WERE OUR FOREBEARS AWARE OF PRENATAL ALCOHOL EXPOSURE AND ITS EFFECTS? A REVIEW OF THE HISTORY OF FETAL ALCOHOL SPECTRUM DISORDER

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ABSTRACT

Many historical records have been taken out of context when reviewing the history of prenatal alcohol exposure, and the impacts of these histories on modern-day FASD research have been overestimated. Historical records, as early as biblical times, do suggest at least a working awareness of an interaction between alcohol and reproduction of some kind. Contrary to assertions made in some fetal alcohol research, these records do not suggest an ancient awareness of the deleterious effects of alcohol on the developing fetus. Historical records regarding alcoholism and reproduction need to be interpreted critically, in context, and in consideration of the Zeitgeist, or the Spirit of the Times.

Key words*: Fetal Alcohol Syndrome; history; alcoholism*

The discovery of the effects of prenatal alcohol exposure on the developing fetus is relatively new. In an unpublished thesis in Paris, 1957, Rouquette described malformations in children born to alcoholic mothers and fathers, and noted that maternal alcoholism could be a serious threat to the fetus.[1,2] Eleven years later, Paul Lemoine, also in France, published a clinical description of 127 children born to predominantly alcoholic mothers.[3] He noted physical malformations, developmental delays, and behavioural problems in the "Lemoine kids".[4] In a series of studies in Seattle, Washington five years later, teratologists Kenneth Jones and David Smith described similar characteristics in a handful of children[5,6], and coined the term Fetal Alcohol Syndrome (FAS).[7] These seminal articles brought worldwide attention to the disorder and initiated a new branch of research and clinical practice into the impacts of alcohol on the developing fetus.[2] The research literature has since evolved to capture a broader view of the effects of prenatal alcohol exposure beyond FAS. The term fetal alcohol spectrum disorders (FASD) is now used to describe a wide range of deficits associated with prenatal alcohol exposure.[8]

In addition to work exploring the nature of FASD, research connecting FASD to historical records and events emerged. Jones & Smith, in their second article about the newly discovered disorder, provided a brief look at some historical accounts that apparently alluded to the effects of alcohol consumption during pregnancy.[7] In 1975, only two years after FAS was given its name, Warner & Rosett conducted a comprehensive review of British and American literature that seemed connected to this newly identified disorder.[8] They refer to the findings of Jones and Smith as a rediscovery of the effects of alcohol on the developing fetus, and suggest that the effects of maternal drinking have been suspected for centuries.

History of FASD

According to Davis[10], a biblical passage may be the first reference to the effects of prenatal alcohol exposure. In the Old Testament it is recorded that an angel spoke to the mother of Samson and said, "Thou shalt conceive, and bear a son. Now therefore, beware, I pray thee, and drink not wine nor strong drink" (Judges 13:3-4). At first glance, this passage appears to be an exhortation to avoid alcohol so that the unborn son may not be exposed to its impacts. For this reason, it has been cited in numerous papers as an indication that there was at least a rudimentary knowledge of the effects of prenatal alcohol in biblical times.[2,11] However, Abel[12] noted that in context, the passage may not be referring to the deleterious effects of alcohol on the development of the fetus. A broader look at

the same passages in the thirteenth chapter of Judges provides some important context:

> "Thou shalt conceive, and bear a son. Now therefore beware, I pray thee, and drink not wine nor strong drink, and eat not any unclean thing: For, lo, thou shalt conceive, and bear a son; *and no razor shall come on his head*: for *the child shall be a Nazarite unto God from the womb*." (Judges 13:3-5, italics added)

According to another biblical scripture, "When either man or woman shall separate themselves to vow a vow of a Nazarite, to separate themselves unto the Lord: *He shall separate himself from wine and strong drink*" (Numbers 6:2-3). In addition, for a Nazarite "there shall no razor come upon his head" (Numbers 6:5). This passage seems to correspond with the warning given to Samson's mother, quoted above. Therefore, the exhortation to not consume strong drink was more likely based on the consecration of Samson as a Nazarite "from the womb" than a reference toward the effects of alcohol on the developing fetus.

