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A shared pathway of antisocial risk: A path model of parent and child effects

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ABSTRACT

Purpose: The current study explores the possibility that the antisocial traits and behaviors of parents and children have persistent, bidirectional effects on each other that contribute to a pathway of shared risk.
Method: We employ data from the Early Longitudinal Child Survey, Kindergarten (ECLS-K), a national, longitudinal study of children. Path analysis was used to test our hypothesis.
Results: The results suggest that there is substantial stability in antisocial traits of parents and children over time. While only child risk was found to predict parent risk during early childhood, both parent risk and child risk influenced each other from late childhood to early adolescence.
Conclusions: Stability in the antisocial traits and behaviors of parents and their children is a function of both parent-driven and child-driven effects over time, with child and parenting effects being differentially relevant depending on the life stage examined.

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Introduction

An extensive line of research has examined elements of family socialization as important predictors of child misbehaviors, including crime and delinquency (Alarid, Burton, & Cullen, 2000; Jang, 1999; Loeber & Stouthamer-Loeber, 1986). This body of research is at least partly predicated on a number of criminological theories suggesting that the family environment exerts a significant influence on child antisocial outcomes, including Hirschi's (1969) social bond theory, Sampson and Laub's (1993) age-graded theory of informal social control, and Gottfredson & Hirschi's general theory of crime (1990). These theories posit that family environments characterized by a lack of warmth, support, appropriate discipline, and parental vigilance will yield poorly-adjusted children that are more likely to become ensnared in a delinquent lifestyle. Importantly, a wealth of criminological research has found that children who grow up in adverse family environments are more prone to develop low self-control and engage in misconduct (Alarid et al., 2000; Booth, Farrell, & Varano, 2008; Hay, 2001; Hay, Fortson, Hollist, Altheimer, & Schaible, 2006; Jang, 1999; Loeber & Stouthamer-Loeber, 1986; Wright & Cullen, 2001). As a result, the role of parental socialization in shaping a child's delinquent trajectory is frequently regarded as both evident and potent within the field of criminology.

Despite the emphasis on parent-driven effects among criminologists, a number of scholars from other disciplines (e.g., behavioral genetics) suggest that the unidirectional family-to-child causal explanation of the association between troubled family life and child criminality is likely oversimplified (Harris, 1998; Pinker, 2002; Rowe, 1994). Specifically, they point to the need for studies of the family environment to account for genetic influences and child-driven effects in order to garner more accurate estimates of the impact of family socialization on child traits and behaviors. These researchers have found that, once genetic influences are taken into account, the effect of parental socialization on child antisocial outcomes is either greatly attenuated or eliminated entirely (Beaver, Ferguson, & Lynn-Whaley, 2010; Harris, 1998; Wright & Beaver, 2005; Wright, Beaver, Delisi, & Vaughn, 2008). This research highlights the fact that, in most cases, parents and children share both the family environment and genetic material, both of which may account for similarities in their traits and behaviors. Furthermore, the work of behavioral geneticists and child psychiatrists has also underscored the ability of children to play a role in shaping the family environment (Boivin et al., 2005; Ge et al., 1996; Kendler & Baker, 2007; Pardini, Fite, & Burke, 2008; Riggins-Caspers, Cadoret, Knutson, & Langbehn, 2003). The implication of this line of research is that, apart from being a cause of child behavior, parenting might also be a reaction to a child's traits and behaviors (Beaver & Wright, 2007; Moffitt, 2005). These child effects may be largely explained by an underlying process known as an evocative gene-environment correlation, wherein genetic risk within a child, which informs their traits and behaviors, can elicit maladaptive environmental responses, including harsher treatment from family members (Moffitt, 2005; Rutter & Silberg, 2002).

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In recent years, a small number of criminologists have empirically examined the distinct interpretations of the association between an inadequate family environment and risky child phenotypes by modeling bidirectional parent and child effects (Beaver & Wright, 2007; Gault-Sherman, 2012; Meldrum, Young, Hay, & Flexon, 2012). These studies generally have two limitations. First, they often fail to examine child-driven effects at the earliest stages of the life course (for an exception, see Meldrum et al., 2012). Studying the first years of life may be particularly important, since research examining only the adolescent period cannot effectively rule out the possibility that the parenting patterns during adolescence are not simply a response to pre-existing child phenotypes. Second, these studies commonly utilize narrow measures of child and parent risk, often using indicators of one or two phenotypes. For example, Meldrum et al. (2012) studied the bidirectional relationship between child low self-control and maternal attachment. While these measures are useful in testing the validity of a specific theory (i.e., Gottfredson and Hirschi's parental management thesis), multidimensional measures of parental and child risk are especially advantageous, as they more fully reflect the breadth of the family environment and the overall character and behavioral profile of the child. Moreover, there is good reason to suspect that the effects of parent and child risk on each other would be especially potent as risk factors accumulate (see Beaver & Wright, 2007; Forehand, Biggar, & Kotchick, 1998; Loeber & Stouthamer-Loeber, 1986). In the current study, we seek to address the limitations of prior research using data from the Early Childhood Longitudinal Study, Kindergarten Class 1998–1999 (ECLS-K). In particular, we explore the extent to which global measures of parent and child antisocial propensity have bidirectional effects on each other across several stages of child development (i.e., early childhood through early adolescence).

