

From Correlates to Causes: Can Quasi-Experimental Studies and Statistical Innovations Bring Us Closer to Identifying the Causes of Antisocial Behavior?

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Longitudinal, epidemiological studies have identified robust risk factors for youth antisocial behavior, including harsh and coercive discipline, maltreatment, smoking during pregnancy, divorce, teen parenthood, peer deviance, parental psychopathology, and social disadvantage. Nevertheless, because this literature is largely based on observational studies, it remains unclear whether these risk factors have truly causal effects. Identifying causal risk factors for antisocial behavior would be informative for intervention efforts and for studies that test whether individuals are differentially susceptible to risk exposures. In this article, we identify the challenges to causal inference posed by observational studies and describe quasi-experimental methods and statistical innovations that may move researchers beyond discussions of risk factors to allow for stronger causal inference. We then review studies that used these methods, and we evaluate whether robust risk factors identified from observational studies are likely to play a causal role in the emergence and development of youth antisocial behavior. There is evidence of causal effects for most of the risk factors we review. However, these effects are typically smaller than those reported in observational studies, suggesting that familial confounding, social selection, and misidentification might also explain some of the association between risk exposures and antisocial behavior. For some risk factors (e.g., smoking during pregnancy, parent alcohol problems), the evidence is weak that they have environmentally mediated effects on youth antisocial behavior. We discuss the implications of these findings for intervention efforts to reduce antisocial behavior and for basic research on the etiology and course of antisocial behavior.

Keywords: antisocial behavior, causal inference, risk factors, quasi-experimental designs

The early years of the twentieth century ushered in a revolution in the academic and clinical study of the “problem child” (Silk, Nath, Siegel, & Kendall, 2000). In 1909, the first child guidance clinic was established to prevent juvenile delinquency and related emotional and behavioral problems, and by the 1930s, a network of clinics existed around the United States (Horn, 1989). The pioneering work of Sheldon and Eleanor Glueck identified developmental precursors of antisocial behavior within the child and the

child’s family (Glueck & Gleuck, 1950), most of which were also identified in longitudinal studies that started in the 1970s and 1980s. These studies followed epidemiological samples over time, assessed children’s behavior and environments according to multiple informants at multiple time points, and adjusted for an extensive array of potential confounders in analyses of risk factors for antisocial behavior. These were largely observational studies of children and their biological parents, and the goal was to identify psychosocial factors that influenced the etiology and development of children’s antisocial behavior. These studies identified a set of robust correlates of antisocial behavior in young people (Loeber & Stouthamer-Loeber, 1986) that have informed prevention and intervention efforts ever since.

Despite their methodological sophistication and public health relevance, these observational studies were limited by an inability to determine whether risk factors for antisocial behavior were actually causative; this limitation characterizes much of the research in developmental psychology and psychopathology (Foster, 2010) and hinders efforts to develop effective interventions. The goal of this review is to identify the challenges to causal inference in observational studies of antisocial behavior and to describe quasi-experimental methods and statistical innovations that allow for stronger causal inference. We then review studies that use these methods, and we evaluate whether risk factors that have been identified repeatedly across observational studies are likely to play a causal role in the emergence and development of youth antisocial

This article was published Online First October 24, 2011.

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This work is supported by Grants No. RES-062-23-1583 from the Economic and Social Research Council (ESRC) and CPF/35191 from the Nuffield Foundation to Sara R. Jaffee and RES-177-25-0013 from ESRC, HD061298 from the (U.S.) National Institute of Child Health and Human Development, and R01AG032282 from the (U.S.) National Institute on Aging to Candice L. Odgers. Luciana B. Strait is supported by ESRC Grant No. ES/i030832/1. Candice L. Odgers is a William T. Grant Scholar.

Many thanks to Barbara Maughan, Temi Moffitt, and Michael Rutter for their helpful comments on an early draft of this manuscript. We also thank Ben Lahey for his insights about the issues we raised.

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behavior. We note that there are already several excellent articles reviewing challenges to causal inference in observational research and new methods for facilitating causal inference (e.g., Foster, 2010; Rutter, 2007). Our goal is to extend these general discussions regarding causal inference to the study of antisocial behavior, specifically.

Although there have been notable efforts in recent years to identify causes of antisocial behavior (Lahey, Moffitt, & Caspi, 2003), the field is in need of an integrative summary to inform research priorities. Specifically, the scope of this article covers a range of threats to causal inference including, but not limited to, threats posed by gene–environment interplay that have also been reviewed by others (D’Onofrio & Lahey, 2010; Moffitt, 2005). Additionally, we review a range of statistical methods and research designs that deal with threats to causal inference, thus building on work reviewing specific research designs (e.g., sibling comparisons) that have been informative in the study of antisocial behavior (Lahey & D’Onofrio, 2010). Finally, this article summarizes the evidence for or against a possible causal role of a range of risk factors for the emergence and maintenance of youth antisocial behavior. In doing so, researchers can identify where the field is now ready to move from descriptive questions—“Does this risk factor play a role in youth antisocial behavior?”—to hypothesis-driven questions—“What are the mechanisms by which this risk factor causes youth antisocial behavior?” In other cases, where the evidence for a causal role is weaker, our review suggests a need for different methodological approaches or different research questions.

