Edited by Edward P. Riley, Sterling Clarren,
Joanne Weinberg, and Egon Jonsson

Fetal Alcohol Spectrum Disorder

Management and Policy Perspectives of FASD

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Preface

In October 2009, the Institute of Health Economics (IHE) staged a consensus development conference to address key questions about the prevention, diagnosis, and treatment of Fetal Alcohol Spectrum Disorder (FASD). Experts in the field presented scientific evidence to a "jury" about prevention and the social determinants that may induce drinking during pregnancy, the importance of diagnosis, the impact of FASD across a person's lifespan, and the community supports needed for those living with FASD, as well as their families. After two days of hearings, the jury developed a statement which answered eight relevant and common questions about FASD, including suggested policy changes for enhanced prevention, and for improving the lives of people with FASD and their families. That statement is available in the Appendix of this book.

During the planning of that conference, IHE invited the experts – who included researchers, clinicians, economists, epidemiologists, social workers, and judicial workers – to expand on their speeches and write chapters for a book that would aim at a worldwide health policy-making audience. An overwhelming majority of the speakers were interested, and this book is the result of their hard work.

In addition to policy makers, this book is for anyone interested in FASD, including those with the condition, family members and other caregivers, researchers, clinicians and others in healthcare and social services, and the justice sector. The chapters describe the impact of FASD on the individual, their families and society, and the many complex issues involved in the condition's prevention, diagnosis, and treatment. The book ends with personal accounts of life with FASD, written by Myles Himmelreich and Charlene Organ, that not only powerfully illustrate the challenges created by having FASD, but also serve as a reminder that FASD does not – and should never – define a person.

We would like to acknowledge the Government of Alberta FASD Cross-Ministry Committee, Canada Northwest FASD Partnership, Health Canada and the Public Health Agency of Canada for their financial support of the consensus development conference and the production of material to this book. We would also like to thank Minister Janis Tarchuk, who was Minister of Children and Youth Services at the time of the conference and Deputy Minister, Fay Orr, for their strong support. Special recognition should be given to Ms Denise Milne, who represented the Cross-Ministry Committee and assisted greatly in all aspects of the conference

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preparations. We are very grateful to the Honorable Anne McLellan for serving as Chair of the Jury for the conference, to Dr Gail Andrew, who acted as the Scientific Chair, and Ms Nancy Reynolds who acted as moderator. Special mention should be made to the Honorable Iris Evans, Minister of International and Intergovernmental Relations for Alberta, who has been a tireless supporter of initiatives to improve the lives of those affected by FASD. We would also like to give special thanks to Gail Littlejohn for her support in the editing process of this publication.

On behalf of the Institute of Health Economics (IHE) (www.ihe.ca)

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1
Prenatal Alcohol Exposure, FAS, and FASD: An Introduction

Tanya T. Nguyen, Jennifer Coppens, and Edward P. Riley

1.1
Introduction

Prenatal Alcohol Exposure (PAE) can result in a wide range of physical, psychological, behavioral, and social problems that affect the individuals, their families, and their communities. Indeed, PAE is a major public health issue placing undue burden on all aspects of society. Among the most severe outcomes of PAE is the Fetal Alcohol Syndrome (FAS), which is characterized by growth deficits, facial anomalies, and neurobehavioral problems. However, FAS is not the only detrimental outcome of heavy gestational alcohol exposure, and the majority of individuals affected by such exposure do not meet the diagnostic criteria of FAS. Currently, PAE is increasingly understood as the cause of a continuum of effects across many domains. Fetal Alcohol Spectrum Disorder (FASD) is a nondiagnostic term used to identify the wide array of outcomes resulting from prenatal exposure to alcohol. These outcomes range from isolated organ damage or subtle developmental disabilities to stillbirths and FAS. Perhaps the most pervasive outcome following prenatal alcohol exposure is what is now commonly referred to as an Alcohol-Related Neurodevelopmental Disorder (ARND). While individuals with ARND may exhibit many of the alcohol-related brain and behavioral abnormalities of FAS, they may not display the characteristic facial dysmorphia required for an FAS diagnosis. Although cases of FASD are often not as easily recognized as FAS, they can be just as serious. Unfortunately, missed diagnoses of FASD can have devastating consequences, placing heavy emotional, financial and social stresses on the individual and all parties involved (Riley and McGee, 2005).

