



Fetal Alcohol Spectrum Disorders: What Pediatric Providers Need to Know **CE**

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ABSTRACT

Prenatal alcohol exposure is the cause of fetal alcohol spectrum disorders (FASDs), the prevalence of which is similar to that of other developmental disabilities like Down syndrome and autism. Children, adolescents, and adults who live with the disabilities associated with prenatal alcohol exposure face extraordinary challenges throughout their lives. Pediatric providers need to be able to identify patients with FASD because early recognition and intervention is known to improve life outcomes for affected individuals. The purposes of this continuing education activity are to report what is known about the prevalence of FASDs; to detail the spectrum of problems experienced by affected individuals; and to suggest specific strategies for preventing, identifying, and managing FASDs in clinical practice. *J Pediatr Health Care.* (2017) 31, 594-606.

KEY WORDS

Developmental disability, FASDs, fetal alcohol spectrum disorders, neurobehavioral disorder, prenatal alcohol exposure

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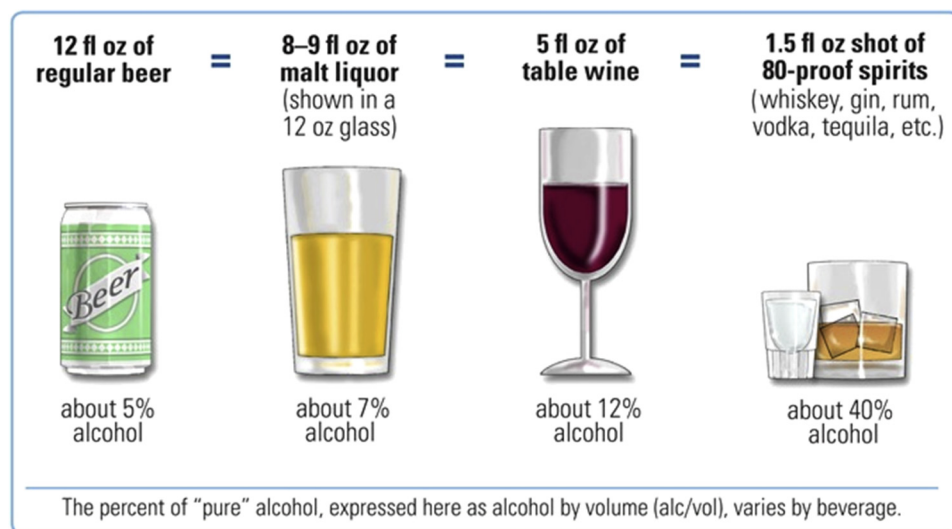
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OBJECTIVES

1. Discuss why the prevalence of drinking by pregnant women suggests the need for universal screening for prenatal alcohol exposure.
2. Describe the spectrum of physical and neurobehavioral problems experienced by individuals with FASD.
3. List factors identified from a history and physical that should alert Pediatric PCPs to the need for referral for an FASD evaluation.
4. Discuss Pediatric primary care management of patients with FASD.
5. List factors that promote optimal development for Pediatric patients with FASD.

Prenatal alcohol exposure (PAE) is the leading known cause of preventable birth defects and developmental disabilities (Burd, 2016; McClellan & Expert Panel on FASD, 2009). Many factors influence the severity of fetal alcohol spectrum disorders (FASDs); however, PAE is the necessary component, making FASDs completely preventable (May & Gossage, 2011; Williams, Smith, & AAP Committee on Substance Abuse, 2015). FASD is an umbrella term describing the range of negative effects that can occur including physical and central nervous system (CNS) malformations, deficits in growth, and neurocognitive impairments (e.g., in behavior, self-regulation, and adaptive skills). Specific disorders include fetal alcohol syndrome (FAS), partial FAS, alcohol-related birth defects, and neurobehavioral disorder associated with PAE (ND-PAE; Hagan et al., 2016; Hoyme et al., 2016; Williams et al., 2015). Although FASDs are permanent conditions, with early recognition and intervention specific symptoms are manageable, and affected individuals' life outcomes can be improved (Bertrand & Interventions for Children with Fetal Alcohol Spectrum Disorders Research Consortium, 2009).

FIGURE 1. Percentage alcohol by volume.



This figure appears in color online at www.jpeds.org.

Pediatric providers need to be able to identify patients with FASD and manage their care. The purposes of this article are to report what is known about FASDs; to detail the spectrum of problems experienced by affected individuals; and to suggest specific strategies for preventing, identifying, and managing FASDs.

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TERATOGENIC EFFECTS OF ALCOHOL

The fetus is sensitive to alcohol's teratogenic effects throughout gestation. At 1 to 2 hours after maternal alcohol ingestion, the fetal blood alcohol concentration reaches a level equivalent to that of the mother, and the process of amniotic reuptake prolongs the time of PAE. Elimination of alcohol from the fetus is dependent on the mother's metabolic capacity, which varies considerably among pregnant women (Burd, Blair, & Dropps, 2012; May & Gossage, 2011). A number of mechanisms contribute to the physical and neurologic effects of PAE on the developing fetus (Gray, Mukherjee, & Rutter, 2009; Kane, Phelan, & Drew, 2012). Toxic byproducts of alcohol metabolism accumulate, disrupting the growth, division, and survival of cells throughout the body. Depending on the timing and severity of PAE, associated malformations can occur in the cardiac, skeletal, renal, ophthalmic, auditory, and neurologic systems (Hoyme et al., 2016). The sensitivity of the fetus to alcohol varies depending on

the dose, pattern, and timing of PAE, as well as on general fetal health and wellness (May et al., 2013; O'Leary & Bower, 2012).

Animal studies have consistently shown that moderate to heavy PAE induces negative effects on neurodevelopment, and several studies have suggested that low to moderate PAE can produce functional damage on the developing fetal brain without obvious effects on other systems (Gray et al., 2009). Experimental human studies to determine the impact of the dose, timing, and pattern of PAE on outcomes are not ethically feasible; however, case-control and cohort studies with humans have shown consistent associations between moderate to heavy PAE and poor neurodevelopmental outcomes (Gray et al., 2009; O'Leary & Bower, 2012). The evidence for adverse effects from low to moderate PAE from observational studies is less robust because of challenges in accurately measuring the dose, pattern, and timing of alcohol consumption from self-reports, controlling for the presence of confounding factors, and a lack of consensus on diagnostic criteria in the scientific literature (Gray et al., 2009; O'Leary & Bower, 2012). Despite these methodologic challenges, evidence on the magnitude of the problem and the spectrum of disabilities associated with PAE support advising all women of childbearing age that there is no known "safe" level of alcohol consumption during pregnancy and that abstinence is the healthiest choice (Hoyme et al., 2016; Williams et al., 2015).

Several maternal factors affect the severity of effects from PAE. These include the pattern of maternal drinking (timing, quantity, and

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