohol in utero. It costs approximately **$120,000/year** to "house" a Young Offender and **$82,000 for** an adult offender. Punishment does not cure neurological damage.

Add on:

- the FAS/E individual's own lifetime loss of income;
- the high costs to the families (foster, adoptive or biological) who raise and care for FAS/E children and adults;
- the lost income of a parent who must care for the exceptionally high needs of an FAS/E child;
- the costs to families whose FAS/E child is permanently dependent upon them;
- the costs of legal services for defending their child in the courts;
- the cost of stress caused divorce, etc.

Don't Ask My Child to Fly

Bruce Ritchie 1997

Don't ask my child to fly,
for he has not wings.

Don't ask my child to see the glint on the eagle's beak,
for his vision has been diminished.

Don't ask my child to remain calm amid the din,
for her ability to screen out the noises has been taken away.

Don't ask my child to be careful with "strangers",
for he is affectionate with everyone and prey for the unscrupulous.

Don't ask my child to "settle down",
for the clock which works for you and I, does not exist for her.

Don't ask my child to not play with the toys of others,
for he has no concept of property.

Don’t ask my child to remember you tomorrow,
although you met today.

Don't ask my child to heal your wounds,
for her hands cannot hold a scalpel or sutures.

Don't ask my child to meet the challenges set by society,
for you have denied her the tools.

Don't ask my child to forgive you for standing idly by,
while he was being tortured in his mother's womb,

for he will,

but **He** may not.

Low dose prenatal alcohol exposure linked to behavior problems

Prenatal Alcohol Exposure and Childhood Behavior at Age 6 to 7 Years: I.

Dose-Response Effect

The study is a significant one. Take note:

Maternal alcohol consumption even at low levels was adversely related to child behavior; a dose-response relationship was also identified. The effect was observed at average levels of exposure of as low as 1 drink per week.

Here is the abstract:

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Beena Sood*, Virginia Delaney-Black*, Chandice Covington, Beth Nordstrom-Klee*, Joel Ager§, Thomas Templin, James Janisse§, Susan Martier, and Robert J. Sokol . From the * Department of Pediatrics, School of Medicine, College of Nursing, § Center for Healthcare Effectiveness Research, School of Medicine, and Department of Obstetrics and Gynecology, School of Medicine, Wayne State University, Detroit, Michigan.

Objective. Moderate to heavy levels of prenatal alcohol exposure have been associated with alterations in child behavior, but limited data are available on adverse effects after low levels of exposure. The objective of this study was to evaluate the dose-response effect of prenatal alcohol exposure for adverse child behavior outcomes at 6 to 7 years of age.

Methods. Beginning in 1986, women attending the urban university-based maternity clinic were routinely screened at their first prenatal visit for alcohol and drug use by trained research assistants from the Fetal Alcohol Research Center. All women reporting alcohol consumption at conception of at least 0.5 oz absolute alcohol/day and a 5% random sample of lower level drinkers and abstainers were invited to participate to be able to identify the associations between alcohol intake and child development. Maternal alcohol, cigarette, and illicit drug use were prospectively assessed during pregnancy and postnatally. The independent variable in this study, prenatal alcohol exposure, was computed as the average absolute alcohol intake (oz) per day across pregnancy. At each prenatal visit, mothers were interviewed about alcohol use during the previous 2 weeks. Quantities and types of alcohol consumed were converted to fluid ounces of absolute alcohol and averaged across visits to generate a summary measure of alcohol exposure throughout pregnancy. Alcohol was initially used as a dichotomous variable comparing children with no prenatal alcohol exposure to children with any exposure. To evaluate the effects of different levels of exposure, the average absolute alcohol intake was relatively

arbitrarily categorized into no, low (>0 but <0.3 fl oz of absolute alcohol/day), and moderate/heavy (0.3 fl oz of absolute alcohol/day) for the purpose of this study. Six years later, 665 families were contacted. Ninety-four percent agreed to testing. Exclusions included children who missed multiple test appointments, had major congenital malformations (other than fetal alcohol syndrome), possessed an IQ >2 standard deviations from the sample mean, or had incomplete data. The Achenbach Child Behavior Checklist (CBCL) was used to assess child behavior. The CBCL is a parent questionnaire applicable to children ages 4 to 16 years. It is widely used in the clinical assessment of children's behavior problems and has been extensively used in research. Eight syndrome scales are further grouped into Externalizing or undercontrolled (Aggressive and Delinquent) behavior and Internalizing or overcontrolled (Anxious/Depressed, Somatic Complaints, and Withdrawn) behaviors. Three syndromes (Social, Thought, and Attention Problems) fit neither group. Higher scores are associated with more problem behaviors. Research assistants who were trained and blinded to exposure status independently interviewed the child and caretaker. Data were collected on a broad range of control variables known to influence childhood behavior and/or to be associated with prenatal alcohol exposure. These included perinatal factors of maternal age, education, cigarette, cocaine, and other substances of abuse and the gestational age of the baby. Postnatal factors studied included maternal psychopathology, continuing alcohol and drug use, family structure, socioeconomic status, children's whole blood lead level, and exposure to violence. Data were collected only from black women as there was inadequate representation of other racial groups.