Methodological Challenges

As defined by Kraemer et al. (1997), a risk factor is “a measurable characteristic of each subject in a specified population that precedes the outcome of interest” (p. 338) and increases the probability that the outcome will be observed. There are four main challenges to observational studies of risk factors for children’s antisocial behavior. The first is that the association between a risk factor and antisocial behavior is confounded by a third variable or set of variables. For example, mothers who smoke during pregnancy differ in many respects from mothers who do not smoke during pregnancy in that they tend to have a history of antisocial behavior, less education, and less income, all of which could explain their children’s antisocial behavior (Wakschlag et al., 2003). Social scientists describe these sorts of confounds or “third variables” as generating spurious associations between risk exposures and outcomes.

Behavioral geneticists are specifically concerned with genetic confounds that generate passive gene–environment correlations, meaning that the same gene variants that influence how parents behave with their children may be transmitted to children and influence children’s behavior or abilities (Jaffee & Price, 2007; Plomin, DeFries, & Loehlin, 1977). For example, antisocial behavior is moderately heritable (Burt, 2009; Rhee & Waldman, 2002), and adults with a history of antisocial behavior tend to provide rearing environments that are implicated in the transmission of antisocial behavior across generations (Jaffee, Belsky, Harrington, Caspi, & Moffitt, 2006). Thus, the rearing environment could be a marker for a genetic liability for antisocial behavior that parents transmit to children rather than a cause of

children’s later behavioral problems. When genetic confounds account for the association between some exposure and children’s antisocial behavior, the association is said to be genetically mediated, rather than environmentally mediated.

The second challenge to observational studies of risk factors for antisocial behavior is reverse causation. For example, although spanking or smacking one’s child could cause the child to become aggressive and noncompliant, children who are already hard to manage could also provoke physically punitive discipline from adults. Behavioral geneticists refer to this sort of challenge as an *evocative* gene–environment correlation, meaning that characteristics of the child (which are under genetic influence) elicit a response from the environment (Jaffee & Price, 2007; Plomin et al., 1977).

A third challenge is social selection. Individuals may select—consciously or unconsciously—environments that are congruent with their abilities or behaviors. For example, youth who are prone to engage in risky behavior may be more likely to affiliate with deviant peers than youth who are not so inclined. Behavioral geneticists refer to this sort of challenge as *active* gene–environment correlation.

A fourth challenge is misidentification; meaning that the risk may stem from some correlated feature of the exposure that is not itself caused by the exposure (Rutter, 2007). For example, smoking during pregnancy is often accompanied by alcohol use and poor nutrition, either of which could be the true causal agents in any association between smoking during pregnancy and children’s antisocial behavior. Identifying when a risk factor is misidentified is complicated by the fact that some correlated factors will result from the focal risk factor (e.g., the divorce process may generate acrimony in couples) in which case they may be better conceptualized as mediators. Other risk factors may temporally precede and lead to the focal risk (e.g., high levels of conflict may lead to a divorce), in which case they may be better conceptualized as confounders.

In some cases, these alternative explanations could be resolved in an experimental design in which youth are randomly assigned to risk exposures, the exposure is tightly controlled, and youth are subsequently assessed for antisocial behavior. In the experimental design, any pre-existing differences among youth (or their families) would not be systematically related to risk exposure and therefore could not account for observed associations between exposures and outcomes. However, the vast majority of risk factors for youth antisocial behavior do not lend themselves to experimental studies. Ethical concerns preclude researchers from randomly exposing some children to domestic violence and others to more harmonious families or assigning some mothers to smoke during pregnancy and others to abstain. In addition, although well-designed experiments achieve high levels of internal validity, they often lack the external validity achieved in epidemiological research.

Until relatively recently, the best designed studies dealt with these challenges to causal inference through the use of multivariate, longitudinal designs to control for potential confounding variables and to clarify the temporal ordering of risk exposures and outcomes (Rutter, 1990). These studies included extensive statistical controls for child, parent, family, and neighborhood characteristics and usually assessed prospective associations between risk

factors and child antisocial behavior, often adjusting for antisocial behavior measured at an earlier point in time.

The major limitation to this approach is that researchers are never able to measure all the relevant confounders, a problem known variously as *omitted variable bias* or *unobserved heterogeneity*. Indeed, comparisons of effect sizes derived from randomized experiments versus studies that statistically control for an extensive array of confounding factors demonstrate the inadequacy of the “measure-the-unmeasured” approach (Duncan, Magnuson, & Ludwig, 2004). A failure to adjust for all relevant confounders—including genetic confounders—will typically inflate the association between exposure and outcome.