Aristotle is often quoted as being aware of the effects of prenatal alcohol exposure: "foolish, drunken, or haire-brain women most often bring forth children like unto themselves, morose and languid".[2,9] But there are a couple problems with that assumption. First, this statement seems more indicative of an awareness of the tendency for children to turn out like their parents, rather than of the effects of prenatal alcohol exposure. The term "drunken" is used as a descriptor or characteristic of certain women, along with "foolish" and "haire-brain" (i.e. what they are, not what they do). According to this statement, these women are likely to have children that are also "foolish, drunken, or haire-brain." It does not imply a direct impact of alcohol exposure (drunkenness) on children, but indicates that children are likely to turn out like their parents. Whether through genetic transmission or environmental influence, it is not specified. The second, and more significant, problem with this statement is that it may not have been spoken by Aristotle at all. In his 1621 publication *Anatomy of Melancholy*, Robert Burton cited Aristotle's *Problemata* and attributed this statement to him.[9] However, Abel[13] noted that this statement does not appear in *Problemata*, nor does it appear in Aristotle's other writings. He also explained that similar quotes mentioned by Burton were not attributable to the figures he cited (i.e. Aulus Gellius and Plutarch). Abel asserts that the Aristotelian quote shared by Burton was his synopsis of what the Greek philosopher would have said about the effects of alcohol, not any actual quote shared by Aristotle.

Although Aristotle did not appear to address the effects of prenatal alcohol exposure on fetal development, he did discuss its effects on fertility and gender. Based on the premise that all things were composed of fire and water, Aristotle asserted that men were made of a greater proportion of fire than women, and conversely, women were made of more water than men.[14] Men were considered more intelligent, courageous, strong, healthy, and morally superior because they had more natural heat. Aristotle believed that if the man had consumed too much alcohol that he became cold (i.e. shivers). During coitus, he would be unable to heat the woman's womb, and as a result would conceive a girl.

Abel[13] pointed out fundamental differences between views about alcohol and fertility in antiquity and our present understanding of the teratogenic effects of alcohol. He noted that the focus of ancient Greeks and Romans was on paternal drinking, whereas ours is on maternal drinking. This may have been the case in antiquity because of the ideological superiority of males and a limited understanding of human reproduction. In addition, the Greeks and Romans believed conception to be the period where alcohol had its active effect, whereas we focus on drinking throughout pregnancy. In antiquity, although too much alcohol was thought to be detrimental, moderate drinking was encouraged, whereas presently no amount of alcohol is considered safe for pregnant women. Although there are apparent fundamental flaws in these views in antiquity, they did recognize a relationship of some kind between alcohol consumption and reproduction.

From antiquity until eighteenth century, there appears to be a lag in recorded information relating alcohol to human reproduction, though thinking regarding this topic may have been present. In England, 1720-1750, there was ample opportunity to observe the effects of alcohol on

reproduction. The country lifted restrictions on distilling, and cheap gin flooded the country. England became mired by a "gin epidemic".[9] The resulting social problems led to a movement calling for gin restrictions. For example, Sedgewick stated in 1725 that "half the train of chronological Diseases" afflicting infants were caused "by the Debauchery of the Mother...So that from the whole, the Regulation of the Mother, during her Pregnancy, is an Affair of the highest Moment and Consideration". A year later, College of Physicians called gin "a cause of weak, feeble, and distempered children".[9] These quotes suggest that the mother's conduct generally, and gin specifically, negatively affected children's health. In addition, increased stillbirth and infant mortality was noticed at that time.

William Hogarth's painting *Gin Lane* reflects many of the social concerns related to the gin epidemic, and interest in the painting has resurged since the advent of historical research in FASD. The painting depicts a myriad of social problems, including drunkenness, misery, poverty, starvation, abuse, suicide, and infanticide. The central figure is a syphilitic woman who unknowingly drops her infant as she reaches for snuff. Apart from the obvious social ills described in the painting, some researchers have suggested that it is a testament that an awareness of the effects of prenatal alcohol existed at that time.[15] For example, Rodin[16] suggested that the infant falling from the woman's arms showed abnormal palpebral features, characteristic of FAS. Additional facial features of FAS, however, were not identified for the child, and similar features were not described in the other figures in the painting.[17]

Another painting by Hogarth, *Beer Street*, indicates that an awareness of the effects of prenatal alcohol exposure was not apparent, contrary to the claims of many researchers who hold *Gin Lane* as an important symbol of the history of FASD in eighteenth century England.[17] Themes of *Beer Street* include commerce, wealth, industry, and happiness, and are in sharp contrast with the significant social ills demonstrated in *Gin Lane*. This suggests that Hogarth was not aware of the deleterious effects of prenatal alcohol exposure, but was commenting on the effects of the "vile" gin, contrasting with the "invigorating" beer. Research of the impacts of alcohol and pregnancy began to emerge in North America in the nineteenth century. Benjamin Rush, who lobbied against alcohol use during pregnancy to avoid dependence, was influential as the temperance movement in the United States took shape. In England, Thomas Trotter proposed that alcohol could cause mental deficiency in children, an innovative idea that was not researched until decades after his time.[9] These two figures helped fuel the temperance debate.