Statistical Analyses. Statistical analyses were performed using the SPSS statistical package. Frequency distribution, cross-tabulation, odds ratio, and 2 tests were used for analyzing categorical data. Continuous data were analyzed using t tests, analyses of variance (ANOVAs) with posthoc tests, and regression analysis.

Results. Testing was available for 501 parent-children dyads. Almost one fourth of the women denied alcohol use during pregnancy. Low levels of alcohol use were reported in 63.8% and moderate/heavy use in 13% of pregnancies. Increasing prenatal alcohol exposure was associated with lower birth weight and gestational age, higher lead levels, higher maternal age, and lower education level, prenatal exposure to cocaine and smoking, custody changes, lower socioeconomic status, and paternal drinking and drug use at the time of pregnancy. Children with any prenatal alcohol exposure were more likely to have higher CBCL scores on Externalizing (Aggressive and Delinquent) and Internalizing (Anxious/Depressed and Withdrawn) syndrome scales and the Total Problem Score. The odds ratio of scoring in the clinical range for Delinquent behavior was 3.2 (1.3-7.6) in children with any prenatal exposure to alcohol compared with nonexposed controls. The threshold dose was evaluated with the 3 prenatal alcohol exposure groups. One-way ANOVA revealed a significant between group difference for Externalizing (Aggressive and Delinquent) and the Total Problem Score. Posthoc tests revealed the between group differences to be significant (no and low-exposure group) for Aggressive and Externalizing behavior suggesting that the adverse effects of prenatal alcohol exposure on child behavior at age 6 to 7 years are evident even at low levels of exposure. For Delinquent and Total Problem behavior, the difference was significant

between the no and moderate-heavy exposure group, suggesting a higher threshold for these behaviors. Prenatal alcohol exposure remained a significant predictor of behavior after adjusting for covariates. Although maternal psychopathology was the most important predictor of behavior, gender was also a significant predictor, with boys having higher scores on Externalizing (Delinquent) and Attention Problems. The amount of variance uniquely accounted for by prenatal alcohol exposure ranged between 0.6% to 1.7%.

Conclusions. Maternal alcohol consumption even at low levels was adversely related to child behavior; a dose-response relationship was also identified. The effect was observed at average levels of exposure of as low as 1 drink per week. Although effects on mean scores for Externalizing and Aggressive behaviors were observed at low levels of prenatal alcohol exposure, effects on Delinquent behavior and Total Problem Scores were observed at moderate/heavy levels of exposure. Children with any prenatal alcohol exposure were 3.2 times as likely to have Delinquent behavior scores in the clinical range compared with nonexposed children. The relationship between prenatal alcohol exposure and adverse childhood behavior outcome persisted after controlling for other factors associated with adverse behavioral outcomes. Clinicians are often asked by pregnant women if small amounts of alcohol intake are acceptable during pregnancy. These data suggest that no alcohol during pregnancy remains the best medical advice.

Key words: Child Behavior Checklist, child behavior, alcohol-related neurobehavioral effects.

Full article is here:
http://www.pediatrics.org/cgi/content/abstract/108/2/e34?maxtoshow=&HITS&hits&RESULTFORMAT=&fulltext=alcohol&searchid20351370447_3336&stored_search=&FIRSTINDEX=0&volume8&fdate=1/1/2001&journalcode=pediatrics
(be sure to get entire URL in browser box)

Fetal Alcohol Syndrome

Alaska's #1 Preventable Birth Defect

Diagnosing FAS

Individuals with FAS are a subset of individuals who are affected by in-utero exposure to alcohol. They are not necessarily the most severely affected individuals; they are simply the subset that can be positively identified because of their characteristic facial appearance (Clarren, S.K., & Astley, S.J. 1993). Other terms are often used to classify individuals who do not have FAS but share characteristics associated with FAS (especially central nervous system dysfunction and other cognitive abnormalities). These terms include fetal alcohol effects (FAE), static encephalopathy, neurobehavioral disorder, alcohol-related birth defects (ARBD), and alcohol-related neurodevelopmental disorder (ARND). These terms are sometimes used interchangeably. A diagnosis of alcohol-related effects may also be made with or without confirmed maternal alcohol exposure.

Fetal alcohol syndrome is a medical diagnosis usually made by a physician specifically trained in the assessment of birth defects. Other professionals often assist in identifying children with known maternal drinking histories or suspected problems. For example, nurses may be trained to recognize the facial features of children with fetal alcohol syndrome. It is important that the physician making the diagnosis is sensitive to the physical characteristics of the racial group with whom he or she is working because the physical characteristics of FAS may look slightly different among different racial groups.

