Fetal alcohol syndrome (FAS) is a clinically identifiable diagnosis, consisting of pre- and/or postnatal growth retardation (below the third percentile), characteristic facial features, including a thin upper lip, indistinct philtrum and short palpebral fissures (two standard deviations below normal for age), and neurobehavioral abnormalities (Abel, 1998; Jones et al., 1973; Plant, 1987; Sokol et al., 2003). Although FAS is the leading known cause of mental retardation in the USA (Abel and Sokol, 1986), brain injury involving milder forms of cognitive dysfunction, and more subtle and complex patterns of neurological impairment, occur in ~30–40% of children born to heavy drinkers, with or without the classic diagnostic features of FAS (Koren et al., 2003; Mattson et al., 1997). These neurodevelopmental disorders and other wide-ranging physical and behavioral problems occurring in conjunction with or in the absence of classic FAS features, are now subsumed under the broader umbrella term, fetal alcohol spectrum disorder (FASD). Many of these alcohol-related neurological impairments, e.g. hypotonia, clumsiness, unsteady gait, fine motor impairment, poor eye-hand coordination (Abel, 1998; Aronson et al., 1985; Barr et al., 1990; Jones et al., 1973; Marcus, 1987), stiff muscles and muscle spasms characterized as ‘spasticity’ (Beattie et al., 1983) are also hallmarks of cerebral palsy (CP; Lin, 2003). Despite these commonalities, the American College of Obstetricians and Gynecologists (ACOG) and the American Academy of Pediatrics (AAP; ACOG, 2003) in a joint statement concluded that ‘there is no known relationship between alcohol consumption and cerebral palsy’ (p. 20). However, direct and indirect evidence warrant a revision of that conclusion.

CP is a group of disorders characterized by non-progressive abnormal control of movement or posture (Lin, 2003; MacLennan, 1999). The incidence is 2.1–2.5 per 1000 live births (Hagberg et al., 2001). Although etiology remains controversial, birth trauma accounts for only a minority of occurrences of CP (Dolk et al., 2006). Most cases (~75%) arise primarily during the third trimester of pregnancy (Dolk et al., 2006; MacLennan, 1999). Prospective and retrospective epidemiological studies as well as several case studies published previous to the ACOG/AAP report, as well as a more recent study, strongly suggest an association between prenatal alcohol exposure and CP, such that CP could reasonably be included as one of the manifestations of FASD. Studies in animals have also documented a causal connection between prenatal alcohol exposure and impaired gait and balance that in humans could be regarded as indicative of CP (Abel and Dintcheff, 1978; Hannigan et al., 1993; Meyer et al., 1990).

The strongest evidence supporting an association between FAS and CP comes from a Swedish prospective study in which one-third of all pregnancies in Goteborg (population 450,000) were followed at antenatal clinics between 1977 and 1978. Four of 48 (8.3%) children with FAS were diagnosed with CP (one hemiplegia and three ataxia) compared with only one of the 7600 children born in the general Swedish population during the same period (Olegard et al., 1979). Less rigorous studies lacking control data noted two cases of CP among 20 children born to mothers with a history of chronic alcoholism in Australia (Lipson et al., 1983) and two cases among 40 FAS children in Scotland (Beattie et al., 1983). CP had also been noted prior to 1992 in several individual case studies of FAS (Bierich et al., 1976; Chan et al., 1991; Palmer et al., 1974). More recently, a 4% prevalence of CP was reported for children with FAS in North Dakota (although no details were given as to ascertainment; Burd et al., 2003). In the most recent retrospective study, from South Africa, 4 of 242 children with CP had FAS, a prevalence of 5.7% (Van Toorn et al., 2007). While these latter studies generally lack experimental vigor and some are sparse in detail (e.g. Burd et al., 2003), they are nevertheless consistent in pegging the prevalence of CP among children with FAS at between 2 and 10%.

A major problem in detecting FASD in children with CP is that neither CP or FASD are usually identified before 1 year of age (MacLennan, 1999). This means that unless a child also has the facial features characteristic of FAS, or a history of maternal alcohol consumption is known, the association will likely go unnoticed. Indirect evidence, that CP may be a concomitant of prenatal alcohol exposure, is the presence of the many common secondary symptoms noted in Table 1.

While there are many possible mechanisms in the etiologies of both CP and FAS/FASD a mechanism both appear to have in common is prenatal oxidative stress (Abel and Hannigan, 1995; Goodlett and Horn, 2001; Korzeniewski et al., 2008; Lin, 2003). Although Korzeniewski et al. (2008) contend that inflammatory phenomena are the more probable causes of CP (Yoon et al., 2000), this does not preclude the involvement of oxidative stress since tissue hypoxia results in oxygen free radicals which in turn lead to inflammatory responses to deal with the injury (Stamler et al., 1997; Vink et al., 2005). Conceivably, both CP and FAS/FASD are conditions related to a more general fetal inflammatory response syndrome (Gomez et al., 1998).
CONCLUSIONS AND IMPLICATIONS

Perhaps one of the reasons an association between CP and prenatal alcohol exposure has largely gone unnoticed is that clinicians/researchers have been overly attentive to the facial features currently required for the FAS diagnosis. However, as previously noted, prenatal alcohol exposure produces a spectrum of clinical conditions that include an increased risk for CNS deficits which can occur in the absence of FAS (Koren et al., 2003; Mattson et al., 1997). Another reason, the association between CP and in utero exposure has not received more scrutiny is that like FASD, CP is an umbrella term for a spectrum of motor dysfunctions. The previously cited evidence indicates that CP may occur in as many as 2–10% of cases of FASD, but has not been widely recognized as such.

Recognition of the occurrence of CP in conjunction with heavy drinking during pregnancy has important implications for medical malpractice. The most common reason, obstetricians are sued for medical malpractice, is the claim that they caused a child to be born with CP (ACOG, 2003). Evidence that such damage occurred prenatally from alcohol is strongly preemptive of such claims. Any case involving CP should, therefore, consider an evaluation of FAS/FASD. In the absence of FAS/FASD, evidence of prenatal alcohol exposure and its associated malformations as causal would be suggestive but much harder to prove since the neuroimaging correlates are similar, but a maternal history of drinking during pregnancy should be considered in cases of CP.

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Aronson M, Kyllerman M, Sabel KG. (1990) *Prenatal alcohol exposure and its associated malformations as causal would be suggestive but much harder to prove since the neuroimaging correlates are similar, but a maternal history of drinking during pregnancy should be considered in cases of CP.*

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