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Fetal Alcohol Syndrome: Implications for the Irish Care System

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Authors Note. Much of the research material was sourced in the U.S.A. Therefore, for reasons of simplicity and in line with academic protocol the original, 'Americanized,' spelling has been retained.

Introduction

Birth defects can result from (a) an abnormal or mutant gene, (b) an infection, or (c) a drug. Today the field of speciality known as teratology investigates birth defects and the substances or teratogens that cause them. Since the thalidomide tragedy we have become acutely aware that the unborn child in the uterus is extremely sensitive to substances ingested by the mother. One such substance is alcohol. There is now a large body of evidence to indicate that maternal consumption of alcohol leads to a broad spectrum of birth defects. This resulting set of defects is known collectively as the Fetal Alcohol Syndrome. The purpose of this paper is to review the literature on Fetal Alcohol Syndrome and to discuss the implications for victims, care givers, social workers, policy makers and educationalists.

Fetal Alcohol Syndrome

Medical Definition

A syndrome of impaired fetal growth and development associated with a high maternal intake of ethanol during pregnancy. The fetus appears to be most vulnerable to the deleterious effects of alcohol immediately following the time of conception before the mother knows she is pregnant. Features of the syndrome include growth retardation, microcephaly, mental abnormality and a characteristic combination of craniofacial deformities. Other malformations, such as neuraltube defects, may also occur (Walton, J. et al 1986).

Mosby's Dictionary (1994) States:

Fetal Alcohol Syndrome (FAS), a set of congenital psychological, behavioural, cognitive and physical abnormalities that tend to appear in infants whose mothers consumed alcoholic beverages during pregnancy. It is characterised by typical craniofacial and limb defects, cardiovascular defects, intrauterine growth retardation and retarded development. The most serious cases have involved infants born to mothers who were chronic alcoholics and drank heavily during pregnancy. Women who drank less, reportedly gave birth to infants with less serious malformations or Fetal Alcohol Effects (FAE) but it is not known if there is a lower limit to alcohol consumption during pregnancy or if there is a particular period in embryonic life when the offspring is most vulnerable to effects of alcohol.

Historical Review

The current interest in the relationship between maternal alcohol consumption and birth defects is not a new phenomenon. On a point of information: "In classic times the consumption of alcohol by newly-weds in Carthage and Sparta was prohibited by law to prevent conception while "under the influence" (Rosett and Sander cited in Osofsky 1979).

In 1726 the College of Physicians attempted to halt the "gin epidemic" by petitioning the English Parliament to pass a law controlling the manufacture and sale of cheap gin. The belief was that this step would prevent the birth of "weak, feeble and distempered children" (Morris cited in Osofsky).

In more recent times reports from Lemoine et al 1968; Ulleland 1970; Jones and Smith 1973; in America, led to renewed interest. The reports from Seattle in the early '70s led to many further case reports from around the world including one from Barry and O'Nuallian 1975 in Cork.

Diagnostic Criteria of Fetal Alcohol Syndrome and Fetal Alcohol Effects

The criteria for diagnosis was developed in 1980 by the Fetal Alcohol Study Group of the Research Society on Alcoholism, and was outlined by Cooper (1986).

- I. Prenatal and/or postnatal growth retardation: weight, length and/or head circumference below the tenth percentile when corrected for gestational age;

2. Central nervous system involvement; signs of neurological abnormality, developmental delay or intellectual impairment;
3. Characteristic facial dysmorphism with at least two of these signs:
 - (a) Microcephaly (head circumference below the third percentile);
 - (b) Micro-ophthalmia and/or short palpebral fissures; poorly developed philtrum (the medium groove between the upper lip and nose), thin upper lip or flattening of the maxillary area.

If all three of these characteristics are present a diagnosis of Fetal Alcohol Syndrome can be made. If some but not all characteristics are present then a diagnosis of Fetal Alcohol Effects can be made. This diagnosis acknowledges that the effects of alcohol on the fetus can occur along a continuum depending on the amount of alcohol consumed and the timing of exposure.