Accurate diagnosis of alcohol-related effects require that the physician be qualified. Accurate identification can improve the child's opportunity to receive appropriate interventions, facilitate communication among clinicians, caregivers, and educators, and provides better self-awareness and understanding by family members.

The following information has been extracted from **Ninth Special Report to the U.S. Congress on Alcohol and Health, June 1997(RP0973)**. Free copies of this report are available from National Clearinghouse for Alcohol and Drug Information, 1-800-729-6686.

A key concern in research and clinical practice continues to be how best to characterize and identify FAS and other ARBD arising from prenatal alcohol exposure. Research has shown that in utero alcohol exposure can produce a spectrum of harmful effects, ranging from a characteristic pattern of gross morphological anomalies and mental impairment (including mental retardation) to subtler cognitive and behavioral dysfunction. FAS is the most severe birth defect produced by in utero alcohol exposure. The terms "fetal alcohol effects" (FAE) and "alcohol-related birth defects" are used to describe individuals who exhibit only some of the attributes of FAS and thus do not fulfill the diagnostic criteria for the syndrome (Clarren and Smith 1978; Sokol and Clarren 1989). A medical diagnosis of FAS is differentiated from a "case definition" for surveillance purposes.

TABLE 1: Diagnostic Criteria for Fetal Alcohol Syndrome (FAS) and Alcohol-Related Effects

Fetal Alcohol Syndrome

1. FAS with confirmed maternal alcohol exposures

A. Confirmed maternal alcohol exposures

B. Evidence of a characteristic pattern of facial anomalies that includes features such as short palpebral fissures and abnormalities in the premaxillary zone (e.g., flat upper lip, flattened philtrum, and flat midface)

C. Evidence of growth retardation, as in at least one of the following:

- low birth weight for gestational age

- decelerating weight over time not due to nutrition

- disproportional low weight to height

D. Evidence of CNS neurodevelopmental abnormalities, as in at least one of the following: - decreased cranial size at birth

- structural brain abnormalities (e.g., microcephaly, partial or complete agenesis of the corpus callosum, cerebellar hypoplasia)

- neurological hard or soft signs (as age appropriate), such as impaired fine motor skills, neurosensory hearing loss, poor tandem gait, poor eye-hand coordination

2. FAS without confirmed maternal alcohol exposure

B, C, and D as above

3. Partial FAS **with confirmed maternal alcohol exposure**

A. Confirmed maternal alcohol exposure'

B. Evidence of some components of the pattern of characteristic facial anomalies Either C or D or E

C. Evidence of growth retardation, as in at least one of the following:

- low birth weight for gestational age

- decelerating weight over time not due to nutrition

- disproportional low weight to height

D. Evidence of CNS neurodevelopmental abnormalities, as in:

- decreased cranial size at birth

- structural brain abnormalities (e.g., microcephaly, partial or complete agenesis of the corpus callosum, cerebellar hypoplasia)

- neurological hard or soft signs (as age appropriate) such as impaired fine motor skills, neurosensory hearing loss, poor tandem gait, poor eye-hand coordination

E. Evidence of a complex pattern of behavior or cognitive abnormalities that are inconsistent with developmental level and cannot be explained by familial background or environment alone, such as learning difficulties; deficits in school

performance; poor impulse control; problems in social perception; deficits in higher level receptive and expressive language; poor capacity for abstraction or metacognition; specific deficits in mathematical skills; or problems in memory, attention, or judgment

TABLE 1 (continued)

Alcohol-Related Effects

Clinical conditions in which there is a history of maternal alcohol exposure,a,b and where clinical or animal research has linked maternal alcohol ingestion to an observed outcome. There are two categories, which may co-occur. If both diagnoses are present, then both diagnoses should be rendered:

4. Alcohol-related birth defects (ARBD)

List of congenital anomalies, including malformations and dysplasias

Cardiac Atrial septal defects Aberrant great vessels

Ventricular septal defects Tetralogy of Fallot

Skeletal Hypoplastic nails Clinodactyly

Shortened fifth digits Pectus excavatum and carinatum

Radiouinar synostosis Klippel-Feil syndrome

Flexion contractures Hemivertebrae

Camptodactyly Scoliosis

Renal Aplastic, dysplastic, Ureteral duplications

hypoplastic kidneys Hydronephrosis

Horseshoe kidneys

Ocular Strabismus Refractive problems 2ndary **to** small

Retinal vascular anomalies globes

Auditory Conductive hearing **loss** Neurosensory hearing loss

Other Virtually every malformation has been described in some patient with FAS. The etiologic **specificity** of most of these anomalies to alcohol teratogenesis remains uncertain.

