Intergenerational transmission of antisocial behavior: How do kids become antisocial adults?

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Abstract

Exposure to many potential environmental risk factors for child antisocial behavior is associated with one of the strongest predictors of antisocial behavior, a family history of antisociality. Because most studies of putative environmental factors do not take into account genetic propensities for antisocial behavior shared between parent and child, the possibility of genetic contributions to these “environmental” markers is typically not evaluated. In this paper, we review research on the environmental correlates of antisociality, their association with parental antisociality, and highlight findings from studies that have controlled for either genetic propensities or parental antisociality.

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1. Introduction

Antisocial behaviors in adulthood are problematic at both the individual and societal levels. At the individual level, antisocial adults tend to have unstable relationships that are marked by conflict. Antisocial adults are also less likely to marry, and those who do marry experience higher rates of domestic violence, divorce, and fatherless children rearing (Cloninger, Bayon, & Przybeck, 1997). At the societal level, antisocial adults are more likely to have unstable work histories and to have multiple arrests and felony convictions (Cloninger et al., 1997). They are more likely to be involved in fights and to use weapons in these fights (Farrington, 1995). Antisociality is both highly problematic and far from rare. While estimates of the prevalence vary, it is safe to say that antisocial behavior is common.

Studies of antisociality have examined several different phenotypic expressions of problematic behaviors (e.g., conduct disorder (CD), antisocial personality disorder (ASPD), oppositional defiant disorder (ODD), delinquency, criminality, aggression, and violence), which we will refer to collectively as antisocial behavior. While each of these phenotypes of antisociality describes different specific behaviors, there is reason to believe they are nonetheless etiologically related. Krueger et al. (2002) reported that high rates of comorbidity among conduct disorder, adolescent antisocial behavior, and a disinhibited personality style are due to these behaviors loading on a shared latent externalizing dimension (see also Kendler, Prescott, Myers, & Neale, 2003; Young, Stallings, Corley, Krauter, & Hewitt, 2000). Additionally, while many behaviors can lead to criminal conviction, there is evidence that delinquency itself is a part of a unitary factor (Farrington, 1995). Thus, it appears that various antisocial characteristics often examined separately frequently co-occur and may be manifestations of a unified domain of human individual differences. Therefore, in this review we do not attach special significance to any one of these many manifestations of antisociality and will not limit ourselves to one particular operationalization of antisocial behavior. Rather, this paper will focus on a range of destructive forms of antisocial psychopathology including delinquency, criminality, violence, CD, ASPD, and aggression.

Despite the individual and societal toll exacted by antisocial behaviors, factors contributing to the development of antisocial disorders are not well understood. While a number of familial factors, such as divorce and maladaptive parenting, have been linked to child antisociality, these factors are also associated with parental antisocial behavior. As antisocial behaviors tend to run in families and to be partially heritable, it is unclear to what extent antisociality in the parents may be leading both to child antisocial behavior and to exposure to the identified risk factors. For example, it is possible that divorce may only be
associated with heightened rates of child antisocial behavior because antisocial adults are more likely to divorce, and thus may be overrepresented in divorced samples. The children of these antisocial parents are at an increased genetic risk for antisocial behavior. Higher rates of child antisociality in divorced samples could be due entirely to the genetic risk transmitted from antisocial parents to their children. Alternatively, environmental disruptions resulting from experiencing divorce could lead to higher rates of child antisociality. Most studies fail to distinguish between genetic and environmental mediation.

Many childhood correlates of adult antisocial behavior have been identified. On the within-person level, they include poor school performance and low IQ, and temperamental traits such as hyperactivity and inattention (Bassarath, 2001; Farrington, 1995), impulsivity, and risk taking behaviors (Bassarath, 2001). Familial correlates include a family history of criminality, poverty, poor parenting (Farrington, 1995), child abuse (Dodge, Bates, & Pettit, 1990), and domestic violence (Jaffee, Moffitt, Caspi, Taylor, & Arseneault, 2002). Childhood behaviors such as affiliation with antisocial peers and substance abuse (Bassarath, 2001) are also correlated with child antisociality. Many of these correlates not only run in families, but tend to co-occur with each other as well.