Later, we review the strengths and limitations of three approaches to dealing with these challenges to causal inference. We emphasize that none of these methods provides a gold standard and argue that scientific progress is made when different designs converge on similar conclusions. The first approach uses randomized control trials, in which individuals are randomly assigned to treatment and control conditions. The second uses quasi-experimental designs (also known as natural experiments). Quasi-experimental designs support causal inference because they naturally disentangle risk exposures from other correlated risk factors. The third approach uses methods that statistically match individuals who have similar background characteristics, but who differ with respect to the exposure in question. In other words, the objective is to conduct an analysis that mirrors the conditions that would exist if the risk factor (or “treatment”) had been randomly assigned. This third approach differs from the first two in using statistical approaches rather than research design elements to rule out threats to causal inference. A more detailed comparison of these approaches appears in Shadish (2010) and West and Thoemmes (2010).

Randomized Control Trials (RCTs)

In RCTs, individuals are randomly assigned to treatment and control conditions; RCTs are often considered the gold standard with respect to causal inference although, as we review, RCTs are characterized by a number of limitations. Although RCTs can achieve a high degree of internal validity, they often suffer from low external validity because they have been designed for a specific population of interest (e.g., single-parent families on welfare; Gennetian, Castells, & Morris, 2010) and because their effectiveness in nonresearch settings is demonstrably lower than their efficacy in research settings (Chambless & Hollon, 1998). Moreover, RCTs are more or less informative about etiology depending on whether they are prevention trials (e.g., designed to prevent the emergence of antisocial behavior) or intervention trials (e.g., designed to reduce existing antisocial behavior). Randomized prevention trials, combined with longitudinal measurement of risk and protective factors that can be shown to mediate prevention effects on outcome, can help to elucidate causal mechanisms (Howe, Reiss, & Yuh, 2002), albeit with the limitation that the mediators are not randomly assigned (Holland, 1986). Similarly, randomized intervention trials can demonstrate that treatment causes change in behavior (e.g., improved parenting is associated with reductions in children’s behavior problems), but they cannot demonstrate conclusively that treatment variables caused antisocial behavior to emerge in the first place (Hinshaw, 2002).

Moreover, because RCTs are typically designed to reduce or prevent antisocial behavior, the evidence that emerges can be indirect. RCTs typically aim to improve young people’s relationships with parents and peers, promote engagement with school, or modify social cognitions. Some show, for example, that increases in positive parenting and reductions in harsh parenting explain why youth in the intervention group have lower levels of antisocial behavior than youth in the control group. However, RCTs do not directly test the proposition, for example, that escalations in harsh, coercive discipline lead to increases in children’s antisocial behavior, and they do not for good reason—such a manipulation would be clearly unethical. Rather they make plausible assumptions about the effects of adverse experiences based on observations of antisocial behavior when those adverse experiences are absent and when positive experiences are present. RCTs are discussed later as they offer unique opportunities to identify a subset of casual factors (those that lend themselves to manipulation) that play a role in the progression and maintenance of antisocial behavior.

Quasi-Experimental Designs

Adoption Design

The adoption design capitalizes on the fact that adoptive parents and their adopted children are not genetically related. Thus, it automatically eliminates at least one source of confounding: that genes common to parents and children account for associations between exposures in the family and child outcomes (i.e., passive gene–environment correlation). The adoption design is characterized by several limitations (e.g., the range of environments to which adoptive children are exposed is restricted compared with that of children in the population) (Stoolmiller, 1999) and a number of assumptions about the relationship between birth and adoptive families (Leve et al., 2007; McGue et al., 2007). The design assumes that birth and adoptive parents have not been matched by social service agencies for characteristics that might influence child outcomes.

Fixed Effects Methods

Analytic methods that compare siblings within the same family or that compare the same individual across different time points are known as *fixed effects methods* (Allison, 2009). In the case of longitudinal data (i.e., within-individual analyses), fixed effects models have the benefit of controlling for all stable characteristics of the individual, as only the within-individual variation is used to estimate effects. One of the advantages of this approach is that the researchers can control for all measured and unmeasured stable characteristics by using each person as his or her own control. For example, if one wanted to know whether being part of a delinquent peer group increased antisocial behavior among middle school children (who tend to change peer groups often), one would estimate children’s level of antisocial behavior when they belonged to a delinquent peer group versus when they did not belong to a delinquent peer group. Averaging across the estimates derived for each child in the sample would provide an estimate of the effect of delinquent peer group membership on children’s antisocial behavior, controlling for all stable characteristics of the child and the child’s environment.