5. Alcohol-related neurodevelopmental disorder (ARND)

Presence of:

A. Evidence of CNS neurodevelopmental abnormalities, as in any one of the following:

- decreased cranial size at birth

- structural brain abnormalities (e.g., microcephaly, partial or complete agenesis of the corpus callosum, cerebellar hypoplasia)

-neurological hard or soft signs (as age appropriate), such as impaired fine motor skills, neurosensory hearing loss, poor tandem gait, poor eye-hand coordination

and/or:

B**.** Evidence of a complex pattern of behavior or cognitive abnormalities that are inconsistent with developmental level and cannot be explained by familial background or environment alone, such as learning difficulties; deficits in school performance; poor impulse control; problems in social perception; deficits in higher level receptive and expressive language; poor capacity for abstraction or metacognition; specific deficits in mathematical skills; or problems in memory, attention, or judgment.

Copied with permission from: Institute of Medicine. *Fetal Alcohol Syndrome: Diagnosis, Epidemiology, Prevention, and Treatment.* Washington, DC: National Academy Press, 1996.

Diagnosis of FAS in a newborn can be particularly challenging for several reasons. Because CNS dysfunction, which is a hallmark of FAS, may not be detected until several years after birth, a clinician often relies primarily on identifying the syndrome's characteristic facial features. These features, however, can be quite subtle and thus particularly difficult to recognize in the neonate. The fact that some of the distinguishing features (specifically, midfacial under-development) can occur normally in many newborns (Aase 1994) and that normal swelling around the eyes in newborn infants often obscures the characteristic anomalies further complicates diagnosis within this age group (Sokol and Clarren 1989). It is not surprising, then, that FAS is diagnosed readily at birth in only the most severely affected children and that many FAS cases go undetected at this time (Abel and Sokol 1987; Little et al. 1990; Sokol and Clarren 1989).

In older children and in adults, diagnosis also is difficult because some of the representative features become less distinctive with age. The onset of adolescence often brings a change to the characteristic slender build of children with FAS, particularly in girls (Streissguth et al. 1991). Facial appearance begins to normalize with age as continued slow growth of the face, chin, and nose through adolescence compensates for underdevelopment of the midface (Streissguth et al. 1991). Although certain characteristic features of FAS can be recognized in severely affected children even in adolescence, FAS becomes more difficult to diagnose in children with mild expression of the syndrome (Spohr et al. 1993). Because adult height and head circumference usually remain below normal, however, and abnormalities of the eyes and upper lip are seen in 80 percent of adolescents and adults with FAS, these features can be valuable for diagnosing older persons Streissguth et al. 1991).

Normal variations of particular facial features in certain racial groups (Abel and Sokol 1991; Ernhart et al. 1989; May 199 1; Sokol et al. 1986) and in particular families (Aase in press) also can influence diagnosis. For example, a moderate degree of midfacial underdevelopment is a normal feature in many Native American groups. Broader lips normally seen in African-American children may mask the thin upper lip that is seen in FAS, and the characteristic tall stature of some northern European and central African populations may obscure FAS-related growth deficiency (Aase 1994). Similarly, such family traits as IQ, height, facial features, and even creases on the palm (Streissguth et al. 1991) may be heavily influenced by heredity and can either mask or mimic the features of FAS (Aase 1994). These variations can complicate diagnostic efforts and should be considered in the diagnostic process.

Given the challenges that face diagnosticians, Abel et al. (1993) designed a study to determine whether medical providers (obstetricians and pediatricians) and biomedical researchers not formally trained in dysmorphology could accurately and consistently identify in facial photographs those infants who had and did not have FAS. Study participants in both groups generally were able to accurately identify FAS on the basis of photographs alone. Moreover, identification of FAS was highly correlated with maternal drinking behavior, thus underscoring that facial features and maternal drinking are associated. With the provision of additional information to the participants, such as birth weight, the accuracy of identification increased only among biomedical scientists. This finding suggests that the clinicians were less influenced in their evaluations by supplementary diagnostic information. The race of the photographed children, however, influenced the accuracy of identification ratings within occupational groups. African American children were more likely than Caucasian children to be incorrectly classified as having FAS. Because all but one of the study participants were Caucasian, this bias implies that lack of knowledge of normal African-American features can influence the accuracy of diagnosis. The authors noted that the findings should be considered in light of a potential shortcoming of the study. Only photographs of children with FAS and healthy children were used. Thus, the study confirms that the participants could distinguish between normal and unusual-looking children but not necessarily between children with FAS and children with other birth defects (Abel et al. 1993).

To date, FAS is more commonly identified by an overall pattern of facial features than by specific individual facial characteristics (Astley et al. 1992; Clarren et al. 1987; Rostand et al. 1990). However, clinicians most frequently use the occurrence of small eye openings, smooth and long philtrum, thin upper vermilion (i.e., narrow red margin of the upper lip), increased inner canthal distance, and an elongated midface to diagnose FAS in infants and older children. An approach that employs a weighted checklist of distinguishing characteristics for FAS (Aase 1994; Smith et al. 1990; Sokol et al. 1986; Vitez et al. 1984) has proved to be of value in research and in "clinical screening" (increasing the number of appropriate referrals to diagnostic clinics), but it appears to be less effective as a diagnostic tool for clinical purposes (Aase 1994).