Non-Detection or Mis-Diagnosis

A study conducted by (Nanson 1992) found that out of a data base of 326 individuals diagnosed as victims of Fetal Alcohol Syndrome six were discovered to be autistic. This represents an incidence of 1:54. Prior to this study no "Link" between Fetal Alcohol Syndrome and autism had been made. This raises the issues of a diagnosis of Fetal Alcohol Syndrome precluding one of autism particularly as some of this secondary diagnosis was not made for some years. Nanson also suggests that some children diagnosed as autistic may not be investigated for Fetal Alcohol Syndrome.

Major Identifying Characteristics of FAS

Central Nervous System	Mild to moderate mental retardation
Growth	Extremely small size for age
Facial	Small head Jutting forehead Short eye slits Skin folds on inner corner of eye (epicanthal folds) Underdeveloped mid-facial region Short, flat nose Low-set ears Absence of vertical ridges between nose and mouth Thin upper lip

Behavioural	Hyperactivity Distractibility
Miscellaneous	Poor fine-motor ability Speech and language defects

Genetic Predisposition

One of the difficulties around the area of drinking during pregnancy is that there is no clear indicator of how much, if any, alcohol is safe e.g. Even allowing for the many variables such as diet, maternal weight gain, smoking etc., it is still unclear as to why some children born to chronic alcoholic mothers display only partial symptoms or effects while others display all the characteristics associated with full Fetal Alcohol Syndrome. One possibility is that of genetic predisposition or susceptibility.

Chasnoff (1984) reported on a case involving dizygotic twins. The twin girls aged 18 months were taken into care as a result of having been hospitalised due to multiple injuries inflicted through abuse. Subsequent to their admission a diagnosis of fetal alcohol exposure was made. Prior to their birth at 36 weeks gestation their mother, a 20-year-old primagravida, had been drinking three times per week. She denied any other drug use, but smoked 10 cigarettes per day.

The results of examination at the time of hospitalisation revealed the following facts.

	Weight	Height	Head Circumference
TWIN A:	7,900g	74cm	45cm

All of these parameters fall well below the third percentile for age. Twin A also displayed the facial abnormalities typical of Fetal Alcohol Syndrome with "small short eyes, prominent epicanthal folds, short upturned nose with a long flat philtrum, thin upper lip, small mouth and underdeveloped mandible". This twin also scored well below the norm when tested on the Bayley Scales of Infant Development.

	Weight	Height	Head Circumference
TWIN B:	9,500g	81cm	46.5cm

All of the above growth parameters were within the normal range for age. Twin B

displayed some of the facial characteristics of the syndrome e.g. "small short eyes, prominent epicanthal folds, short upturned nose with a long flat philtrum, thin upper lip, and small mouth". The mandible was well developed. However, developmental testing indicated that this twin also fell well short of the normal range for age in terms of cognitive functioning.

The results of these tests led to a diagnosis of Fetal Alcohol Syndrome in Twin A and Fetal Alcohol Effects in Twin B. Over the next three years the growth development of both twins was monitored. It became clear from the chart that Twin A had consistently fallen below the third percentile, while Twin B had maintained a growth rate within the normal range (Chasnoff 1984).

Nature Vs Nurture

At age 18 months the twins were placed in permanent substitute care i.e. adoption. By 24 months they had both recorded a marked improvement on the Bayley Scale and at age 4 years Twin A had an I.Q. rating of 110 while Twin B had a rating of 118, well within the normal range (Ibid.)

Toxicity of Alcohol - Can a casual relationship be established between maternal consumption of alcohol and specific birth defects?

Reports from various case studies over the past 20 years (Crain, Fitzmaurice & Mondry, 1983; Jones & Smith 1973) and animal studies (Brown, Goulding & Fabio, 1979) have all provided evidence to support the conclusion that alcohol is a teratogen. This evidence also supports the conclusion that alcohol is most likely the major causative agent in producing those characteristics that collectively lead to a diagnosis of Fetal Alcohol Syndrome.

