Commentary: What might have been: Sullivan may have impacted modern prenatal alcohol research under different circumstances

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William Sullivan’s 1899 paper, ‘A Note on the Influence of Maternal Inebriety on the Offspring’, often cited as a precursor to modern fetal alcohol research, was published at a time when alcoholism was a topic of great interest in Europe and North America. Emerging theories of heredity, eugenics and temperance movements drove alcoholism research from 1860 to 1910. In addition to an upsurge of medical literature on alcoholism, a number of journals devoted to the topic were formed (e.g. Journal of Inebriety). Benedict Morel’s theory of hereditary degeneration was influential in the medical field generally, including research on alcohol and reproduction. This theory postulated that degenerative traits, of which alcoholism was thought to be one of the more obvious, pass on hereditarily and accumulate over generations, eventually leading to the extinction of the family bloodline by about the fourth generation. Since heredity was the transmitting mechanism for degenerative traits, paternal alcoholism was generally considered as detrimental as maternal alcoholism. While some researchers and activists were concerned that alcoholism would degenerate society as a whole, others suggested that through degeneracy and extinction of family lines the propensity for alcohol would limit itself. Since alcohol was readily available and its effects observable and treatable, the study of alcoholism as a degenerative condition was of particular interest, and most alcoholism research during this period supported the degeneration theory. Sullivan strongly advocated the theory of degeneration in his alcoholism research.

As a physician serving in a Liverpool prison, Sullivan had access to life histories of alcoholic female inmates. His 1899 study involved two main components: an epidemiological study of female inebriates and their children, and a case study of 11 alcoholic mothers who were imprisoned while pregnant. Citing prior research summarizing the alcoholic tendencies of parents of ‘degenerates’, Sullivan’s aim was to distinguish alcoholism as the primary degenerative agent by investigating ‘not alcoholism in the ancestry of the degenerate, but degeneracy in the descendants of the alcoholic’. The research design constructed by Sullivan, the epidemiological data he collected and the analyses he conducted were quite impressive. Sullivan acknowledged limitations to the data while maintaining that results could be generalized to parental alcoholism. The validity of the study was enhanced by excluding inmates who were suspected of ‘other degenerative factors’, such as tuberculosis or syphilis, or who were ‘markedly neurotic’. When analysing outcomes of 600 children born to 120 alcoholic mothers, he found that the majority of children died before the age of 2 years. In addition, child mortality rates were higher among alcoholic mothers compared with sober female relatives.

Perhaps, a more important aspect to Sullivan’s paper was a case study of 11 alcoholic mothers who were forced to abstain from alcohol during pregnancy because of imprisonment. Among these mothers, Sullivan found decreased mortality of children who were in utero during imprisonment relative to their siblings who were likely exposed to alcohol prenatally. Although Sullivan acknowledged that other environmental factors related to parental alcoholism could play a role in child mortality rates or negative outcomes, he suggested not only that the impact of maternal alcohol consumption was considerably greater than that of paternal alcohol consumption, but that alcohol appeared to have a ‘direct toxic action on the embryo’.

A direct action of alcohol on the developing fetus proposed by Sullivan has been confirmed in modern day prenatal alcohol research. Increases in stillbirths and infant mortality have been confirmed among mothers who drink heavily or binge while pregnant. The impact of alcohol on the developing fetus extends beyond increased mortality, however, as research has confirmed a neurodevelopmental impact caused by heavy prenatal alcohol consumption. Although the neurocognitive profile continues to emerge,
consequences of heavy prenatal alcohol exposure includes deficits in intellectual ability, attention and speed of information processing, executive functioning, language, visual perception and construction, learning and memory, and number processing.8

Research on the neurodevelopmental effects of prenatal alcohol exposure exploded in the 1970s, spurred by papers published by dysmorphologists David Smith and Kenneth Lyons Jones of the University of Washington in Seattle, USA. These physicians and their colleagues described developmental delay, microcephaly, growth deficiency and aberrant facial features in a handful of children born to alcoholic mothers9,10 and named the condition Fetal Alcohol Syndrome (FAS).11 Researchers and clinicians soon observed that the physical and neurobehavioural consequences of prenatal alcohol exposure varied.12 Currently, the term Fetal Alcohol Spectrum Disorder (FASD) is used to describe the range of deficits associated with prenatal alcohol exposure.