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FAS Physical Abnormalities

Studies by Prof.Dr.med. Hermann Löser from the University Childrens Clinic, Münster, Germany. He has followed hundreds of FAS children for over 20 years. His results are in "Ratgeber zur Alkoholembryopathie" published by Lambertus Verlag Freiberg.

98% are under normal height and weight
84% Microcephalic
89% Mental and Motor Retardation
80% Speech impediments
20% Hearing problems
20% Swallowing/Feeding problems
72% Hyperactive
58% Slack muscles
20% Autism/Aggressive/Social Problems
95% Facial anomalies
29% Heart defects
10% Kidney defects
46% Genital deformities
25% Eye problems
16% Bent crooked little finger
51% Shortened and bent little finger
13% Underdeveloped fingers
9% Hip deformaties
16% Small teeth
30% Pidgeon Chest
7% Concave chest
7% Cleft palate
44% Spinal dimple
12% Hernia
35% Hairgrowth on back of neck

Fact Sheet:
Women and Alcohol

Email the Alcohol Policies Project

Alcohol Use

- 77.6% of women age 12 and older reported ever using alcohol, while 60% reported past year use and 45.1% reported using alcohol in the past month.1
- 82.5% of white women reported ever using alcohol, while 65% reported past year use and 49.7% reported using alcohol in the past month.1
- 67.9% of black women reported ever using alcohol, while 45.1% reported past year use and 32.3% reported using alcohol in the past month.1
- 60.8% of Hispanic women reported ever using alcohol, while 48.4% reported past year use and 33.6% reported using alcohol in the past month.1
- Among current female drinkers, 7.16% of whites, 10.22% of blacks, 22.16% of American Indians/Alaska Native, and 9.03% of Hispanics reported alcohol dependence.2
- Men and women reported different levels of alcohol involvement. 58.7% of men age 12 and older reported past month alcohol use compared to 45.1% of women, while 23.2% of men age 12 and older reported binge drinking in the past month compared to 8.6% of women.3

Health

- Women absorb and metabolize alcohol differently than men.4
- Although the mean lifetime dose of alcohol in female alcoholics is only 60% of that in male alcoholics, one study noted that cardiomyopathy (a degenerative disease of the heart muscle) and myopathy (a degenerative disease of skeletal muscle) was as common in female alcoholics as in males. The study concluded that women are more susceptible than men to the toxic effects of alcohol on the heart muscle.5
- Brain shrinkage in men and women was found to be similar despite significantly shorter periods of alcohol exposure or drinking histories in women.6
- Women with chronic pancreatitis have shorter drinking histories than that of men. Women with alcoholic hepatitis and cirrhosis were found to have consumed less alcohol per body weight per day than men. These findings

indicate that women are more vulnerable to alcoholic liver disease than men.7

- Although alcohol problems are more common in male trauma patients, women with alcohol problems are just as severely impaired, have at least as many adverse consequences of alcohol use, and have more evidence of alcohol-related physical and psychological harm.8

Suicide

- One study showed that 40% of alcoholic women attempted to commit suicide, compared to 8.8% of non-alcoholic women.9
- Younger women who are alcoholics are nearly twice as likely to attempt to commit suicide (50.5%) than older women who are alcoholics (25.5%).9
- A study of suicides among females in New Mexico found that 65.5% of the decedents had alcohol or drugs present in their blood at the time of autopsy.10

Use During Pregnancy

- Since 1990 the *Dietary Guidelines for Americans* have stated that women who are pregnant or planning to become pregnant should not drink alcohol.
- A national survey found that 58.8% of women age 15-44 drank while pregnant.11
- 65.8% of pregnant women in their first trimester reported using alcohol, while 56.6% of women in their second trimester and 53.9% of women in their third trimester reported alcohol use.11

Victimization12

- 57% of female victims of intimate violence (i.e., current or former spouses, boyfriends, etc.) reported that the offender had been drinking at the time of the offense.
- 62% of female victims of alcohol-related violence reported experiencing some form of injury.

Criminal Behavior[13]

- An estimated 4 in 10 women committing violence were perceived by the victim as being under the influence of alcohol and/or drugs at the time of the crime.
- An estimated 25% of women on probation, 29% of women in local jails, 29% of women in state prisons, and 15% of women in federal prisons had been consuming alcohol at the time of the offense.

Drinking and Driving[14]

- Women are less likely than men to be involved in fatal alcohol-related crashes. However, from 1977 to 1997 the number of male drivers involved in alcohol-related fatal traffic crashes decreased 31%, while the number of females drivers involved in alcohol-related fatal crashes has increased 12%.

Moderate Drinking[15]

- Moderation is defined as no more than one drink per day for women.
- One drink is 12 ounces of regular beer, 5 ounces of wine, and 1.5 ounces of 80-proof distilled spirits.

