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A Brief History of Awareness of the Link Between Alcohol and Fetal Alcohol Spectrum Disorder

Un bref historique de la découverte du lien entre l'alcool et le trouble du spectre de l'alcoolisation fœtale

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Abstract

Objective: *Fetal alcohol spectrum disorder* (FASD) is a medical term used to describe a range of mental and physical disabilities caused by maternal alcohol consumption. The role of alcohol as a teratogen and its effects on the cellular growth of the embryo and the fetus were not determined on scientific grounds until the late 1960s. However, the link between alcohol use during pregnancy and its harms to offspring might have been observed frequently over the many thousands of years during which alcohol has been available and used for social and other reasons.

Methods and Results: Using sources ranging from the biblical *Book of Judges* (pre-1700) up until the first public health bulletin (1977), we seek to provide an overview of the academic debate around early historical accounts ostensibly attributed to the awareness of alcohol as a prenatal teratogen as well as to describe the social and political influences that sculpted developments leading to the public recognition of FASD.

Conclusions: Our analysis provides a brief overview of the discourse regarding historical awareness of the detrimental effects of prenatal alcohol exposure on fetal development leading to the formal recognition of FASD as a distinct clinical entity. Further research will be required to fully appreciate the scientific, medical, and societal ills associated with prenatal alcohol exposure.

Abrégé

Objectif : Le trouble du spectre de l'alcoolisation fœtale (TSAF) est un terme médical servant à décrire une gamme de déficiences mentales et physiques causées par la consommation d'alcool de la mère. La découverte de l'alcool comme tératogène et de ses effets sur la croissance de l'embryon et du fœtus n'a pas été déterminée pour des raisons scientifiques avant la fin des années 1960. Cependant, le lien entre la consommation d'alcool durant la grossesse et les torts causés à l'enfant peut avoir été observé fréquemment au cours des milliers d'années où l'alcool était disponible et consommé pour des raisons sociales et autres.

Méthodes et Résultats : Depuis le *Livre des Juges* de la bible (pré-1700) jusqu'au premier bulletin de santé publique (1977), nous avons cherché à offrir un aperçu du débat scientifique sur les premiers récits historiques ostensiblement attribués à la connaissance de l'alcool comme tératogène prénatal, ainsi qu'à décrire les influences politiques et sociales ayant joué dans les développements qui ont entraîné la reconnaissance publique du TSAF.

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Conclusions : Notre analyse offre un bref aperçu du discours sur l'historique de la prise de conscience des effets nuisibles de l'exposition prénatale à l'alcool pour le développement du fœtus qui a mené à la reconnaissance officielle du TSAF comme entité clinique distincte. Il faudra plus de recherche pour connaître pleinement les maux scientifiques, médicaux et sociétaux associés à l'exposition prénatale à l'alcool.

Keywords
fetal alcohol spectrum disorder, fetal alcohol syndrome, history, maternal alcoholism, alcohol, pregnancy, placental barrier

Maternal consumption of alcohol can negatively affect fetal development, resulting in a range of mental and physical disabilities clinically termed *fetal alcohol spectrum disorder* (FASD). Deficiencies in fetal growth and development of the brain and central nervous system occur and are manifest after birth as cognitive and emotional deficiencies, including problems with memory, learning, attention, and social communication.[1,2] Impairments may also include growth retardation and malformations of the face. Fetal alcohol syndrome (FAS) is the most severe and visually apparent subtype of FASD and is considered a leading cause of preventable mental disability in Canada and the United States.[3]

Although the official recognition of FASD as a clinical disorder is relatively recent,[4,5] details pertaining to the first documented cases suggesting social awareness have been debated. This paper reviews the discourse on the history of this preventable disorder, from the biblical *Book of Judges* (pre-1700) to the first public health bulletin (1977).