The initiation of the temperance movement in the mid-nineteenth century was highly driven by moral and religious attitudes, and resulted in several groups and societies debating the subject, such as the American Temperance Society in 1826. As the temperance movement took shape, an interest in studying the effects of alcohol on human development emerged in scientific literature in Europe and North America, with some studies evaluating the effects of prenatal alcohol exposure. A report for the House of Commons by a committee on drunkenness indicated that children of alcoholic mothers have "a starved, shrivelled, and imperfect look".[9] Journals were formed specific to the temperance movement (i.e. *Journal of Inebriety*), and temperance began to be an accepted moral standard for many Americans. In the early twentieth century, experimental studies of the effects of prenatal alcohol exposure in animals emerged, showing delayed growth, physical malformations, and high mortality[9], similar to results in modern research.

A seminal study at the time of medical temperance was provided by Sullivan.[18] In his research, Sullivan compared 600 children of alcoholic mothers with controls and found higher rates of stillbirths and mortality among them. In addition, Sullivan found that alcohol consumption of fathers and grandparents were not correlated with these effects. When alcoholic mothers who drank in previous pregnancies and had damaged offspring abstained under coercion (such as by imprisonment), their children did not experience the same rates of mortality. These results suggested that maternal alcohol consumption, not paternal, is a cause of stillbirth and infant mortality. The eugenics movement began to take shape at the turn of the twentieth century, about the same time that the medical temperance research was at its peak. Proponents of the eugenics movement suggested that "imbecility"

was genetic and that restricting or eliminating the reproductive practices of "idiots", "imbeciles", and "morons" (three degrees of intellectual disability) would lead to better care and less strain on resources, or in some cases, a purer race. Many eugenists argued that alcohol was a "race poison"[9] that could contribute to the downfall of humanity. However, some Darwinists, such as Archdall Reid, asserted that alcohol was "the cause of an evolution protective against itself"[19], in that the effects of alcohol use through natural selection would offset the hereditary propensity for alcohol as a whole.