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NEWS

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Researchers Identify Damage Mechanism in Fetal Alcohol Syndrome

St. Louis, Feb. 11, 2000 – For years, physicians and scientists have known that alcohol has detrimental effects on the human fetus. A new study from investigators in Berlin, Tokyo and St. Louis identifies how the damage associated with fetal alcohol syndrome might occur.

A paper in today's issue of *Science* reports that a single exposure to high levels of ethanol (the alcohol in beer, wine and spirits) can kill nerve cells in the developing brain. The researchers found that the rat brain is sensitive to this toxic effect during a brain development stage that corresponds to the brain growth spurt in humans. Called synaptogenesis because it is the time when brain cells form most of their interconnections, the brain growth spurt lasts from about the sixth month of pregnancy to a child's second birthday. In rats, synaptogenesis occurs after a pup is born.

The scientists intoxicated infant rats by giving them ethanol for periods of four or more hours. This maintained the alcohol level at about 0.2 (200 mg ethanol per deciliter of blood) — about twice the level that defines legal intoxication in humans. This one-time exposure caused brain cells to commit suicide by a process called apoptosis or programmed cell death. The rate of cell death exceeded the spontaneous rate of cell death by almost 30 times in some parts of the brain — spontaneous cell death removes surplus cells from the developing brain.

The investigators say finding that cells can die after a single episode of alcohol intoxication means it would be prudent for expectant mothers to avoid alcohol intoxication during pregnancy.

"For many years, scientists studying fetal alcohol syndrome have tended to expose rats to alcohol for longer periods of time rather than studying the damage more transient

exposure might cause," said John W. Olney, M.D., the study's senior investigator and the John P. Feighner Professor of Neuropsychopharmacology at Washington University School of Medicine in St. Louis. "We exposed the infant rats just once, keeping them intoxicated for a period of just a few hours, and we found that was sufficient to trigger considerable damage in the developing brain."

The paper's lead author, Chrysanthy Ikonomidou, M.D., associate professor of pediatric neurology at Humboldt University in Berlin, previously was a postdoctoral fellow in Olney's laboratory, as was another author, Masahiko J. Ishimaru, M.D., Ph.D., who now is based at the Tokyo Medical and Dental University. The three collaborated with other colleagues at their institutions to conduct the study. The researchers found no evidence that exposure to small amounts of alcohol had cumulative effects on the developing brain. Rather, substantial intoxication was required before significant damage occurred. While translating effects from rats to humans is difficult, Olney believes it is unlikely that a single glass of wine would cause the damage observed in these experiments, even if expectant mothers consumed a very small amount of alcohol every day. Because it is not entirely clear how rats and humans compare in sensitivity to alcohol, however, the investigators believe it is best to avoid alcoholic drinks completely during pregnancy.

The investigators also studied the mechanism of this alcohol-induced brain cell death. It is known that alcohol can interfere with certain transmitter systems in the brain. The systems use chemical molecules, such as glutamate and GABA, to activate nerve cell receptors and transmit messages from one cell to another. In research reported last year in *Science*, Olney and colleagues found that drugs called NMDA antagonists, which interfere with glutamate transmission in the same way that alcohol does, have a similar cell-killing effect in the infant rat brain when given as a single high dose. In the current study, the investigators found that drugs that excessively activate GABA receptors, as alcohol does, also can kill nerve cells in the infant rat brain.

"Our evidence documents that alcohol acts by two mechanisms — blockade of glutamate transmission and excessive stimulation of GABA transmission. By combining these two mechanisms, it produces a compound pattern of damage that is greater than either mechanism would produce by itself," Olney said.

Much of the significance of the findings comes from the fact that alcohol is so widely used throughout the world. **"However, it must be recognized that numerous other drugs act either by blocking glutamate receptors or activating GABA receptors, and many of these drugs are drugs of abuse and/or are used in pediatric medicine as sedatives, anticonvulsants or anesthetics," Olney said. "In fact, the only drugs available for anesthetizing human infants act either by blocking NMDA receptors or activating GABA receptors."**

Drugs of abuse that block NMDA glutamate receptors include phencyclidine (PCP or "angel dust"), ketamine (special "K") and nitrous oxide (laughing gas). Both ketamine and nitrous oxide are used frequently in pediatric anesthesia. GABA receptor activators

that are frequently abused and/or used in pediatric anesthesia include benzodiazepines, barbiturates, isoflurane and propofol.

"In light of this new evidence, it obviously is prudent for expectant mothers to avoid any of these drugs," Olney said. "It also will be important to carefully reevaluate how these drugs are used in pediatric medicine with an aim toward developing guidelines that ensure an adequate margin of safety."

The death of neurons by apoptosis occurs naturally. It enables the brain to get rid of unhealthy cells or cells that are not needed for normal brain development. "But what we saw was cell death at many times the normal rate," Ikonomidou explained. "And alcohol and these other drugs don't just cause cells that are going to die anyway to die more quickly. They cause cells that never would have died under normal circumstances to commit suicide. And millions are involved."