Although the relationship between alcohol consumption during pregnancy and abnormal fetal development has been alluded to throughout history (Warren and Hewitt, 2009), FAS went unrecognized until the late 1960s and early 1970s (Lemoine *et al.*, 1968; Jones and Smith, 1973; Jones *et al.*, 1973). Since those initial defining case studies, the scientific literature on the effects of PAE on the developing fetus has grown rapidly. A simple search of pubmed.gov (U.S. National Library of Medicine) using "fetal alcohol syndrome" as a search term turned up almost 3500 citations. This research has improved our understanding of the relationship

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between alcohol exposure and developmental deficits, and has resulted in an increased social awareness of the risks of drinking during pregnancy, prevention efforts to reduce these risks, and development of intervention programs to help promote positive outcomes for individuals with FASD. However, despite our current knowledge and the progress that has been made, many challenges remain in understanding how alcohol exerts its effects, in developing efficacious and effective prevention and intervention programs, and how best to improve the daily functioning of these individuals.

1.2 History

It has been suggested that the adverse effects of alcohol on the developing fetus have been recognized for centuries. Some of the earliest references date back to Greek and Roman mythology and Judeo-Christian tradition, such as the ancient Carthaginian custom that forbade bridal couples from drinking wine on their wedding night, and the belief that alcohol consumption at the time of procreation leads to the birth of defective children (Jones and Smith, 1973). Passages in Robert Burton's *The Anatomy of Melancholy* allegedly quote Aristotle describing an association between alcoholic mothers and disabled children in *Problemata*: "... foolish, drunken and harebrained women [for the] most part bring forth children like unto themselves, morose and languid" (Burton, 1621). However, there remains much controversy regarding the validity of these claims and sources. Although many authors have assumed Burton to be quoting Aristotle's words verbatim, there is no evidence of any such statement in *Problemata*, nor in any of Aristotle's other works (Abel, 1999). Others have claimed that the Carthaginians did not truly understand that drinking during pregnancy caused problems; rather, they believed that intoxication at the *exact moment of conception* led to the birth of a deformed offspring (Calhoun and Warren, 2007).

More recent and credible historical reports, however, have documented alcohol's teratogenic effect. During the 1700s, a group of English physicians described children born to alcoholic mothers as "weak, feeble, and distempered" (Royal College of Physicians of London, 1726). A deputy medical officer of the Convict Prison in Parkhurst, England, noticed that imprisoned pregnant alcoholic women had high rates of miscarriage, and that those offspring which survived displayed distinctive patterns of birth defects (Sullivan, 1899). From these observations, Sullivan concluded that alcohol had a direct effect on the developing embryo.

Despite these observations and early animal studies supporting an association between gestational alcohol exposure and adverse outcomes (e.g., Stockard, 1910), the first clinical accounts of alcohol's teratogenic effects were not published until the late 1960s. In 1968, Lemoine and colleagues published their report entitled "Outcome of children of alcoholic mothers" (Lemoine *et al.*, 1968), which established a connection between maternal alcohol consumption during pregnancy and abnormal fetal development, describing common problems of children born to

mothers who drank heavily during pregnancy. Unfortunately, the authors did not present any diagnostic criteria to facilitate the recognition of fetal alcohol effects in future cases (Hoyme *et al.*, 2005), and the paper was published in French, which limited its wide availability. As a result, FAS remained unrecognized for five more years until Jones and colleagues reported a series of case studies which documented consistent patterns of physical and developmental abnormalities in infants and children exposed to alcohol *in utero* (Jones and Smith, 1973; Jones and Smith, 1975; Jones *et al.*, 1973). These authors coined the term "fetal alcohol syndrome," and laid the foundation for the diagnosis of this disorder.