However, not everybody supports the view that alcohol is the sole contributory agent responsible for producing these characteristics. In a letter published in the *Lancet* (Dec 6th, 1986, pg 1337) Livingston and Hermoine express the following reservations.

Since Fetal Alcohol Syndrome was described by Jones et al in 1973 it has become clear that the clinical features are not specific to exposure to alcohol during pregnancy. Animal studies suggest that any teratogen exposure during [gestation] may result in the craniofacial, brain, and eye defects corresponding to those in severe Fetal Alcohol Syndrome. Hingson and colleagues found that several other factors, such as maternal marijuana smoking, exposure to x-rays, and poor maternal weight gain

during pregnancy, were strongly associated with features compatible with Fetal Alcohol Syndrome. It has thus been suggested that the dysmorphology of Fetal Alcohol Syndrome may represent a common pathway of numerous agents, or a combination of agents rather than a specific teratogenic effect of alcohol (Livingston, J & Hermoine, L, 1986).

In 1982 the results of a study carried out by (Astley et al 1982) into the association between fetal exposure or marijuana and alcohol and the distinguishing features of Fetal Alcohol Syndrome were published.

In this study a series of standardised side and front facial photography's were taken of forty children aged between 5 and 7 years of age whose mothers had frequently used marijuana during the first trimester of pregnancy and forty mothers who had not used marijuana during pregnancy. All eighty children were group matched for alcohol exposure during pregnancy.

The photograph's were then examined by a dymorphologist and by computerised landmark analysis. The findings indicated that among the group exposed to marijuana prenatally no consistent pattern of facial features was evident. However in the group where maternal consumption of alcohol was two or more ozs per day during early pregnancy a clear pattern of facial anomalies associated with Fetal Alcohol Syndrome was found. (Astley et al 1992).

Summary of Effects of Prenatal Substance Use

Outcome								
Animal Studies					Human Studies			
Drug used	Key	Pregnancy Outcome	Fetal growth	Offspring effects	Congenital Anomalies	Pregnancy Outcome	Fetal growth	Postnatal problems
Cigarettes	=	-	+	+	=	-	-	-
Alcohol	+	+	+	+	+	+	+	+
Marijuana	=	-	+	+	-	=	=	=
Opiates	=	=	+	+	-	=	+	+
Cocaine	=	=	=	+	=	=	+	=

- + = outcome occurs
- = outcome does not occur
- = = no consensus on outcome can be drawn.

The above table clearly indicates that alcohol abuse results in a deleterious outcome across all categories.

How much Alcohol if any is Safe and When

Hawkins (1987) states that a quantity of alcoholic drinks equivalent to 30ml of ethanol consumed on a daily basis "*has been considered to place the fetus at risk*" (pg 188).

Quantities of Beverage Containing 30ml of Ethanol

White Wine	300ml	2.5 wine glasses
Red Wine	250ml	2 wine glasses
Fortified Wine	150ml	2 sherry glasses
Spirits	75ml	4 measures
Beer	1000ml	2 pints

He also suggests that while "*... No safe lower limit of alcohol intake in pregnancy has been established it seems likely that there is little risk of the baby being significantly affected if the average daily intake is less than 30ml of ethanol and there are no episodes of intoxication from "binge drinking"*" (Hawkins 1987 pp 192-3).

The critical period in relation to defects depends upon the particular fetal structures that are developing during the time of exposure e.g. the brain which is constantly developing throughout the period of gestation and is therefore virtually "at risk" during the entire pregnancy.

Fetal Reaction to Alcohol

Smotherman et al (1986) conducted a study to observe fetal behaviour after exposure to alcohol. In the study pregnant rats were intubated with varying levels of ethanol in saline solution. After a period of four hours fetal behaviour was observed and recorded. As a result of this study he concludes:

Circulating levels of alcohol in maternal blood, fetal homogenate and amniotic fluid at the time fetuses were observed which confirmed that fetuses were exposed to alcohol in utero but the measured concentrations of alcohol were not predictive of fetal activity. We suggest that some of the developmental consequences of Fetal Alcohol Syndrome may be the consequence of fetal inactivity induced by alcohol in the utero (Smotherman et al 1986 pg 165).