Methods

A search of biomedical electronic databases was conducted to identify primary studies and systematic reviews that analyzed, evaluated, and/or compared and contrasted historical accounts of prenatal alcohol exposure (PAE) and/or FASD recognition. Reference lists obtained from reviews and retrieved articles were used. Non-English-language studies were largely excluded.

Ancient History (pre-1700)

Evidence of historical knowledge of the negative effects of drinking during pregnancy can be dated back hundreds of years.[5-9] The first proposed acknowledgement of the teratogenic effects of PAE is attributed to the biblical *Book of Judges* 13:3-4, from the Old Testament, which conveys to the mother of prominent biblical character Samson, "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." Multiple academic references attribute this statement as recognition that drinking alcohol during pregnancy will affect the well-being of the child.[6,8]

Another quotation referenced intermittently in descriptions of FASD-related awareness is from Aristotle's *Problemata:* "foolish, drunken, or haire-brain women most often bring forth children like unto themselves, morose and languid."[9,10] As *Problemata* was assembled from a variety of sources over several eras and translated several times, sources that use or refer to this quote have drawn from Robert Burton's 1621 book *The Anatomy of Melancholy*[9] as a "primary" source and attribute the quote as indicative of Aristotle's awareness of prenatal alcohol effects.[7]

A third historical reference, identified by Jones and Smith[5] in 1973, entails the Carthaginian injunction prohibiting the consumption of alcohol on the eve of a couple's wedding night "in order that defective children might not be conceived." In 1999, Abel[11] attributed this order in part to Plato's *Laws*,[12] which states that couples trying to conceive should not do so while intoxicated: "it is not right that procreation should be the work of bodies dissolved by excess of wine, but rather that the embryo should be compacted firmly, steadily and quietly in the womb." Although there are several different interpretations supporting and contending the historical recognition of FASD,[11,13,14] these historical references likely represent, at minimum, observations between maternal alcohol consumption and birth defects deriving from prenatal insult. Other researchers have made similar arguments and reflections on the history of FASD.[9]

Select Artistic and Literary References in the 18th and 19th Centuries

In the first half of the 18th century, England lifted distilling restrictions, which in turn flooded the market with gin, leading to what was later called the "gin epidemic." The artist William Hogarth's (1697-1764) two paintings of interest during this time are "Gin Lane" and "Beer Street." Both pieces were designed to be viewed alongside each other and are believed to depict the evils of consuming gin in comparison with the virtues of drinking beer.[14,15] Gin Lane has been suggested to reflect the known social ills of exuberant drinking, with special attention given to the mother dropping a baby with facial malformations suspiciously resembling those observed in FAS.[7] An anthropologic study analyzing the gin epidemic and Hogarth's paintings concluded that the struggle conveyed is that of social and economic control between classes. Gin Lane symbolized urban decay: "gin was not simply about drunkenness; it was about the new drunkenness of the lower classes."[15]

Regardless of Hogarth's motives for creating Gin Lane, the gin epidemic itself led to what Warren[16] described as the

first clear "writings identifying negative outcomes of alcohol consumption on progeny." These writings came from the College of Physicians in London, which, in a presentation to the House of Commons, blamed gin as a culprit for "weak, feeble and distempered" children (Minutes of the College of Physicians in London, 1725; referenced in Hoyme et al.[17]). But as Warren[16] noted, "It is not clear whether these early social commentators viewed the harm befalling offspring as rising from the consumption of the beverage by females or by males, or both; as a consequence of drinking prior to or during pregnancy; or as a consequence of drinking after pregnancy through breast milk; or the feeding of gin in place of, or in addition to breast milk."

The literary world provides evidence that the general public may have had some awareness that drinking during pregnancy may be detrimental to a baby's health. For example, Charles Dickens[18] wrote in his 1836 novel, *The Posthumous Papers of the Pickwick Club*, "Betsy Martin, widow, one child, and one eye, . . . but knows her mother drank bottled stout, and shouldn't wonder if that caused it. . . . Thinks it not impossible that if she had always abstained from spirits, she might have had two eyes by this time." Aldous Huxley's 1932 novel *Brave New World*[19] refers several times to the perceived ills of adding alcohol to an incubating embryo: "They say somebody made a mistake when he was still in the bottle—thought he was a Gamma and put alcohol into his blood-surrogate. That's why he's so stunted."

