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Abstract

The diagnosis of Fetal Alcohol Spectrum Disorders (FASDs) is embedded in a matrix of biological, social and ethical processes, making it an important topic for crossdisciplinary social and ethical research. This article reviews different branches of research relevant to understanding how FASD is identified and defined and outlines a framework for future social and ethical research in this area. We outline the character of scientific research into FASD, epidemiological discrepancies between reported patterns of maternal alcohol consumption during pregnancy and the incidence of FASD, and the social and ethical considerations that may impact on who is, and is not, diagnosed. We highlight what further research investigating FASD diagnostic processes, as well as the multi-generational impacts of FASD, is needed. Important research priorities are to: 1) enumerate the variety of stakeholders involved in seeking FASD diagnoses; 2) understand the experiences and perspectives of mothers from different backgrounds who have consumed alcohol during pregnancy and their affected children; and 3) collect health histories of maternal alcohol consumption in families to determine the effect of FASD at sub-cultural and cultural levels.

Keywords

Fetal Alcohol Spectrum Disorders Critical neuroscience Diagnosis

Introduction

Fetal Alcohol Spectrum Disorders (FASD) comprise a constellation of physical abnormalities and neurodevelopmental disorders that are caused by prenatal alcohol exposure [1, 2]. At the most severe end of the spectrum is Fetal Alcohol Syndrome (FAS), diagnosed when a child exhibits specific facial dysmorphism, malformed organs, arrested mental development and growth, and when the mother is known to have consumed alcohol during pregnancy [3–5]. Subsequent to the characterisation of FAS in the early 1970s, a range of other conditions, such as alcohol-related birth defects (ARBD) and alcohol-related neurodevelopmental disorders (ARND), have been included under the umbrella of FASD [1, 2]. In these latter conditions, the facial deformities that characterise FAS may be absent. Advances in epidemiology, biogenetics and neurobiology are changing how FASD is understood [2, 6–9] but there are as yet no biomarkers or definitive diagnostic tests for FASD. A diagnosis of FASD is conferred on the basis of clinical judgement according to a number of different diagnostic criteria, and there is some debate as to the validity of different approaches to its diagnosis [10]. FASD is primarily a diagnosis of exclusion made after other more common genetic disorders and diagnoses have been ruled out [11]. Diagnosis also requires explicit disclosure, or other confirmation, of maternal alcohol consumption during pregnancy [4].

In this paper, we argue that we need to understand FASD as a condition that arises from a matrix of interrelated social, ethical and biological factors. Conceptualising diagnosis as both a category and as a process [12–14], we argue that research on FASD can benefit from a cross-disciplinary approach that includes contributions by sociologists and philosophers of science, anthropologists and neuroethicists [15–17]. We review the character of, and challenges facing, scientific research into FASD. The latter include the puzzling mismatches between reported patterns of maternal alcohol consumption during pregnancy and the incidence of FASD in groups of different socioeconomic status; and the social and ethical considerations that may affect who is, and is not, diagnosed with FASD. We make two conjectures: first, that the social and ethical issues that surround maternal alcohol consumption during pregnancy and fetal development are also important in the evolution and understanding of FASD diagnosis; second, a multi-generational examination of FASD is important to

understanding the impacts of alcohol on society, and the impact of the environment on social disadvantage.

Current Understandings of FASD

The FAS was first characterised in 1973 on the basis of structural malformations observed in children born to women who were chronic alcoholics, although there was evidence before the 1970s that alcohol had deleterious effects on the developing fetus [3, 5, 18]. Even though alcohol is known to cause birth defects in humans and non-human animals, scientists have been unable to specify how much alcohol, and at what stage of pregnancy, the fetus is at most risk of these outcomes [19–21]. Efforts to better understand these processes are complicated. Ethical imperatives preclude experimental studies on human populations of alcohol's effects on neurodevelopment. Animal models are limited by alcohol's variable effect on different laboratory species compared to humans and by important differences in the cognitive and behavioural functioning of humans and commonly studied non-human species. For these reasons, observational studies of relationships between alcohol consumption by women during pregnancy and birth outcomes have been the most important way of studying FASD [2]. Most recently, longitudinal cohort studies have tested possible cause and effect relationships between low to moderate alcohol consumption and reduced IQ using novel statistical approaches that use genetic variations to control for the effects of confounding (via Mendelian randomization [22]). These approaches seek to measure associations between women's self-reported alcohol consumption during pregnancy and genetic factors that have been shown to affect the metabolism of alcohol [8]. While this approach is purportedly less subject to confounding than traditional epidemiological methods, the validity of these inferences still depends on the reliability and validity of self-reported alcohol consumption during pregnancy.