Animal Studies

Many animal studies have indicated that binge drinking during critical periods of prenatal development can cause severe abnormalities in the fetus. One such study conducted by Sulik et al (1981) at the University of North Carolina in which a pregnant mouse was exposed to alcohol had startling results. The exposed fetus had suffered eye damage, stunted brain and facial abnormalities, which were easily identifiable. These defects are similar to the facial abnormalities associated with Fetal Alcohol Syndrome in human babies.

The blood alcohol level reached during the experiment would be similar to that in a woman of average size if she had consumed a quart of Vodka in a 24 hour period.

Extent of the Problem

Estimates as to the extent of Fetal Alcohol Syndrome have varied over the past twenty years e.g. Hawkins (1987) states what while there is a risk, it is small and should not be a cause for undue alarm. The Centre for Disease Control (1984), (cited in Salkinn 1990) puts the figure at one in every 750 live births. More recently however in a letter published in the Journal of the American Academy of Child and Adolescent Psychiatry (1992 p563), Dr. Leslie Atkinson acknowledges that Fetal Alcohol Syndrome is now "...the leading known cause of mental retardation in the United States". In the same Journal, Streissguth et al stated that, "We have been concerned about the discrepancy between the small number of reports in the scientific literature on groups of children with Fetal Alcohol Syndrome, especially in comparison with the magnitude of the problem" (Streissguth et al 1992 pp 563-4).

Implications for the Victims

The range of problems and concerns associated with Fetal Alcohol Syndrome and Fetal Alcohol Effects include poor habituation, sleep disturbances, poor sucking response, failure to thrive, delays in walking and talking, delayed toilet training, difficulty following instructions, temper tantrums and disobedience, distractibility and/or hyperactivity.

Habituation:

According to Dworetzky,

One of the simplest forms of learning is habituation, which takes place when a person becomes accustomed to a stimulus because it has been presented repeatedly. (Dworetzky 1991).

For example if you went on holidays to the coast and just as you were drifting off to sleep a "fog horn" sounded outside your hotel, you would probably be startled. This would be a natural response because being startled is an inborn, not a learned response. However, if the noise continued over and over you would become habituated to it and would learn not to react.

In the case of a child with F.A.S. / F.A.E. this process of habituation is impaired and the child never fully adjusts to normal background noise. This failure to habituate is significant according to Trotter.

"Habituation is now considered a primary indication of brain and nervous system functioning and ... Is seen as a good predictor of later intelligence. Habituation, which becomes increasingly acute over the first ten weeks of life, is commonly assessed as a measure of an infants maturity and well being. Infants who have brain damage or have suffered birth traumas such as lack of oxygen, do not habituate well, and may go on to have developmental and learning problems."
(Trotter; In Dworetzky 1991 pg 348).

Sleep Disturbance

During the first month of life an infant sleeps for up to eighteen hours per day, decreasing to about twelve hours per day at the age of two. During this period the brain grows and develops rapidly. While asleep the body slows down, temperature drops and pulse and breathing rates decrease. One interesting exception to this state of reduced activity, particularly in children, is the production of growth hormone, which peaks during sleep (Dworetzky 1991). Poor sleeping patterns have also been associated with a diagnosis of Fetal Alcohol Syndrome / Fetal Alcohol Effects.

Failure to Thrive

An infant with Fetal Alcohol Syndrome / Fetal Alcohol Effects will generally be lighter and shorter at birth. However, unlike other children that experience similar disadvantages, they often fail to make up the loss, irrespective of the quality of their postnatal care. In short children consistently fail to achieve normal physical development milestones.

Delays in Walking and Talking

The study of development is the study of the changes that the human organism goes through over time. It is now recognised that some of these changes are universal across cultures. However, it is also acknowledged that there are individual differences i.e. the

developmental milestones or stages do not occur at exactly the same age for every child. What is apparent from the research is that children with Fetal Alcohol Syndrome / Fetal Alcohol Effects consistently fall behind their peers in terms of development. It is also a fact that a change of environment never fully compensates for the poor start.