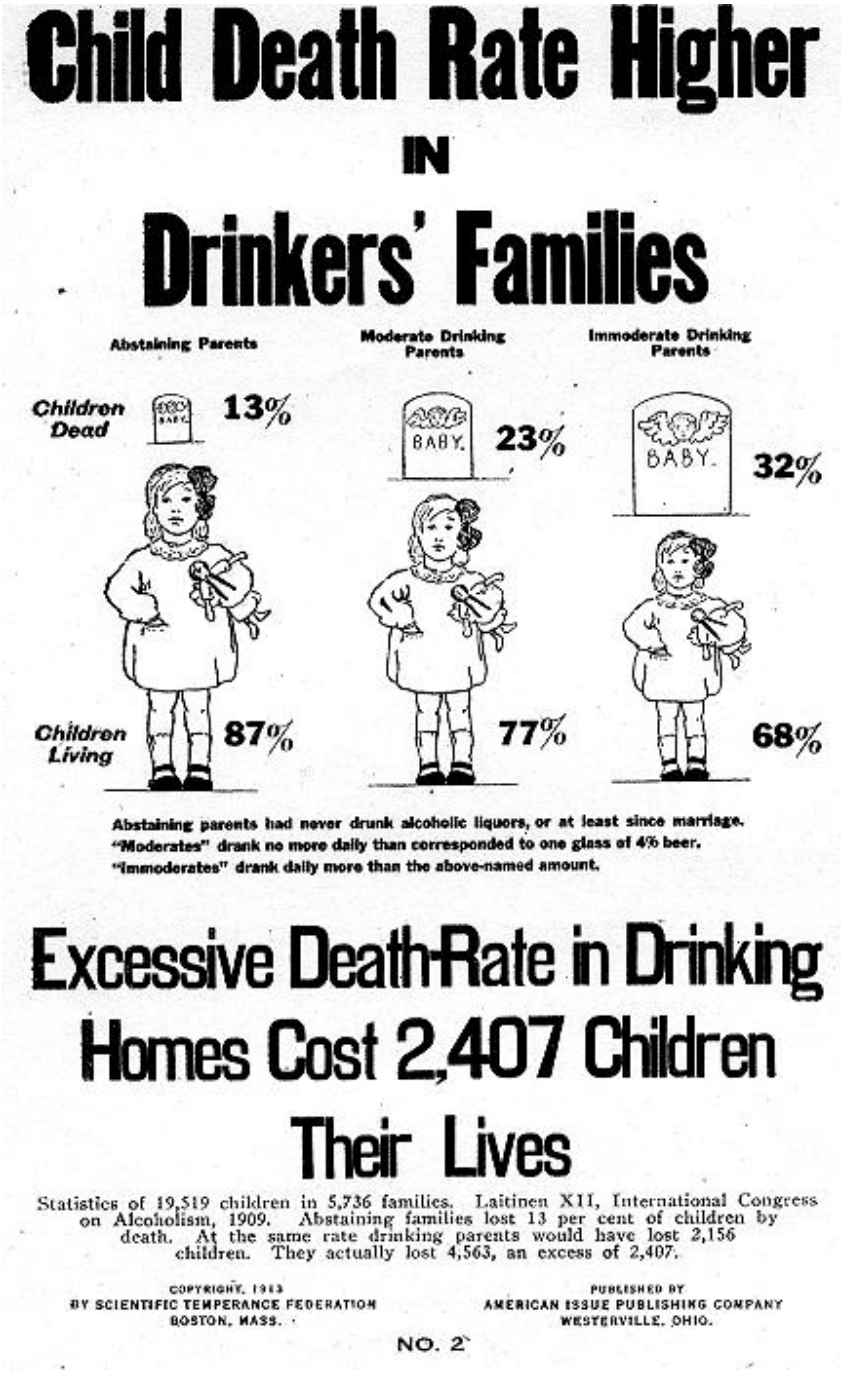
Goddard's study of the Kallikak family was central to the eugenics movement, as he recommended that the "feeble-minded" have their reproductive organs removed to prevent the procreation of feeble-minded children.[20] This, of course, was based on the premise that those like the Kallikak family would pass on feeble-mindedness to their children genetically. This movement gained momentum, and was still practiced into the mid-twentieth century, well after the medical temperance movement ended. Interestingly, after retrospective analysis of Goddard's data, some researchers suggest that members of the Kallikak family suffered the effects of prenatal alcohol exposure[21], but those assertions are speculative. Still, it is somewhat unsettling to think that the Kallikak study, which fuelled the now defunct eugenics movement, may have to do with prenatal alcohol exposure, not strictly genetics, and that an alternate method of preventing feeblemindedness may have been to restrict alcohol use rather than ovariectomy and castration.[20]

The research on alcohol and pregnancy of this area resulted in significant debates leading to, in many cases, personal attacks. Malcolm summarized a heated debate that occurred in 1903 between researchers opposed to the temperance movement (including Archdall Reid, quoted above) and those promoting abstinence from alcohol.[22] C. Mercier, for example, admitted that in comparison with Reid he and his colleagues, "are but worms, but why do they address their opponents in the tone of Almighty God addressing a peculiarly ignorant and recalcitrant black beetle?".[22]

Much of this research was encouraged and supported by groups and societies promoting abstinence, and became a moral, religious, and political quest, rather than a scientific quest for knowledge. Research was largely conducted to support the platform of these organizations. The Anti-Saloon League, for example, created periodicals, an encyclopedia, numerous flyers, and other publications to combat the use of alcohol in North America. The research they used was arguably biased and sensational. For example, one flyer (Fig.1) uses statistics to demonstrate that children of alcoholic parents have higher mortality, without accounting for other variables that might contribute to increased death rate, such as poverty.[23] In addition, much of the imagery in these flyers can illicit an emotional reaction, such as children pictured next to tombstones.