Neuroimaging studies have explored the effects of alcohol on neurodevelopment [6, 9]. These studies often compare in utero alcohol-exposed persons with 'normal' controls who have had no or low in utero alcohol exposure [9]. However, there are a number of issues that may affect the validity of these studies: 1) the way in which 'normal' and 'exposed' cases are identified may be subject to a number of systematic biases; 2) studies to date have not followed up subjects over time and, therefore, it is unclear whether observed differences between cases and controls persist, or tend to the population norm, over time; 3) neuroimaging studies can only identify brain differences associated with cognitive deficits between cases and controls; they cannot determine whether prenatal alcohol exposure is the cause of the difference; and finally 4) prenatal alcohol exposure can produce microcephaly, where a significantly smaller brain develops compared with that of non-alcohol exposed individuals. However, neuroimaging requires the projection of images onto a standardized brain template. The difference in size between an alcohol affected brain and that of a 'normal' brain may cause skewed projections [9].

Epidemiological Puzzles in Relationships Between Maternal Alcohol Consumption and the Prevalence of FASD

The documented incidence of FASD in human populations varies with maternal poverty, poly-drug use, and nutritional deficiencies as well as socio-historical factors such as the impacts of colonialism on indigenous peoples. As Ernest Abel [23] has noted: 'FAS is not an equal opportunity birth defect'. Studies of maternal alcohol consumption in OECD countries suggest that women from upper socioeconomic groups are more likely to drink alcohol during pregnancy [24–32] but have significantly lower rates of FASD than women in lower socioeconomic groups [23]. One recent study has found that women from more socially disadvantaged backgrounds are more likely to either abstain or binge drink during pregnancy, while those who are from more socially advantaged backgrounds are more likely to drink consistently at low to moderate levels [33].

The relationship between socioeconomic status, ethnicity and alcohol consumption during pregnancy adds further complexity. Different researchers argue for, and against, the significance of cultural differences in maternal alcohol consumption [23, 27, 34]. Very high rates of FASD have been found in some Indigenous populations in postcolonial nation-states, where extremely problematic drinking has arisen in a setting of socio-historical upheaval producing severe socioeconomic disadvantage [35, 36]. Some population level studies have shown a greater reduction in maternal alcohol consumption

during pregnancy among Caucasian populations than other ethnicities [30] while others have shown the opposite [28].

These puzzling relationships between maternal alcohol consumption during pregnancy and prevalence of FASD suggest at least two possible hypotheses: 1) socioeconomic status could have a ‘protective effect’ on neurodevelopment and/or the adverse neurodevelopmental effects of prenatal alcohol exposure may be amplified by factors linked with poverty such as cigarette smoking, poly-drug use, nutritional deficiencies, stressful social circumstances and poor prenatal care; and 2) the diagnosis of FASD may be more likely to be given to a child of a low SES mother and (related) diagnoses such as ADHD and autism given to children of higher SES mothers. Qualitative research has shown that Aboriginality, as well as location of residence (urban versus rural), are factors taken into account by Canadian physicians in deciding whether or not to diagnose FASD but the prevalence and impact of these possible biases is unknown [37]. Biases against diagnosis may be more likely to occur in countries such as Australia where there is no established surveillance program for FASD [38].

Social and Ethical Implications of Diagnosing FASD

Another potential source of bias is the difficulty in confirming maternal alcohol consumption during pregnancy, a necessary condition for a FASD diagnosis. In other words, there are a number of ways in which the social processes surrounding women’s drinking could impact on FASD diagnosis. First, fear of stigmatisation, incarceration, compulsory treatment or loss of custody could lead women to conceal problem alcohol use during pregnancy and avoid seeking help. The fear of FASD has in some jurisdictions created political and legal pressure for women to stop drinking alcohol during pregnancy [39, 40]. Potter [41] invokes the concept of ‘biopower’ [42] in arguing that such guidelines and legislation make pregnant women’s bodies the subject of state surveillance. This observation is perhaps most applicable to countries such as the USA and Finland where women can be subject to compulsory commitment and/or incarceration if their drugs or alcohol use is deemed to be potentially harmful to the fetus [3, 40, 41, 43]. Targeted health promotions may exhibit similar characteristics.

Some have commented that this kind of regulation of women grants certain legal rights to the fetus i.e. it produces ‘fetal citizens’ or the ‘fetal subject’, where the rights of the fetus are deemed superior to that of the mother [3, 40, 44] and the state’s assumed authority to protect the fetus overrules the interests of the mother. Some feminist philosophers and sociologists have gone as far as to criticise what they see as overstated risks of alcohol consumption during pregnancy [45, 46]. Others however, have been concerned that these criticisms are at odds with the precautionary principle when so little is known about the risks of prenatal alcohol exposure [47].