The Seattle team indicated in the first of their seminal articles that ‘This seems to be the first reported association between maternal alcoholism and aberrant morphogenesis in the offspring’,9 suggesting that they were unaware of Sullivan’s publication. In their second article, however, Jones and Smith cited Sullivan’s work as well as other historical references. Although his paper preceded modern fetal alcohol research by many decades, Sullivan’s work did not appear to directly influence the emergence of modern prenatal alcohol research. Although Sullivan’s findings were suggestive of a direct toxic effect of alcohol on the fetus, and although subsequent animal studies suggested the same,13 the moral undertones of the temperance movement in the UK and the USA created a climate where alcoholism research was considered by many as moralistic and unscientific.14 In addition, research may have been hindered by a developing but rudimentary understanding of genetics, heredity, toxicity and teratology.15 A controversial, yet influential 1910 paper by biometrician Karl Pearson and his colleague Ethel Elderton set the tone for research in parental alcoholism for the immediate future. They concluded that they found no marked biological relationship between intelligence, disease or physical health and parental alcoholism. Rather, they suggested that such deficits, more readily observed in alcoholic families, were due to social factors and poor care.16 World War I also had an impact on the availability of resources for research in this area. By 1919, when prohibition was put into effect in the USA, the perceived need for alcoholism research declined, with some researchers abandoning the topic entirely. Philip Pauly suggests that research in alcoholism and reproduction became ideologically driven. The end result was that by the 1930s the topic of alcohol and reproduction became ‘scientifically uninteresting’.17 The following half-century is marked by a lack of research on the topic, described by Warner and Rosett as ‘forgetfulness’.2 In a different social–political environment, perhaps Sullivan’s findings could have been a springboard to further investigate the ‘direct toxic effects’ of prenatal alcohol exposure.

Although his contribution is acknowledged in modern prenatal alcohol research literature, Sullivan is typically not credited with what appears to be the first demonstrable evidence of a direct toxic effect of prenatal alcohol exposure on the fetus. Jones and Smith are often credited with discovering the harmful consequences of prenatal alcohol exposure, but it is more accurate to suggest that they brought the neurodevelopmental impacts of prenatal alcohol exposure to the forefront. In fact, the impetus for the Seattle team’s research emerged with observations made by Christie Ulleland,18 a student who was listed as a secondary author on the first paper. Yet, in 1968, Paul Lemoine, a physician in Paris, France, published a clinical description of 127 children of alcoholic mothers.19 His descriptions of physical malformations, developmental delays and behavioural challenges were very similar when compared with the descriptions of 11 children by the Seattle team.9,11 Lemoine’s study, however, did not appear to have any immediate influence. Lemoine also acknowledged that an unpublished thesis in 1957 by Jacqueline Rouquette in France, describing malformations of children born to alcoholic parents, preceded his work.20 The Seattle team was not the first to discover the consequences of prenatal alcohol exposure, as has been frequently cited in research articles glossing over the history of prenatal alcohol research.21 Rather, their contribution, and a critical one at that, was successfully generating clinical and research interest in the topic.

The recent evolution of research on alcohol and reproduction can provide important lessons for reviewing the histories of research in scientific publications; that those who make a discovery are not always immediately credited with their discoveries; that discoveries made at an earlier time may or may not influence later re-kindling of research topics; that the evolution of knowledge is not necessarily linear or constant; and that social–political thinking may guide, promote or hinder research. To avoid making inappropriate assumptions when writing on history, researchers should think critically about the primary and secondary sources they are citing, consider the contexts of their references and ensure they are giving credit where credit is due.

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References

Commentary: On W.C. Sullivan and his ‘[N]ote on the influence of maternal inebriety on the offspring’

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Background

Debate raged through the 19th and early 20th centuries as to whether maternal alcoholism, per se, was a cause of the mental retardation and delinquency found in children of alcoholics.\(^1\)\(^-\)\(^7\)

Three causes were listed:

(i) inherent toxicities of alcohol \(in\) utero;