These mechanisms may contribute to the wide variety of neurological and psychiatric symptoms seen in individuals with fetal alcohol syndrome. Symptoms range from hyperactivity and learning disabilities in childhood to depression or severe psychosis in adulthood. Olney believes the variety of symptoms may be explained by the timing of alcohol exposure. In the rat, he found that different populations of neurons were vulnerable at different times during synaptogenesis.

"So if the toxic event occurs early in synaptogenesis, it will delete groups of neurons that develop sensitivity early. If it occurs later, those neurons will be spared, but other groups will be deleted," Olney explained.

Olney and Ikonomidou will continue to investigate the impact of alcohol and drug exposure on the developing brain by studying rats as the animals mature. They will determine which specific brain damage patterns are associated with specific behavioral and neurological problems that develop later in life.

"Many psychiatric and neurological disorders are thought to originate from events that occur during development," Olney explained. "This model will allow us to establish some correlations between damage to specific cell populations during development and subsequent neuropsychiatric problems."

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The full-time and volunteer faculty of Washington University School of Medicine are the physicians and surgeons of Barnes-Jewish and St. Louis Children's hospitals. The School of Medicine is one of the leading medical research, teaching and patient care institutions in the nation. Through its affiliations with Barnes-Jewish and St. Louis Children's hospitals, the School of Medicine is linked to BJC Health System.

FAS/E grossly under-diagnosed and under-reported
Bruce Ritchie

Response to a query on FASlink.

The current rates of FAS/E are grossly under-diagnosed and under-reported. There are many reasons for this.

1. Physicians do not receive training in FAS diagnosis at medical school, with very few exceptions. It is not part of the curriculum, yet. This is obvious from the difficulties we have in referring people to doctors who are trained to diagnose FAS. If physicians are not trained to diagnose FAS they will attach other labels to symptoms. Often the prescribed treatment will be inappropriate and the prognosis will be poor.

2. Studies on addiction in Ontario have shown about a 10 - 12% alcohol addiction rate among adults, with another 20% drinking to a level that places them at high risk.

3. Doctors usually do not screen their patients for alcohol use and avoid broaching that issue with patients of child-bearing age.

(a) They are often uncomfortable with the issue themselves.

(b) They received very little training on the entire subject of alcohol in medical school. According to my son's birth mother, a family physician, her entire training on the subject of alcohol was less than 1⁄2 day in medical school. Nothing on the effects of prenatal alcohol exposure and nothing on diagnosing FAS/E.

(c) Physicians should be routinely using urine alcohol test strips to screen for alcohol use just as they do for glucose, pH, keytones, protein, etc. Yet they are often resistant to the concept. Many even avoid asking non-threatening questions about alcohol use during regular physicals.

(d) Some physicians also have substance abuse problems. Doctors who treat their own problems chemically will also tend to treat their patients problems chemically. My son's birth mother, a family physician, died at age 41 from advanced alcoholism. She had been through many treatment programs

(including Doctors on Chemicals, Alcoholics Anonymous and several major treatment centres).

(e) For years, some doctors prescribed "alcohol drips" to delay onset of labour. This usually happened in the third trimester. A current study reports that a single exposure to high levels of ethanol (the alcohol in beer, wine and spirits) can kill nerve cells in the developing brain. This exposure causes brain cells to commit suicide by a process called apoptosis or programmed cell death.

4. With any condition, doctors generally deal with the more extreme (obvious) cases and with FAS it is the extreme end of the spectrum that is most likely to be diagnosed, if at all. Yet research has not found a safe level of alcohol exposure during pregnancy and we have seen that the damage is a continuum, rather than a threshold condition. So likely most affected children are dealing with reduced skills, talents, IQ, AQ, and have more mental health, physical and learning issues than necessary. Most damage is undiagnosed, but not unpunished. If it is undiagnosed it is not part of the statistics.

5. In our middle-income neighbourhood public school, 10% of the students have been formally “identified” as having serious learning and/or behavioural disabilities. Another 5% require “identification”. My son, David, was diagnosed with FAS shortly after he was born. He has an IEP (Individual Education Plan) based on a Communications Disability. The other students generally have been diagnosed ADHD. Sure thing. ADHD does not come with the social baggage of an alcohol associated disability. Unfortunately, it also means the kids don't get the proper intervention. Many other schools in other areas have far higher rates of IPRC “identification”. Some other school districts discourage “identification” because they would have to provide services with little funding backup. The Canadian Centre for Studies for Children at Risk, at McMaster Unversity Medical Centre, states that 20% of Canadian children have a serious mental health problem.