Social concerns about maternal drinking have also been linked with the ‘individualisation’ of choice which blames a mother for decisions that may affect her unborn child [39, 40]. Theorists of mothering, and scholars interested in the process of ‘mother-blame’, note that mothers, especially those from marginalised social groups, can be held solely responsible for actions that may affect their offspring, ignoring the social determinants of the mothers’ behaviour and their children’s outcomes [44, 48, 49]. Ultimately, the effects of these social pressures on women’s preparedness to disclose alcohol consumption during pregnancy may pose a major barrier to diagnosis and, in fact, place their fetuses at increased risk of FASD.

Second, concern that an FASD label may stigmatise the parent or child may reduce practitioners’ willingness to make a diagnosis of FASD [50, 51] and increase the likelihood of their making another diagnosis. The latter process is facilitated by the overlap between the behavioural phenotypes of FASD and other neurodevelopmental disorders, notably attention deficit/hyperactivity disorder (ADHD) and autism spectrum disorders [52–54]. Furthermore, in many jurisdictions a diagnosis of ADHD or autism has a major advantage over that of FASD: it increases parents’ access to services or treatments for affected children. This fact may encourage doctors to diagnose these disorders rather than FASD. However, advocates of FASD diagnosis argue that correct (and early) identification and

intervention for FASD are important in reducing its adverse outcomes and that FASD can be readily distinguished from ADHD or autism [10, 52].

The ethical and legal issues raised by diagnosing FASD on the basis of self-reported maternal drinking during pregnancy also apply to the use of putative biomarkers, such as meconium tests, for detecting prenatal exposure to alcohol in the fetus or newborn baby [55, 56]. These tests may establish if a child has been exposed to alcohol in utero more accurately than women's self-reported drinking but they raise similar considerations over the comparative 'rights' of the mother and her child.

These social factors might discourage pregnant women from disclosing their alcohol consumption and dissuade clinicians from making a diagnosis of FASD. Parental and medical advocates' belief that a diagnosis will lead to better care for an affected child is, however, increasingly a 'push' factor for a FASD diagnosis [57]. Further, for those children who have been exposed to alcohol prenatally and end up in court appointed care, FASD diagnosis can be helpful to their caregivers in understanding, accepting and working with a child's limitations. There are also instances of mothers who consumed alcohol during pregnancy expressing relief when their child is given an FASD diagnosis because it allows them to understand the reason for their affected child's learning difficulties and behaviour. Diagnosis with FASD also potentially has implications for the criminal responsibility of young offenders [58–61]. It has been estimated that up to 60 % of people diagnosed with FASD will come into contact with the criminal justice system [62]. If an individual comes to the attention of police, obtaining a diagnosis could be attractive as a possible mitigating factor in sentencing [60].

A Proposed Model for Examining Social Causes and Effects of FASD Diagnosis

The perceived social impacts of exposing maternal alcohol consumption during pregnancy and FASD diagnosis itself may systematically affect which individuals are diagnosed with FASD, thereby affecting the evolution of the FASD construct by defining it in terms of a specific subset of persons prenatally exposed to alcohol. It is because FASD diagnosis is the outcome of such a complex biosocial process that insights from sociology may be helpful in advancing biomedical research into this condition [63, 64].

Figure 1 provides a model summarising the processes affecting FASD diagnosis. This figure shows how the social and ethical implications of a FASD diagnosis interact with the evolution of FASD as a diagnostic category and the specification of its diagnostic indicators. Moving from left to right, the model shows how the application of the diagnostic criteria of FASD is filtered through the beliefs of those in the social network of a person possibly prenatally exposed to alcohol as to the possible impacts (adverse or beneficial) of making a FASD diagnosis for the mother and child. It can also be affected by the diagnostic tools available to assist the clinician in making a FASD diagnosis. The impacts of diagnosis and diagnostic tools are themselves shaped by broader cultural influences and expectations, such as beliefs about what constitutes 'good' mothering, and about the rights, autonomy and importance of informed consent of pregnant women.

Fig. 1

Interaction of social and ethical issues of FASD diagnosis with the evolution of FASD construct. These beliefs and tools both shape, and are shaped by, the individuals who are diagnosed with FASD. The characteristics of the individuals included within the diagnoses of FASD and non-FASD will feed back into the definitions of the diagnostic category, affect the identities (and therefore stigma) that become associated with the diagnosis and the biomarkers that may become available to assist in making a diagnosis.

FASD as a Model for Understanding the Interaction of Biology, Society and Environment

Given its situation within a complex web of disadvantage, the study of FASD should also be of interest to sociologists and anthropologists who want to understand how culture and society, and social inequalities in life chances, are shaped by (neuro)biology and social environments [16, 65, 66].

