Prenatal Smoking Exposure, Low Birth Weight, and Disruptive Behavior Disorders

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ABSTRACT

Background: Prenatal problems are among theorized etiologies for child disruptive behavior problems. A key question concerns whether etiological contributors are shared across the broad range of disruptive psychopathology or are partially or largely distinct. **Method:** We examined prenatal smoking exposure and low birth weight as risk factors for attention-deficit/hyperactivity disorder (ADHD), oppositional defiant disorder (ODD), and conduct disorder (CD) in a population-based longitudinal design from ages 6 to 17 years. Multiple informants were used, with emphasis on parent and teacher report for ADHD, parent- and self-report interview for ODD, and self-report interview for CD, in keeping with evidence about the most valid sources of information for these respective syndromes. **Results:** The association of prenatal smoking exposure with ADHD was highly confounded by family variables. In contrast, low birth weight independently predicted ADHD, even with family variables statistically controlled. The opposite pattern appeared for ODD and CD. Prenatal smoking exposure but not low birth weight predicted ODD independent of potential confounding variables. Prenatal smoking exposure also predicted CD. The effect on CD was via its effect on ODD. **Conclusion:** Prenatal smoking exposure may contribute to ODD and via that route to later CD, but does not have a specific association with ADHD in this sample. Findings have implications for distinct etiological contributors to these often comorbid aspects of the disruptive behavior domain. *J. Am. Acad. Child Adolesc. Psychiatry*, 2007;46(3):362–369. **Key Words:** attention-deficit/hyperactivity disorder, oppositional defiant, conduct disorder, prenatal nicotine, prenatal smoking, low birth weight.

Numerous studies have linked prenatal factors with subsequent disruptive behavior disorders, including attention-deficit/hyperactivity disorder (ADHD) and conduct problems, although few studies have examined oppositional defiant disorder (ODD). Among the most well-replicated early correlates of these disruptive behavior outcomes have been prenatal smoking exposure and low birth weight (LBW). Few studies have looked at these suspected causes together, in particular to evaluate whether LBW accounts for smoking effects, and few studies have looked at ADHD and other disruptive behaviors in the same sample to gauge relative specificity of effects across disruptive behavior disorders. Clarifying these effects is of great importance because it can illuminate etiological theories of related but distinct forms of psychopathology. Although symptoms of ADHD are highly heritable, multiple etiologies are likely (Levy et al., 1997; Sherman et al., 1997), including a role of prenatal risk factors. Likewise, the etiology of aggression and conduct problems is likely multifactorial (Waldman et al., 2002).

Some 11.4% of women in the United States reported that they smoked cigarettes during pregnancy in the year 2002 (Centers for Disease Control and Prevention, 2004). Prenatal smoking exposure has been associated with child externalizing problems in prospective population studies that included multiple methods of assessing child behaviors, after controlling for socioeconomic status, other demographic factors, maternal alcohol or drug use, and maternal depression (Brook et al., 2000; Williams et al., 1998). Findings, however, have not been entirely consistent. For example, Silberg et al. (2003) reported that after familial (genetic)

Accepted August 22, 2006.

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0890-8567/07/4603-0362©2007 by the American Academy of Child and Adolescent Psychiatry.

DOI: 10.1097/01.chi.0000246054.76167.44

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transmission of antisocial traits was controlled, there was no independent association between maternal prenatal smoking and offspring conduct problems in a sample of twins. Button et al. (2005) found that prenatal smoking effects on conduct problems and ADHD were independent of each other and of genetic influences. Effects on ODD have scarcely been examined and may account for the observed effects on conduct disorder (CD) because ODD often precedes CD developmentally.

Many of the largest population-based studies did not assess ADHD. However, prenatal smoking exposure has been associated with ADHD in case-control designs (Milberger et al., 1996, 1998), a small number of large prospective cohort designs (Kotimaa et al., 2003; Linnet et al., 2005), and several smaller studies. Linnet et al. (2003) reviewed 24 studies and concluded that a link between maternal smoking in pregnancy and child ADHD could not be ruled out.

Despite these emerging findings, a number of issues remain. First, few studies examined other disruptive behavior disorders along with ADHD to clarify specificity of effects to ADHD versus CD or ODD, which often co-occur with ADHD. Second, parental behavioral problems were controlled only in some studies, with inconsistent results. Thapar et al. (2003) found that maternal smoking during pregnancy still had a significant, environmentally mediated effect on ADHD symptoms after genetic factors, birth weight, and child CD were statistically controlled. However, in another twin study, Knopik et al. (2005) found that smoking was not related to ADHD after controlling for genetic effects and confounders.

A second issue is that LBW itself is an established outcome of smoking during pregnancy (Kramer, 1987). However, LBW exerts effects on development independently of smoking exposure (Breslau et al., 2005). LBW (defined as <2,500 g) has been linked relatively specifically with ADHD and behavioral attention problems (Breslau et al., 1996; Pinto-Martin et al., 2004). Indeed, child problems with inattention and hyperactivity/impulsivity, as well as increased incidence of ADHD, are among the most reliable outcomes of LBW in prospective population-based designs (Foulder-Hughes and Cooke, 2003) as well as retrospective case-control studies (Mick et al., 2002). Breslau et al. (1996, 2000) examined neuropsychiatric outcomes of LBW in a large-scale, stratified, random sample of children followed longitudinally. They reported associations between children's verbal, spatial, fine motor, tactile, intellectual, and attentional abilities and birth weight up to 3,300 g (Breslau et al., 1996). At age 6, children with LBW had increased rates of ADHD but not anxiety or ODD. This relationship was stronger in inner-city versus suburban children (Breslau et al., 1996), which could suggest LBW by environment interactions. At age 11, LBW children had an excess of teacher-rated symptoms of inattention/hyperactivity in the urban setting (Breslau and Chilcoat, 2000), but this relationship was not seen in the suburban setting; these findings suggest that some environments interact with or are protective in relation to LBW effects on attention problems.

It is important to clarify these etiological relationships for the *DSM* constructs of ODD, CD, and ADHD at later ages after children have passed through the period of onset risk. When ADHD, ODD, and CD have been examined together in twin studies, evidence of partially distinct as well as overlapping etiological influences has emerged (Waldman et al., 2002). Clarifying the degree of specificity of prenatal exposure to maternal smoking and of LBW to these overlapping outcomes may contribute nosologically relevant evidence. Indeed, as the *DSM-V* comes under discussion, there has been increasing recognition of the importance of potential unifying etiological mechanisms that may cut across related disorders and help explain their co-occurrence as well as their taxonomic relationships and distinctions (Krueger, 1999).

We address a series of questions concerning whether prenatal smoking effects are specific to ADHD versus ODD or CD when LBW is well represented and can be examined simultaneously in a group of children followed to age 17; in other words, completely through the period of risk of onset of ADHD, ODD, and CD. We hypothesized, based on prior literature, that LBW would be related to ADHD but not to ODD or CD and examined the relationship of prenatal smoking exposure to ADHD, ODD, and CD.

METHOD

Sample

Data are from a longitudinal study of LBW (≤2,500 g) and normal birth weight children who were assessed at ages 6, 11, and 17. We targeted the 1983 to 1985 birth year cohorts of newborns

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