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Prenatal smoking and genetic risk: Examining the childhood origins of externalizing behavioral problems

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ABSTRACT

An ever-growing body of research has begun to focus closely on the role of prenatal smoke exposure in the development of conduct problems in children. To this point, there appears to be a correlation between prenatal nicotine exposure and behavioral problems. We build on this prior research by examining the coalescence of prenatal smoke exposure and genetic risk factors in the prediction of behavior problems. Specifically, the current study analyzed data from a nationally representative sample of twin pairs collected during early childhood. Our findings suggested that an interaction existed between prenatal smoke exposure and genetic risk factors which corresponded to increased risk of behavior problems. These findings provide evidence of a gene–environment interaction, in that prenatal smoke exposure conditioned the influence of genetic risk factors in the prediction of aggressive behavior. Interestingly, the association between genetic risk and prenatal smoking was sex-specific, and only reached statistical significance in females. Given the nature of our findings, it may shed light on why heterogeneity exists concerning the relationship between prenatal smoke exposure and externalizing behavioral problems in children.

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1. Introduction

Development in utero represents one of the most critical time periods for human growth and wellbeing (Davis and Sandman, 2010; Lillycrop and Burdge, 2011). Not surprisingly, exposure to certain toxins during pregnancy has a well-documented association with a host of adversities that may persist beyond childhood and pose problems across years of development. Prenatal exposure to alcohol, for instance, has been linked to a variety of deleterious outcomes including fetal alcohol syndrome, preterm delivery, miscarriage, stillbirth, and sudden infant death syndrome (Bailey and Sokol, 2011). Similarly harmful effects have been uncovered for lead (Needleman, 2004), mercury (Grandjean et al., 1997), as well as other heavy metal toxins (Lewis et al., 1992). In short, fetal development represents a time period worth guarding against exposure to noxious agents that might derail typical growth patterns.

Building on the knowledge base regarding the import of the prenatal environment, one of the most frequent subjects of research involves the study of nicotine exposure (Olds, 2006). Prenatal exposure to maternal smoking specifically (perhaps because it is the most common method of ingesting nicotine) has attracted a large amount of attention from scholars across a range of academic disciplines (Boutwell and Beaver, 2010; Boutwell et al., 2011b; Ellis et al., 2012; Ernst et al., 2001; Wakschlag et al., 2006). In the wake of all of this research, a consistent link between smoking while pregnant and a host of physiological, neurological, and behavioral deficits has emerged for both non-human animals and humans (DiFranza et al., 2004; Lambers and Clark, 1996; Olds, 1997; Wakschlag et al., 2002).

2. Causal, spurious, or something else?

Ultimately, there are three possible reasons why exposure to maternal smoking might correlate with the emergence of antisocial behavior. First, prenatal smoking may impact the development of behavioral problems after birth by negatively impacting cognitive development.

The numerous teratogens in cigarette smoke are known to collect in the placenta and expose the developing fetus to a myriad of chemicals at levels approximately 15 percent higher than that of

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the mother (Lambers and Clark, 1996). Animal models have provided evidence that nicotine smoke directly alters brain structure and function—thereby offering one particular pathway from smoke exposure to behavior (Ernst et al., 2001). Developmental problems associated with exposure to cigarette smoke often emerge when the child is still an infant and can include lower levels of arousal, decreased autonomic regulation (e.g., lower heart rate, see Raine, 2002), and habituation to stimuli (Olds, 1997).

Importantly, similar deficits to those mentioned above have been observed in older children, adolescents, and adults with higher levels of antisocial behavior (Armstrong and Boutwell, 2012; Armstrong et al., 2009; Raine, 2002; Raine et al., 1997). Children exposed to prenatal smoking, for instance, were found to have increased rates of delinquency across eight offense types (significant t-values range from 1.73 ($p < .05$) to 4.51 ($p < .001$); Ellis et al., 2012), and greater levels of behavioral problems at age nine (OR = 1.35–1.37 for matched samples of smokers and non-smokers; McCrory and Layte, 2012). Moreover, a prospective longitudinal study garnered direct support for a link between prenatal smoking and chronic offending (individuals exposed to prenatal smoking were 1.16 times more likely to be LCP offenders; Piquero et al., 2002).

Second, and contrary to the first possibility, is that the relationship between prenatal smoking and behavioral problems may be spurious. Despite a wealth of research linking prenatal exposure to cigarette smoke to persistent antisocial behavior, not all children exposed to smoke prenatally consistently engage in adverse forms of conduct (Beaver et al., 2010; Boutwell and Beaver, 2010; Wakschlag et al., 2006). An emerging line of research, for example, suggests that once key confounders (e.g., variables that might simultaneously predict both maternal smoking and offspring behavioral problems [or otherwise contribute to a selection bias], such as parental depression, substance use, and antisocial behavior, and labor/delivery complications) are controlled, the relationship between smoking and behavior dissipates entirely (Boutwell and Beaver, 2010; D'Onofrio et al., 2012; Maughan et al., 2004). Maughan and her team (2004) found that the relationship between prenatal smoking and behavior problems was almost entirely washed away when parental antisocial behavior and genetic factors were held constant.