Difficulty following Instructions: Temper Tantrums, Disobedience, Distractibility and Hyperactivity.

McCreight (1991) outlined a number of additional behavioural characteristics that have been associated with Fetal Alcohol Syndrome / Fetal Alcohol Effects.

- Poor impulse control.
- Cannot relate behaviour with consequence.
- Poor short term memory.
- Poor personal boundaries.

Irish Foster Carers who shared some of their experiences with me identified many of these characteristics. However, I feel it is important to stress that while many carers with whom I spoke “suspected” that children in their care were victims of Fetal Alcohol Syndrome / Fetal Alcohol Effects not all the children had been diagnosed.

In relation to poor impulse control one carer said “He never stopped to think he would literally jump into a pool with his clothes on” she also spoke about her frustration at having to repeat the same instructions over and over again e.g. “Do not leave the garden” only to find the child two streets away presumably having made his escape over a six foot garden wall. A total lack of awareness in relation to physical danger and the possible results of reckless actions e.g. “... He did not seem to understand that climbing out of his bedroom window (on the first floor) was dangerous”. Another carer spoke about the problem of short term memory or distractibility “.... two hours after sending him to the local shop, I found him playing with friends in the local park...One of my greatest fears is that he would go off with anyone”.

Many of my own observations and those of my partner are of similar occurrences. The usual “Why” questions that all children ask have taken on a different significance. The same “What”, “Why”, or “When” is asked over and over again as apparently the child has forgotten the answer. Simple instructions can be repeated word for word but the child appears incapable of translating the words into actions. Or indeed the shock of seeing a normally affectionate, loving little boy lose all sense of control for the most trivial reason and kick, scream and throw whatever object is near at hand can be frightening. What is even more frustrating is the confusion of the child at this inability

to understand these actions himself. During the period of calm that follows he is always apologetic and invariably asks "why was I bold? I'm a good boy".

In relation to poor personal boundaries he "invades" people's space and tends to be overly intrusive. In a young child this may not be problematic but later on as a teenager it could lead to problems. He also has difficulty understanding that not everyone is "good" and expects everyone to be his friend.

Early Identification

Giunta and Streissguth (1988) states that "... Patients with Fetal Alcohol Syndrome are at a higher than average risk for physical abuse, sexual abuse and neglect. They are frequently raised in a high risk environment by mothers who struggle for sobriety have few resources and little support" (pg 456).

Given that many victims of Fetal Alcohol Syndrome / Fetal Alcohol Effects live in 'dysfunctional' homes the need for early identification is essential. Without this identification the child has little chance of his / her special needs being catered for. In fact those very needs may lead to a situation where the child suffers abuse or neglect at the hands of a mother whose parenting skills are impaired due to alcohol dependency.

Intervention with Birth and / or Foster Parents

Support systems should be established for birth parents to enable them to provide the stable loving home environment needed by the child in order to reach his / her full potential. Where this is not possible a substitute family should be provided. When discussing alternative care for victim's Giunta and Stressguth (1988) state that

.... Foster parents who are calm and low key individuals, secure and comfortable with themselves, and who live stable and predictable lives, have the highest likelihood of success. Busy professionals who live complex and hyperstimulating lives are often discouraged by the slow or erratic progress of some patients with Fetal Alcohol Syndrome and by the children's failure to perform normally when provided with stimulating and nurturing environments. Families who treat the Fetal Alcohol Syndrome child as normally as possible, combining loving acceptance with firm limit setting, seem more satisfied than do those who have high performance expectations (pg 457).

Education of Parents

An essential part of any intervention with birth and / or substitute parents is education in relation to the needs of each individual child, and realistic expectations relating to capabilities.

Development and Health

The medical needs of the individual with Fetal Alcohol Syndrome will vary depending on severity of effects. Therefore provision should be allocated accordingly. In some of the more severe cases victims may have multiple medical needs, given the high occurrence of congenital anomalies. Malformations of the eye, ear, heart defects, cleft lip and palate are common in many cases (Giunta and Streissguth 1988).