The study of whether these correlates of antisocial behavior, which are often referred to as risk factors, are in fact causal is made more difficult because the correlates are confounded by antisociality in the parents. According to Kraemer et al. (1997) a risk factor should be associated with the specified outcome and precede that outcome. To be considered a causal risk factor, it must be possible to change the factor and manipulation of the factor must result in a change in the specified outcome. Few risk factors for the development of antisociality are likely to pass this stringent causal test. Moreover, while many potential risk factors are present before this outcome unfolds, they are also associated with the presence of a family history of antisocial behavior. As we will show, there is considerable familial resemblance for antisocial behavior, and antisociality has a strong genetic component. While a family history of antisocial behavior is recognized as a strong marker for the development of antisociality, it is often ignored when other markers (e.g., poor parenting, divorce, child abuse) are examined. The possibility of passive gene–environment correlations accounting for the association between these markers and child antisociality is strong. Passive gene–environment correlations are operating when a parent’s genes influence the child’s environment (e.g., genetically influenced characteristics of the parent lead to engagement in maladaptive parenting of the child). According to Rutter et al. (1997), environmental correlates influenced by the parent’s genes may represent a true environmentally mediated path from parent disorder to child disorder, or the environmental correlate may not have any actual effect, and the transmission instead is genetically mediated. While the concept of gene–environment correlations is not new, many studies continue to ignore the possibility that genetic predispositions in the parents may be leading to the parent’s maladaptive behaviors and to genetic predispositions in the children, instead concluding that these maladaptive parental behaviors are causing the child’s antisociality.

1.1. Issues addressed

The primary aim of this paper is to better understand the etiology of antisocial behavior and the nature and extent of environmental risk. We review potential causes of adult antisocial behaviors, focusing in particular on the factors that appear most likely to be leading to the familial perpetuation of antisociality across generations. We begin by examining evidence of familial resemblance on a range of antisocial behaviors. Then we discuss the relative importance of genetic and shared environmental influences (that is, any aspect of the environment that makes children growing up together more similar for a trait) as well as moderators of these influences. Next we focus on research into potential risk factors in the development of antisocial behavior, highlighting studies that take a family history of antisociality into consideration and examining what is currently known about the potential causal role such correlates might play in the development of antisocial behavior. In reviewing factors influenced by the child’s family (such as divorce and poor parenting), we ask whether these environmental phenomena have any association above and beyond their co-occurrence with antisociality in parents (i.e., after controlling for parental antisociality) and whether children already at risk for antisociality (viz. having a family history of antisocial behavior) might be differentially affected by these factors. In conclusion, this paper synthesizes research on the commonly identified potential risk factors for antisociality and evaluates whether these factors remain significantly associated with child antisociality after taking into account genetic propensities shared between parent and child.

2. Familial resemblance for antisocial behaviors

Antisocial behaviors tend to run in families. There is not only resemblance between siblings growing up in the same family (Rowe, Rodgers, & Meseck-Butshey, 1992) but between parents and children as well (Herndon & Iacono, 2005). The familial aggregation of antisociality has been demonstrated through twin, family, and adoption studies.

2.1. Family studies

The familial aggregation of antisocial behavior has been demonstrated through family studies of both clinic-referred and epidemiological samples and by taking the index case as both the parent and child. Studies of clinic-referred boys have shown higher rates of antisociality in the first degree relatives of conduct disordered boys. Parents of boys with CD had higher rates of ASPD than did parents of non-conduct disordered boys, both in childhood (Frick, Lahey, Strouthamer-Lober, Christ, & Hanson, 1992; Lahey, Hardtgen, et al., 1988) and in adolescence (Miles et al., 1998). Additionally, sisters of clinic-referred conduct disordered boys exhibited higher rates of CD than did the sisters of control boys (Miles et al., 1998).

In a community sample, Foley et al. (2001) found that mother’s and father’s ASPD diagnosis was associated with a significant increase in number of CD symptoms in their offspring, and that the effect was significant for sons, but not
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