1.3
Diagnosing the Effects of Prenatal Alcohol Exposure

1.3.1
Fetal Alcohol Syndrome

There are several suggested diagnostic schemas for FAS (e.g., Bertrand *et al.*, 2004; Chudley *et al.*, 2005; Hoyme, 2005) and, while there are minor differences between them, all require anomalies in three distinct areas: (i) prenatal and postnatal growth deficits; (ii) facial dysmorphology; and (iii) central nervous system (CNS) dysfunction. Typically, growth retardation is defined as evidence of prenatal or postnatal weight or height at or below the 10th percentile, after correcting for age, gender, race, and other appropriate variables. The Canadian guidelines also recommend evidence of a disproportionately low weight-to-height ratio at or below the 10th percentile. Most guidelines recommend three essential dysmorphic features – a smooth philtrum, a thin upper vermillion border, and small palpebral fissures – although the revised Institute of Medicine (IOM) guideline requires only two of the three characteristics (Hoyme *et al.*, 2005). Finally, a diagnosis of FAS requires evidence of CNS abnormality. Within this criterion, the diagnostic schemas differ more substantially. For example, the revised IOM guideline only requires evidence of structural brain abnormalities, such as diminished head circumference at or below the 10th percentile. The CDC criteria are more extensive, outlining structural, neurological, and functional CNS dysfunction. Structural anomalies may be evidenced by the two criteria delineated in the IOM guidelines, as well as brain abnormalities observed with neuroimaging techniques. Seizures or other signs of neurological damage not attributable to postnatal insult may qualify as evidence of neurological problems. Lastly, functional abnormalities are defined as a global cognitive deficit (such as a decreased IQ), or deficits in three different functional CNS domains, which include cognition, behavior, executive functioning, and motor functioning. The Canadian guidelines outline eight domains that must be assessed: hard and soft neurologic signs; brain structure; cognition; communication; academic achievement; memory; executive functioning and abstract reasoning; and attention deficit/hyperactivity. Diagnosis requires evidence of impairment in three of these domains.

1.3.2
Fetal Alcohol Spectrum Disorder(s)

It is now recognized that there is a spectrum of deficits arising from PAE; FASD is the umbrella term used to describe this broad range of outcomes. Since the term FASD is not diagnostic, some of the guidelines (Chudley *et al.*, 2005; Hoyme *et al.*, 2005) use the terms ARND or ABRD (alcohol-related birth defect) to describe these FASDs. ARBD is a term which refers to individuals with a confirmed history of PAE and who display congenital birth defects, such as physical malformations or organ abnormalities. The ARND classification refers to individuals with a confirmed history of PAE who have behavioral and cognitive deficits related to CNS dysfunction. For example, an association between maternal alcohol use and sudden infant death syndrome (SIDS) has been suggested (e.g., Burd and Wilson, 2004). This would make SIDS an FASD in those cases where PAE was suspected, if other causes could be ruled out. Similarly, an increased risk of congenital heart defects has been associated with prenatal alcohol exposure; thus, such heart defects might be considered an FASD/ARBD if the mother drank heavily during pregnancy. Behavioral problems in children exposed to alcohol *in utero*, but who do not meet the diagnostic criteria of FAS, are perhaps the most commonly cited type of FASD/ARND.

1.4
Risk factors influencing FAS and FASD Conditions

The variation in the range of phenotypes of individuals with PAE suggests that alcohol's teratogenic effects can be moderated or exacerbated by other variables. Not every woman who drinks heavily during pregnancy will give birth to a child with an FASD (Warren and Foudin, 2001), and not all children with an FASD have the same deficits (Bertrand *et al.*, 2004). In fact, there have been reports of discordance among twin pairs in regards to FAS (Warren and Li, 2005; Streissguth and Dehaene, 1993). Numerous biological and environmental factors have been shown to influence the effects of alcohol on the developing fetus, with the most obvious and important factors being those related to the nature of the PAE. The amount of alcohol consumed is highly correlated with the severity of outcome; typically, a higher level of alcohol consumption, along with longer duration of exposure, will generally lead to more adverse effects (Bonthius and West, 1988; Maier, Chen, and West, 1996). However, a linear relationship between dosage and severity may not always be expected. Studies in both animals and humans have revealed that the pattern of alcohol consumption may moderate dose effects. A binge-like exposure results in more severe neuropathology and behavioral alterations than does chronic exposure (Bonthius, Goodlett, and West, 1988), and those women who binge drink are at a higher risk of having a child with neurobehavioral deficits than those who drink chronically during pregnancy (Maier and West, 2001). In fact, Jacobson *et al.* (1998) have proposed that describing consumption by the average number of drinks per occasion is more useful in predicting outcome than