Recommendations for Managing Children with F.A.S. / F.A.E

- Early Identification.
- Intervention with birth and/or foster parent
- Education of parents/carers regarding physical and psychosocial needs of an infant or child with F.A.S./F.A.E
- Careful monitoring of physical development and health
- Safe, stable and structured home
- Assignment of a case manager for co-ordination of services and support to parents
- Placement of the child in pre-school.
- Respite care for care-takers

Case Manager / Social Worker

Once a child has been diagnosed as having Fetal Alcohol Syndrome/Fetal Alcohol Effects a "Case Manager" should be responsible for drawing up a long term "care plan" for each individual child relative to their particular needs. The plan would cover areas such as (1) support, e.g. home help, counselling, financial aid and an alternative home where necessary (2) Medical need, the manager would ensure that appropriate medical attention is provided when needed (3) Educational needs, the case manager in

consultation with an educational psychologist would ensure that the child had access to an educational programme that would enable him/her to achieve their maximum potential (4) During the period of transition into adulthood those individuals lacking the capability to support themselves through work should have the opportunity to live in safety and with dignity in sheltered accommodation. They should also have work in a sheltered workshop suitable to their capabilities.

The Role of Educationalists

The educational system has a number of tasks to deal with in relation to Fetal Alcohol Syndrome/Fetal Alcohol effects. Ugent et. al. (1986) discuss the role that the educational psychologist has to play in helping to prevent Fetal Alcohol Syndrome / Fetal Alcohol Effects. They state that:

An important thrust in prevention should be education by the school psychologist at the school and community levels. The school psychologist can obtain and distribute pertinent literature, conduct presentations to school and civic groups and participate in seminars to educate professionals who may come into contact with both prospective mothers and affected children.

They also go on to suggest that the school psychologist should spear head information campaigns. Some of the activities suggested include posting warnings in Liquor stores, doctors' surgeries etc as well as warnings in the media. In relation to warnings, the ABA Journal of March 1988 carried a report on three law-suits taken against seven Liquor Companies in which plaintiffs allege that a warning should have been on the bottles. The law-suit was taken on behalf of a number of families who claim that as a result of consuming alcohol while pregnant the mothers gave birth to children that:

"..... Suffer from impaired growth and mental retardation, facial deformities, hyperactivity, heart defects and defects in the fingers".

(Moss 1988 pg 17).

The primary aim was compensation for the children but using a warning label on alcohol containers would be a welcome side effect. The other major task facing the educational system according to Burgess and Streissguth (1992) is to facilitate the children affected by Fetal Alcohol Syndrome.

They state that:

Like children with other disabilities, those with Fetal Alcohol Syndrome and Fetal Alcohol Effects benefit from early diagnosis and intervention. There is so much to learn and so little time during the school years. When diagnosis is not possible because of the unavailability of trained medical personnel or other reasons, school personnel can still identify (not to be confused with diagnose) children they believe may have been prenatally exposed to alcohol.

... To maximise the effectiveness of educational programmes therefore, it is crucial to begin early to ensure the learning of appropriate, functional skills and to decrease the occurrence of inappropriate behaviours.

(Burgess and Streissguth 1992).

Discussion

Fetal Alcohol Syndrome is arguably one of the major causes of mental retardation in the Western World today with a prevalence of 1 in 750 in the general population. In the case of alcoholic mothers it can be as high as 690 per 1,000. The full syndrome is characterised by (1) Facial anomalies; (2) growth retardation; (3) damage to the central nervous system and (4) various other physical abnormalities. Partial Fetal Alcohol Syndrome or Fetal Alcohol Effects can be harder to diagnose with some children suffering from mild retardation and others with a low I.Q. or one bordering on the normal. The fact that many of these children are born into 'dysfunctional' families results in a disproportionate percentage of them ending up in the care system. Early identification and a stable home environment coupled with appropriate educational programmes can greatly improve the chances of these children achieving their full potential.

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