Are children blank slates?

Historically, criminologists have modeled the relationship between the family environment and child delinquency as unidirectional, positing the family environment as antecedent to child behaviors. Relying on both traditional criminological theories and cultural notions concerning parenting, scholars have largely assumed that parents are the sole managers of risk in the family environment and that children are only passive recipients of this environmental risk (Beaver & Wright, 2013). In short, differences in children are thought to be the product of differences in their socialization. In recent years, however, several scholars have begun to challenge these assumptions. Pinker (2002) argues that children are born with certain innate capacities that enable them to be active participants in shaping their environment, even during the earliest stages of life. In other words, his work disputes the assumption so often made in the criminological literature that children are "blank slates" – empty vessels whose development is at the sole mercy of their environment.

Pinker's (2002) argument is buttressed by a large body of research in the field of behavioral genetics suggesting that between-individual differences in antisocial temperament and behavior can be partly attributed to genetic and biological factors (Ferguson, 2010; Rhee & Waldman, 2002; Wright et al., 2008). For example, a recent meta-analysis conducted by Ferguson (2010) found that 56% of the variation in antisocial personality and behavior can be attributed to genetic influences, with only 11% being attributable to shared non-genetic influences. The results cast doubt on the blank slate assumption, since genes a) precede exposure to the family environment and b) play a significant role in behavioral outcomes. Similarly sizeable genetic influences have been discovered for other factors associated with suboptimal parenting and delinquency, including low self-control (Beaver, Wright, DeLisi, & Vaughn, 2008) and affiliation with delinquent peers (Wright et al., 2008).

A number of brain imaging studies also seem to undermine the blank slate assumption. For instance, a recent study by Yang et al. (2012) revealed that more than 80% of the variation in the thickness of the prefrontal cortex, primarily in the bilateral dorsolateral and mesial superior frontal regions, can be explained by genetic factors. Thompson et al. (2001) garnered similar results, finding increasing similarities in the brain structure of individuals with greater genetic similarity (e.g., MZ twins), especially in Broca's and Wernicke's areas as well as the prefrontal cortex (PFC). Notably, variation in PFC functioning corresponds to differences in impulsivity, attention, and self-regulation (Damasio, 1994; Raine, 2008), all of which are predictive of crime and delinquency (Pratt & Cullen, 2000; Pratt, Cullen, Blevins, Daigle, & Unnever, 2002).

In sum, research across several disciplines suggests that an assumption of unidirectional causality from the family environment to the child is unwarranted. Such an assumption is becoming increasingly untenable in light of a growing body of research suggesting that genetic and biological factors exert a powerful influence on individual traits and behaviors. In light of such findings, the most likely scenario is a more complex one, in which the genetically influenced traits and behaviors of both the parent and the child are interacting in complex ways, contributing to a shared pathway of antisocial risk. The process by which children come to influence their parents' traits and behaviors may be best explained by the behavioral genetic concept of gene-environment correlation.

Evocative gene-environment correlation (rGE): An explanation of child effects

Gene-environment correlation refers to the ways in which genes and genetically influenced traits can structure one's exposure to certain environments. Simply put, genes, heritable phenotypes and environments are not randomly paired, but are instead patterned, so that individuals possessing high-risk genes will tend to find themselves in similarly high-risk environments (Moffitt, 2005). Genes can be correlated with environments through a number of co-occurring mechanisms. For example, genes and environments can be correlated passively by virtue of receiving genetic material and a home environment from one's parents (i.e., passive rGE). Alternatively, genes might compel an individual to actively seek out environments that reflect their genotype (i.e., active rGE). Perhaps the rGE that is most capable of explaining child-driven effects is the evocative rGE, in which an individual's heritable traits and behaviors cause others to react in ways that mirror the individual's genotype. To illustrate, children with particularly low levels of self-control, a notably heritable trait, may be more likely to frustrate and otherwise exhaust their parents, which can lead to a more hostile home environment, typified by familial conflict and harsh discipline.

The vast majority of the extant research on evocative rGEs has been conducted outside the field of criminology (for a notable exception, see Beaver, Shutt, Vaughn, DeLisi, & Wright, 2012). Many of these studies have used behavioral genetic methodologies, including twin and adoption studies, to tap genetic effects on several home environments. One such study by Ge et al. (1996) provided some early evidence of evocative rGEs at work in the home environment. The authors examined 45 youth who had been adopted shortly after birth and compared the behavior and adoptive home life of participants with an antisocial biological parent to the behavior and adoptive home life of participants without an antisocial biological parent. The results were striking. Despite not sharing an environment with their biological parent, a youth's behavior tended to a) reflect that of their biological parent and b) influence that of their adoptive parent, so that the most antisocial children tended to elicit the harshest treatment from their adoptive parent (see also Riggins-Caspers et al., 2003 for similar results).

More recent work by Boivin et al. (2005) suggests that evocative rGEs are likely at work during the earliest months of life. Using a large sample of twins and singletons, the authors found that hostile-reactive behaviors of the mother were moderately influenced by genetic factors in the child, and that this relationship was primarily mediated by the difficult temperament of the infant. In short, even young infants, who have barely been exposed to parental socialization, are capable of eliciting reactions from their parents that reflect the infant's genetic