Here, the 'environment' is to be understood both broadly to include the availability of ethanol and the social factors that affect a pregnant women's drinking and narrowly to describe the fetal environment, and the way that alcohol and other substances ingested by the mother may affect fetal development.

As already mentioned, persons with FASD are more likely to be involved in delinquent or criminal behaviour and to develop substance use disorders [62]. Therefore, FASD may not just affect one generation but have wider social and potentially multi-generational impacts. This may make FASD both a consequence of social stratification and a health condition that reproduces structural social inequalities. The interaction between social and individual level drinking practices might cause FASD but those affected by FASD are disproportionately likely to engage in similar harmful behaviour potentially reproducing neurodevelopmental damage over multiple generations.

We do not assume that harmful drinking is confined to children who have been prenatally exposed to alcohol. Nor do we mean to imply that all prenatally exposed children will develop harmful drinking behaviours. Rather, we suggest that it is possible that certain cognitive-behavioural causes and effects may become concentrated within families over time. If this process continues across several generations, we can hypothesise that the effects of prenatal exposure to alcohol might have adverse impacts in particular subcultural and cultural domains. Thus, the study of FASD offers a means to study the specific ways that biology, environment and society influence each other to produce social inequalities and other harms.

Future Research Directions

Two important avenues for future research arise from this review: 1) the need to investigate the social causes and effects of FASD diagnosis in order to improve our understanding, detection and treatment of FASD [63, 64]; 2) the need to investigate FASD as a case study for understanding how individuals and society are shaped by bio-social interactions.

The first research avenue involves elaborating the model of FASD diagnosis as a biosocial process (Fig. 1). This model hypothesises that our understanding of FASD will be enhanced if stigma can be reduced and social supports put in place for affected individuals and their families. While efforts to reduce stigmatisation of affected individuals and their families are important in their own right, they may also be important in empowering clinicians to offer a diagnosis they may otherwise avoid for fear of stigmatising their patients and their families. Such work is needed to better understand: 1) how beliefs about the benefits and harms of FASD diagnosis impact on clinicians', and other stakeholders', decisions to confer or avoid making a FASD diagnosis; and 2) how this might vary in a range of cultural and geographical contexts. The perspectives and experiences of children and adolescents diagnosed with FASD have been neglected in research to date and this should also be a key area for future research. The diagnostic process is not confined to the dynamics between clinician and patient but encompasses a range of parties that includes parents, caregivers, youth workers and special education officers. It is important that future research enumerates the views and levels of influence of these stakeholders in order to understand the factors that encourage or discourage FASD diagnosis. This research is important in guarding against the possibility that, due to systematic biases, FASD diagnosis becomes a proxy for disadvantage rather than a diagnosis of alcohol damage to the fetus.

The second avenue for research, exploring FASD as a biosocial phenomenon, will require a longer term research commitment. Methodologies for tracing problematic alcohol use and behaviours across generations will need to be developed to understand the possible impact of prenatal exposure to alcohol on social stratification. Classical longitudinal cohort studies which collect data on alcohol consumption as well as relevant genetic data and diagnostic indicators of FASD, such as the Mater-University of Queensland Study of Pregnancy (<http://www.socialscience.uq.edu.au/musp>), are one option. However, the prospective use of this approach suffers from the length of time required to collect such data; data gathering cannot exceed the time taken for generational replenishment. This temporal barrier could be reduced by combining a longitudinal cohort study with qualitative

approaches to collecting family medical histories, including collecting oral health histories from families and archival research of medical records and other publically held medical data. Such approaches would be subject to recollection biases and missing data, nevertheless, they would provide a useful baseline evidence-base.

The implications of this research are potentially far reaching for understanding whether, and to what extent, behaviours labelled as delinquent or criminal may be public health issues rather than the simple result of 'bad character' or poor life choices by an individual or individuals. The historical depth that tracing health histories provides could usefully inform debates about the extent to which concerns about FASD reflect contemporary social anxieties versus concerns derived from increased harmful drinking by pregnant women.

Conclusions

Critical examination of FASD takes us beyond a 'nature or nurture?' debate on human behaviour. It presents an opportunity to interrogate whether, and if so then how, human action is explained by the interaction of 'nature' and 'nurture'. Such a programme of research also offers to empirically inform whether healthcare governance should be deemed central to the effective and just functioning of the state [67, 68]. Explicitly characterising the ways in which biological, environmental and social factors interact in the case of FASD provides an opportunity to empirically test whether individualist rationales for care and responsibility, that focus on human intentions and actions alone, are appropriate regulatory tools for structuring governance or, alternatively, whether it provides evidence for the need for state welfare, judicial and public health structures to exist that are responsive to the ways that human action is shaped by its interaction with biological and environmental factors.

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