The third and more nuanced pathway from smoking to behavior is that prenatal exposure to cigarette smoke might condition the effects of other risk factors (e.g., biological and genetic factors). Put differently, exposure to prenatal smoking might represent a risk factor capable of moderating the influence of certain genetic (and perhaps other environmental) risk factors for antisocial behavior. Moreover, such an effect may further explain why not all children who are exposed to prenatal smoking exhibit overt conduct and behavioral difficulties (Boutwell and Beaver, 2010). Prenatal smoking may only predict behavior problems when genetic/biological predispositions for antisocial behavior are also present.

3. Evidence for an interaction effect

A mounting line of research is offering up evidence that biological factors (including genes) may interact with environmental risk factors to predict behavior problems (Beaver et al., 2009; Gibson and Tibbetts, 2000; Moffitt, 1993, 2006; Tibbetts and Piquero, 1999). Indeed, some research has suggested that the most chronic criminal offending results from neuropsychological and environmental risk factors coalescing in certain individuals (Moffitt, 1993). The general hypothesis is that biological risk factors will predict aggressive or antisocial behavior, but only in the presence of an environmental trigger (Boardman et al., 2014).

In one of the more notable tests of an interaction between biological and environmental correlates of antisocial behavior, Raine et al. (1994) reported that approximately 20 percent of all violent offenses were restricted to individuals who suffered from the presence of birth complications *and* maternal rejection. Importantly, the idea that the relationship between prenatal smoking and childhood antisocial behavior may be explained through such an interaction may have physiological support.

Cigarette smoke has been found to prompt genes to alter the ways in which cells multiply and differentiate during prenatal development (Ernst et al., 2001). In animal studies—which have been used for decades and represent a viable model for studying the impact of noxious substances (Hajar, 2011; American Physiological Society, 1994)—nicotine has been found to directly influence neurodevelopment, reduction of brain cells, and underresponsive neurotransmitter systems (Landmesser, 1994; Navarro et al., 1989; Slotkin et al., 1997; see Ernst et al., 2001 for a review). Further evidence can be found in research utilizing molecular genetic techniques which have begun to untangle specifically which genes may be affected by prenatal smoking (Altink et al., 2008; Wakschlag et al., 2010; Wiebe et al., 2009).

A final caveat concerning the interaction between biological risk factors and prenatal smoking is that the moderating effects of smoking may not work identically in both males and females. In order to better contextualize the current study, it is important to investigate how interactive effects might, and sometimes do, differ by sex. Recent findings in the prenatal smoking literature, for instance, indicate a moderating effect of prenatal smoking for females but not males (Ellis et al., 2012; Wakschlag et al., 2010). Research examining the confluence of variants in the MAOA gene, prenatal smoking, and sex found that prenatal exposure to cigarettes influenced behavior problems through different genetic polymorphisms for males and females (Wakschlag et al., 2010).

Other evidence for similar "gendered" interactions is not without precedent. Utilizing data drawn from a large national sample of children, Boutwell et al. (2011a) reported evidence that the use of corporal punishment conditioned the influence of genetic risk factors on externalizing problems for males but not females. A similar set of findings emerged from the data analyzed by Paaver et al. (2008). These researchers examined data from 222 boys and 261 girls who participated in the European Youth Heart Study (EYHS). The results of the study suggested that the impact of a measured gene (5-HTTLPR) on impulsive behaviors was moderated by the presence of family adversity. Importantly, the effect was isolated to females in the sample; males were unaffected by the interaction between genetic risk and adverse familial influences.

Recently, Vaske et al. (2012) uncovered evidence that a polymorphism in the serotonin transporter gene (5-HTTLPR) conditioned the influence of childhood abuse and neglect on a range of criminogenic outcomes, including marijuana use, alcohol abuse, and criminal involvement. Vaske and her team analyzed data drawn from the National Longitudinal Study of Adolescent Health and found that the genetic variant in question conditioned the influence of abuse, but only for females in the sample. There was no evidence that 5-HTTLPR moderated the impact of abusive experiences on later life criminality for males. It is important to keep in mind that discrepancies in prior research could be a consequence of the subpopulation in which the study was conducted; however, findings from this and other studies demonstrate that sex may further moderate genetic effects on various outcomes.

Currently, the bulk of research examining the association between prenatal exposure to cigarette smoke and persistent childhood behavior problems has produced varying conclusions, and the true nature of the effect of prenatal smoking on behavioral problems remains in debate. The prenatal environment has been linked to early onset developmental problems, but few studies have considered whether prenatal smoke exposure moderates the effect of other biological risk factors. As a result, the aim of the current study is to

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