Temperance Movement, Prohibition, Eugenics, and Clinical Practice

The temperance movement produced significant medical research linking PAE to adverse birth outcomes. Additionally, with the introduction of Charles Darwin's "On the Origin of Species" in the mid-19th century, researchers sought to distinguish between alcohol's hereditary effects on offspring (generational degradation caused by alcoholism of a parent) and the direct prenatal insult of alcohol on a developing fetus (nonhereditary).[7] In a significant study published in 1899, Sullivan[20] observed a higher mortality rate among the newborns of alcoholic women compared with others in the mother's family and/or incarcerated mothers who had limited or no access to alcohol. In 1900, Nicloux[21] discovered that the placenta did not serve as a barrier to stop the transfer of alcohol from the mother to the embryo or fetus. In 1904, Ballantyne[22] attributed alcohol to an increased risk of adverse birth outcomes including structural dysmorphia, spontaneous abortion, and premature labor. These initial studies, along with momentum from the temperance movement, led to a modest spur in research from 1909 to 1914 (e.g., Laitinen[23] and Stoddard[24]).[16] Several animal studies were conducted between 1903 and 1922 that demonstrated physical defects in offspring of alcohol-exposed mothers.[7,25]

When the temperance movement resulted in Prohibition, there was a major shift in public attitudes on alcohol and pregnancy, which allegedly caused some in academia to reverse the conclusions of earlier research due to the now-controversial nature of the topic. For example, Warren[16] states that Charles Stockard, a Cornell University academic, conducted a study on PAE in guinea pigs, first aligning his discussion of results with the temperance movement in 1914 and then reinterpreting his findings 18 years later to suggest that alcohol served to "strengthen the stock by eliminating weak fetuses." Other researchers stepped away from the focus altogether, which resulted in a scarcity of published articles from 1930 to 1950. Warren[16] attributes the gap to "the rejection of earlier evidence pertaining to alcohol and pregnancy following the repeal of Prohibition in the United States, Canada, and several European countries; and misinterpretation of earlier research findings in a eugenic rather than toxicological context." Saunders[14] and Warner and Rosett[7] suggest that this lull was the result of alcohol being considered a "moot" or "dead" issue following prohibition and that pre-Prohibition era research was mostly disregarded due to "its moralistic and 'unscientific' language."[7] Randall[25] adds that there was pushback from the medical community, as "most physicians continued to believe that the placenta acted as a barrier to all agents and protected the unborn child." This dismissal of evidence may have occurred in part because obstetricians frequently used alcohol for mothers at risk of premature labor, in addition to the fact that alcohol was one of the few accessible anesthetics at the time for pregnancy.[7] "As a result, obstetrical writers took an ambiguous position toward alcohol."[7] In contrast, Martin and Holloway[26] argue that although "it would take the next few decades" for physicians and researchers to fully acknowledge that presence and harm were synonymous in the maternal consumption of teratogens, the scientific and medical communities were aware that the placental barrier did not in fact protect the fetus from insult, referencing multiple obstetrics textbooks from 1938 to 1951[26-30] that explicitly acknowledge this fact. Martin and Holloway suggest that the aforementioned references, along with a proliferation of prescriptions given to pregnant women during this time, suggest that "fetal harm from maternal ingestion of drugs was simply not a concern of the average physician." Greene and Podolsky attribute the potential oversight by physicians as follows: "The rapid escalation of innovation and promotion in the pharmaceutical industry at mid-century provoked a broader crisis of overflow in medical education in which modern physicians were trapped between commercial and professional sources in an attempt to keep modern by incorporating the glut of emerging technologies, therapeutics, and related information into their practices."[26,31] Furthermore, as early as the mid-1800s, pro-life advocates emphasized the organic distinction of mother and fetus by placental barrier as evidence of intrauterine autonomy of the child, further politicizing and stigmatizing research and discourse around women's reproductive health.[26,32]

