Is foetal alcohol syndrome in children as old as alcohol consumption?

Foetal alcohol syndrome (FAS) is the leading cause of preventable intellectual disability in the world, according to the World Health Organization, and the true prevalence is almost certainly underestimated. As long as alcohol is present in our everyday lives, FAS will continue to challenge families, communities and countries.

In this issue of *Acta Paediatrica*, Dr Feldmann suggests that a historical study of children living in a 1930s German wine-producing community showed widespread FAS. We believe that FAS and foetal alcohol spectrum disorder (FASD) have been around for as long as alcohol has been available, and not at all restricted to times and places where alcohol has been used as reward or payment for work.

Pulque, a fermented agave juice with 4%-7% alcohol, can be traced back thousands of years to native Meso America. Ancient Aztecs ritualised Pulque and strictly limited its use, but it was still widely used when water was scarce. By the 1500s, Pulque had become so popular that it was temporarily prohibited to quell the devastating health and social problems it wrought on native Mexicans. It is still extensively consumed in poorer rural communities and pregnant women are encouraged to drink it due to its nutrient content and beliefs about its lactogenic effects. It has been suggested that heavy and early Pulque use in pregnancy adversely effects offspring growth and development.

The College of Physicians suggested that excessive gin drinking in early 18th century, London, after distilling restrictions were lifted, caused ‘weak, feeble and distempered’ children. In 1889 and 1895, the French Psychiatrist Maurice Legrain linked alcohol to intrauterine and neurological problems and grave mental issues. More than 40% of inmates in his asylum were diagnosed ‘hereditary insane’, due to their parents’ alcoholism. In the early 1900s, emerging research showed hereditary effects, direct teratogenic effects on the foetus, and other physical defects in animal models.

A presentation at the Fourth International Congress against Alcohol Abuse in The Hague, in 1892, stated that alcohol was a toxic agent and alcohol abuse could poison the foetus in utero. Blastotoxie occurred when one partner was drunk during conception and blastophobie was when one partner was a chronic alcoholic whose germ plasma had been seriously poisoned by alcohol.

Literary works by authors such as Charles Dickens suggest that the general public were also aware of the deleterious effects that maternal alcohol use had on offspring. Many physicians of that time took an ambivalent stance, as alcohol was used as an anaesthetic, with few alternatives, and when premature labour was threatened. Some practitioners continued to believe the placenta was impervious to alcohol. When American prohibition arrived in the 1920s, it dampened enthusiasm for new studies, and scientists eschewed the pre-prohibition toxicological research, viewing it as tainted by moralistic tones. It was not until Lemoine’s rediscovery of FAS in the 1960s that interest and research were revived.

The lives of women and children in ancient Meso America, 18th century London and 19th century France, may have been very different to life in a 1930s German wine region, but for Kaiserstuhl children, the implications of regular parental drinking were the same. Even so, we should avoid unfairly judging past research against today’s standards and we were cautious when drawing inferences as we only had Feldman’s essay to go on.

Given the lack of interest in alcohol and human development in the 1950s, the Kaiserstuhl paper was probably novel. It might even have been a stand-out study, but for the author’s conclusion that daily alcohol use by children and parents had no negative impact on their growth. What’s more, it seemed to have been met with little scholarly opposition; the documented studies, suspicions and theories raised in the previous century were apparently insufficient to spur on a challenge by the medico-scientific community.

Feldmann suggests that because Fischer did not connect the children’s facial features to maternal alcohol consumption, he did not report any sub-group analyses. This may have been because the Kaiserstuhl children routinely drank wine, but their growth and gross motor skills appeared normal, even superior, to other groups. A more recent South African study also showed no detectable deficiencies in gross motor skills among FAS children in a wine-producing area compared with controls. As for the Kaiserstuhl children’s normal height and weight, there may have been compounding differences in nutritional status between them and the control population in urban Freiburg, which was very badly affected by World War I. It is plausible that rural Kaiserstuhl had better access to nutritious food. Perhaps the wine also provided a source of nutrition in an otherwise limited diet.

It is unfortunate, but understandable, that the Fisher study lacked reliable measures of cognitive function. Fisher used a Norwegian 60-m timed run as his key measure. The Kaiserstuhl children showed good running speeds, probably because they were unusually fit from...
their gruelling work. On the basis of this criterion alone, they were judged normal for concentration and tenacity.

A 10% FAS prevalence, based upon the facial features of the Kaiserstuhl children,\(^2\) does not seem excessive for a population with a high daily alcohol consumption. The worldwide estimate for FAS is 119 000 children a year, or 15 per 10 000 births, led by Europe with 37 per 10 000.\(^6\) Prevalence of the more broadly defined FASD may be 10 times greater than FAS at about 1.5% of global births but almost 4% in Europe.\(^8\) It is noteworthy that both wine production and FAS are higher in Europe than anywhere else in the world, although strong spirits in Eastern Europe undoubtedly plays a role in high FAS prevalence.

Although the brain is the most severely impacted organ, prenatal alcohol exposure can also cause abnormalities in the heart, kidney, liver, gastrointestinal tract and endocrine system.\(^5\) These severe consequences are not always easily detected and are often difficult to retrospectively connect to alcohol exposure. Newer studies indicate that the effects on offspring may not be limited to maternal consumption, as paternal alcohol use reduces sperm count and quality and may also affect offspring through epigenetic changes.\(^7\)

Fisher did not report how much the women drank during pregnancy, only that they drank less than the fathers, who consumed 3-5 L of wine with an alcohol content of 7% on a daily basis. If the ratios in those days were similar to modern day Germany, where men drink three times as much as women,\(^1\) the Kaiserstuhl women may have consumed 50-90 g of pure alcohol a day. If Kaiserstuhl parents maintained their wine consumption up to, and during, pregnancy, perhaps as a source of nutrition, then it is very likely that the prevalence of FAS and FASD was even higher than the 10% hinted at by Fischer’s observation of facial features.\(^2\)

It does seem likely that the Kaiserstuhl children were at serious risk of FAS or FASD, but their plight was not unique. High FAS can also be found in vulnerable populations, including aboriginal communities, children in care, prison populations and those receiving psychiatric care.\(^10\) These high-risk populations deserve special attention when it comes to planning and organizing targeted screening strategies, improved access to diagnostic and support services and, prevention of maternal alcohol consumption.

Compelling scientific evidence tells us that the teratogenic effects of alcohol are vast and diverse and create severe long-term consequences for the individual, their family, society, public health and the economy. Alcohol use and pregnancy remains one of the most important parental lifestyle choices, and drinking before pregnancy can influence drinking during pregnancy. That is why policies that address alcohol consumption by the whole population are needed, to learn from the past and reduce the future incidence of FAS and FASD.

**CONFLICTS OF INTEREST**
The authors have no conflicts of interest to declare.

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