The moral and religious undertones that fuelled research into the effects of prenatal alcohol exposure may have also contributed to its temporary downfall. When Prohibition came into effect, alcohol was largely considered a moot issue, resulting in a significant decrease in research of the effects of prenatal alcohol exposure. Later, scientists discounted research in alcoholism as moralistic and unscientific.[24] In addition, sociological explanations for the deficits experienced by children of alcoholic parents began to be forwarded, such as poverty and abuse.[25] About fifty years after Prohibition and the near shut-down of studies into the effects of prenatal alcohol exposure, research again exploded when Jones & Smith provided a clinical description of the fetal alcohol syndrome.[5] Based on evidence to date, it is anticipated that this research topic should not fall out of favour again.

FIG. 1 One of the many flyers published by the Anti-Saloon League promoting alcohol abstinence. This particular flyer juxtaposes images of death with images of children, possibly eliciting an emotional reaction for the reader. Image courtesy of the Westerville Public Library, Westerville, Ohio.



DISCUSSION

According to this literature search, there is apparently only one comprehensive study of the history of FASD, by Warner & Rosett, which occurred over thirty years ago.[9] Another limitation of historical research in FASD is that the majority of rigorous articles on the topic were authored by only one researcher, Earnest Abel. Shibley & Pennington are excepted, as their review of the history of FASD emphasizes historical reliability, citing objectively, and a return to original sources where possible.[15] Other exceptions may exist but were not found in literatures searches. More sound, critical research and writing is needed regarding the history of FASD because many of the same erroneous assumptions are being made again, which tend to be an over-emphasis of the validity of historical data. Calhoun & Warren correctly recognize that it is hasty to assume that the effects of prenatal alcohol exposure were known to ancient Greeks and Romans, but they also reference the Biblical scripture discussed earlier out of context.[11]

Although historical records and research in the late-nineteenth and early-twentieth centuries discuss the effects of alcohol on fertility, it does not seem likely that these events directly influenced the discovery of fetal alcohol syndrome. Initial clinical descriptions of this condition[3,5] were conducted when these authors noticed differences within a population of children born to alcoholic mothers. It was exploratory, not confirmatory research, and references to previous studies and historical accounts were made after the fact. However, broader areas of alcoholism and teratogenics seem to be influential; Jones and Smith, for example, were teratogenecists.

Jones et al.[5] and Lemoine's[3] clinical descriptions mirror in many ways conclusions found in alcohol and pregnancy research in the early nineteenth century. Stillbirths and infant mortality were identified frequently in early research. Growth delays were recognized, and physical and central nervous malformations were identified in animal studies. Children of alcoholic parents were identified as being cognitively impaired by Trotter and later researchers. Behavioural and moral "degeneracy" were thought to be associated with these children.[9] Even a propensity for addiction was identified, which has been shown to be a significant and common problem for adolescents and adults with FASD.[26] One significant attribute was identified by Jones et al. and Lemoine that was not apparent in early research; the characteristic facial features of FAS. These were identified by these modern researchers as short palpebral features, epicanthal folds, smooth philtrum, flat midface, short nose, and thin upper lip.[3,6] Although their research was arguably more sound and unbiased than older research in prenatal alcohol exposure, they also introduced the Face of FAS to the world. Although Lemoine's research was "not taken seriously"[4], the Face of FAS may have played a role in increasing serious interest in this syndrome worldwide. Ironically, FAS facial features are now considered relatively unimportant in predicting central nervous system dysfunction.[27]

Although the construct of FAS/FASD has been questioned by some in the past[28], there is much evidence to suggest that alcohol has some teratogenic effect on the fetus that creates real and significant developmental concerns.[26] There is substantial evidence to suggest that prenatal alcohol exposure can negatively impact the central nervous system[29], and that these impacts often lead to functional deficits.[30] It is therefore probable that unrecognized effects of prenatal alcohol exposure have impacted lives for centuries. It is possible, for example, that members of the Kallikak family had FASD.[21] Although the presence of what is now known as FASD was likely present in times past, a knowledge of prenatal alcohol exposure specifically seemed to occur later, possibly as early as the late nineteenth century[18] but certainly by the mid to late twentieth century.[3,5]

Many historical records have been taken out of context when reviewing the history of prenatal alcohol exposure, and the impacts of these histories on modern-day FASD research have likely been overestimated. Historical records do suggest an awareness of an interaction between alcohol and reproduction of some kind, as early as biblical times. However, these records do not necessarily suggest an awareness of the deleterious effects of alcohol on the developing fetus. Historical records regarding alcoholism and reproduction need to be interpreted critically, in context, and in consideration of the Zeitgeist, or the Spirit of the Times.

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