In the 1950s to 1960s, researchers began observing physical malformations in children exposed to radiation from the atomic explosions in Hiroshima and Nagasaki and from the maternal ingestion of thalidomide, which increased the scientific community's interest in the field of teratology. Thalidomide specifically is attributed to having "opened the gates for more researchers, more funding, and more institutional infrastructure in the areas of placentology, teratology and reproductive science."[26] Research on the maternal consumption of alcohol, however, remained unpopular until 1973, when Jones and Smith[5] produced convincing evidence that PAE caused a pattern of fetal malformations.[33]

Recognition

Although an unpublished 1957 thesis was allegedly developed by Jacqueline Rouquette that observes patterns of "facies" of children born to alcoholics and attributes responsibility to the mother in the contribution of these malformations,[34] the first acknowledgement of a pattern of symptoms for FASD is largely attributed to Lemoine in 1964 and again in 1968.[4] Lemoine claims he received little recognition for his findings, stating that "my French colleagues did not believe me then and they do not believe me to this day" (letter from Lemoine to Jones and Streissguth[33]). Additionally, as Lemoine's publication was available only in French[25] and did not present specific diagnostic criteria or result in FASD recognition or diagnosis in France,[17] Hoyme and colleagues[17] argue that the true recognition of FASD was made in 1973 by Americans Jones and Smith. Jones and Smith[5] specifically coined the term *fetal alcohol syndrome*, provided diagnostic criteria for the disorder, and "described in detail the consistent pattern of malformations among children of the mothers with significant prenatal alcohol intake."[17] Of important note is that this 1973 publication was followed by additional supportive research to provide clarity in areas of possible contention regarding the teratogenic effects of PAE,[33] as "the existing prevalent view that alcohol was safe in pregnancy led to much skepticism of the proposed teratogenicity of alcohol and whether or not the agent underlying this newly named syndrome was indeed alcohol."[16] In 1974, Jones et al.[35] published a study that adjusted for confounding factors, to clarify that the patterns observed in the 1973 study were due to socioeconomic factors. A proliferation of animal[36,37] and human studies[38-41] then followed, seeking to confirm or refute the existence of FAS.[16] Findings from these studies not only supported Jones and Smith's 1973 conclusions but also defined alcohol as a classic teratogen that could affect behavior in the absence of obvious physical deformations.

The first public awareness warning was delivered via the U.S. Food and Drug Administration *Drug Bulletin* on June 1, 1977, and subsequently by the National Institute on Alcohol Abuse and Alcoholism (NIAAA) in the Centers for Disease Control and Prevention (CDC) *Mortality and Morbidity Weekly Report*.[16] "This first advisory recommended a 2 drink per day limit for pregnant women and defined a clear risk at a level equivalent to 6 drinks per day."[16] Following this public service announcement, the advisory was endorsed by the American Academy of Pediatrics, March of Dimes, American Medical Association, and American Society of Addiction Medicine; eventually the American Congress of Obstetricians and Gynecologists formally acknowledged the advisory. The CDC has since updated its policy, emphasizing that "there is no known safe amount of alcohol use during pregnancy or while trying to get pregnant."[42]

Conclusion

We have provided a brief historical review of social, academic, and clinical recognition of the effects of PAE on fetal development and formal acceptance of FASD. The historical literary references clearly recognized that excessive alcohol intake may be harmful to both mother and infant, but publications did not make a clear link to fetal deformities until quite recently. Further work is needed to understand the extent to which social knowledge about the ills of drinking during pregnancy may have existed and the delay in clinical and academic acknowledgement. An additional layer of interest involves the extent to which pro- and anti-abortion activism, through discourse around intrauterine autonomy, threaded into the proliferation and/or obstruction of research.

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