6. Often the woman does not know she is pregnant until the third + month. It only takes a party or two to do damage to the baby. The baby may have been swimming in alcohol at many parties before the pregnancy was known. In some cases, it is much longer. If she stops drinking then, she will often assume no damage has been done. Psychologically, it is extremely difficult for anyone to admit something they did could have seriously harmed their child. That being the case, what are the odds she will discuss the issue with anyone, including her doctor?

7. Massive Denial is a hallmark of alcohol addiction. So those 10 - 12% who are addicted to alcohol might not recognize they have a problem and may not stop drinking. Addiction by definition is the removal of voluntary control. Subsequently they will likely verbally minimize the quantity they drank and will resist future diagnoses that might implicate them as the source of their child's problems. With

some exceptions, couples tend to have similar drinking habits and they tend to socialize with others with similar lifestyles. Sudden abstinance during pregnancy requires the support of the father, families and friends. Yet the pregnant mother is often placed in social situations where she is encouraged to "have just one drink. It won't hurt you or the baby". The social pressure to deny alcohol has affected the child is great.

8. Social condemnation of alcohol problems leads families to conceal the problems. At the higher end of the income scale, it is easier to hide the problem than it is among those in regular contact with social service and health care agencies. Families become enablers and the problem drinking progresses. Again, social baggage attached to the word "alcohol" results in concealment of problems in the children. The concealment leads to failure to properly treat the child and results in development of secondary disabilities. Many receive the label, "Black Sheep".

9. Many FAS/E children are born to multi-generational drinkers. FAS/E children are having FAS/E babies. Inability to predict consequences, impulsivity, high potential for alcohol addiction and a hormone driven twitch in the kilt are a dangerous combination. These individuals are less likely to get proper prenatal care, admit to alcohol consumption (let alone accurately admit the quantity and frequency), or maintain proper nutrition during pregnancy. Many of these FAS babies will never be diagnosed.

10. Studies indicate that more than half the prison population was likely exposed to high levels of alcohol prenatally and should be formally diagnosed. 3% of the U.S. population is currently guests of the prison system. Some prisoners are regular repeaters. Some are one-time offenders. What percentage of the population is a guest of the “justice” system at some point during their life? How many were prenatally exposed to alcohol?

11. Canada's Justice cost is roughly $10 billion annually. When I administered a police services computer system, including Occurrence Records, less than 20% of police calls did not involved alcohol or other drugs. If only half the Justice budget were attributable to alcohol, then the cost would be $5 billion per year. Alcohol taxes generate $3.2 billion per year, a $1.8 billion shortfall.

12. It is estimated that perhaps 80% of children in protection by Children's Aid Societies, DFS, etc. are FAS/E. However, diagnosis is often withheld so that FAS/E does not have to be disclosed in adoption proceedings. If they get a diagnosis, they have to disclose or face lawsuits. If they do disclose it could reduce the number of potential adoptive parents for that child. If they don't get a diagnosis, they don't have to disclose and the child could be easier to adopt out. Unfortunately, it also means the proper interventions are not done and the child and family deal with years of frustration and heartache. In my opinion, failure to

diagnose borders on criminal negligence. Foster parents have been threatened with dismissal if they take a ward in for diagnosis.

13. Governments have failed to recognize the gravity of the situation and only provide the tiniest of token funding. In Canada, the federal government announced $11 million (Canadian funds) ($3 Cdn = $2 US) for FAS over 3 years. Virtually none of it has made it to the grassroots where the battles are being fought. At $3 million expected lifetime cost to the taxpayers per FAS/E individual, David's birth mother's 4 FAS/E children wiped out the Canadian national FAS budget.

14. Massive Denial among governments. If the true extent of prenatal alcohol related damage were “known”, the cost required to address the issues would be massive. In some cases, the true extent of FAS/E, if recognized, would bring “shame” to the village and is officially minimized or ignored. In some cases, the leaders themselves could be alcohol affected, an added incentive for denial. In any case, governments are themselves addicted to alcohol tax revenue. They see booze and gambling as the geese that lay the golden eggs, as long as they can ignore the social fallout.

15. The beverage alcohol industry has a very powerful advertising program, showing alcohol as a healthy part of the lifestyle of youth. Attacking such fun and games would paint one as a “spoil sport”, anal retentive and otherwise general poop.

16. The beverage alcohol industry has the same level of morality as the tobacco industry. Any attempt to control them is met with well financed attacks. They finance tainted research to promote the benefits of their product. (Most Australian wineries are owned by doctors). They finance organizations purporting to deal with the results of alcohol. If anyone goes against the party line, they kill the funding. Some politicians receive significant financial support from booze related sources. It costs money to get elected. Who says you can't buy votes?

17. Independent, data collecting systems have not been established and properly financed to identify the extent of the problem. The truth about FAS would be politically and financially embarrassing.

All of the above, and more, work together to grossly under-report and under-estimate the incidence of damage caused by pre-natal exposure to alcohol. The effects of prenatal alcohol exposure are pervasive throughout society. In human and financial terms, the cost is astronomical and rising by the minute.