With respect to using fixed effects methods to compare siblings within a family, the method provides unbiased estimates of the effects of a given risk factor on antisocial behavior to the extent that unobserved effects on antisocial behavior are static over time or across family members (assumptions that may often be violated). The design also eliminates genetic confounds due to passive gene–environment correlation because the process of meiosis results in a random distribution of genes to children within a family and, hence, genotype will not be systematically associated with risk exposures (Lahey, D’Onofrio, & Waldman, 2009). Moreover, when the relevant exposure is temporally prior to the emergence of antisocial behavior (e.g., prenatal exposures), then fixed effects methods also control for reciprocal causation (Lahey et al., 2009).

There are, however, several disadvantages to fixed effects methods: they do not produce unbiased estimates when unobserved effects are time varying (or vary within-families), and they do not control for reciprocal causation when exposures and outcomes occur simultaneously (Singer & Willett, 2003). For example, assuming single motherhood is a risk factor for children’s antisocial behavior, sibling comparisons of the relationship between teen motherhood and children’s antisocial behavior may be biased if a mother was younger than 20 years and single when her first child was born, but older than 20 years and married when her second child was born. In this case, differences across siblings in their mother’s age when they were born may be misidentified as the cause of sibling differences in antisocial behavior. That is, the fact that the mother was unmarried when the elder sibling was born and married when the younger sibling was born could be the real explanation for why the elder sibling engages in higher levels of antisocial behavior than her younger sibling. Under the circumstances, it would be appropriate to control statistically for mother’s marital status in a fixed effects model. In contrast, if the mother’s marital status was unchanged across children, it could not be a source of misidentification.

Discordant Monozygotic (MZ) Twins Design

This design identifies MZ child twins who are discordant for some experience that is thought to increase risk for antisocial behavior. To the extent that within-pair differences in experience are correlated with within-pair differences in behavior, one can be confident that the effect of the experience is environmentally rather than genetically mediated (i.e., does not arise from evocative, active, or passive genotype–environment correlations). This is because MZ twins share 100% of their DNA, so differences in their experiences cannot logically arise from differences in the structure of their DNA (although they could result from epigenetic differences within a pair; Fraga et al., 2005; Mill et al., 2006; Wong et al., 2010). In addition, MZ twins are virtually always raised in the same family, so between-family differences in socioeconomic status, parental psychopathology, or neighborhood quality (i.e., many of the most robust confounding factors in research on antisocial behavior) will be held constant for twins within a family. Thus, the discordant MZ twin design is a special case of the family fixed effects method. As such, the discordant MZ twins design is characterized by the same limitations described earlier for fixed effects methods (for detailed discussion, see Vitaro, Brendgen, & Arseneault, 2009). Crucially, the design assumes that within-pair differences in exposure (e.g., one twin affiliates with

delinquent peers and the other does not) are the cause of within-pair differences in outcome, but it cannot definitively eliminate the possibility that some other event not shared by the twins accounts for observed differences between them (Jaffee, 2011).

Other Twin Designs

The Children of Twins (CoT) design includes adult twin siblings and their children. CoT studies test whether differences between twins (e.g., in terms of parenting behavior) are associated with behavioral differences among cousins. The design is premised on the observation that a mother who is an MZ twin is as closely genetically related to her nieces and nephews as she is to her own children. In DZ pairs, a mother is more closely related to her own children than to her nieces and nephews. The design deals with the first challenge to causal inference—confounding—by estimating the extent to which differences between cousins experiencing different types of parenting (or other family circumstances) are confounded by genotype or family background factors in the twin (parent) generation. The model can also include information about the twins’ spouses and other covariates that potentially differ for cousins (Jacob et al., 2003). The CoT model can be extended (ECoT) to include data from adult twins and their offspring and child twins and their parents (Narusyte et al., 2008). The ability to distinguish passive from evocative genotype–environment correlations is a unique characteristic of the ECoT model, and the model is able to detect relatively small genotype–environment correlations.

Finally, the Longitudinal Twins and Parents (LTaP) design estimates the magnitude of genetic and nongenetic pathways by which (nontwin) parental behavior is associated with (twin) offspring antisocial behavior. The design also estimates the degree of phenotypic assortment (i.e., assortative mating) between parents. The LTaP model includes information about twin behavior prior to adulthood and in adulthood. Because behavior is measured in adulthood for parents and offspring, this model upholds the assumption that the same genes influence parent and offspring phenotypes (assuming there are not sizable cohort effects). The longitudinal data can then be used to identify a model that measures the influence of parental genotype and risk exposure on twins’ behavior prior to adulthood, including the measure of passive genotype–environment correlation (Eaves, Prom, & Silberg, 2010).

These CoT, ECoT, and LTaP designs particularly deal with the problem posed to causal inference by genetic confounding. However, they are characterized by a number of assumptions and limitations. All three designs assume that mating is random. The CoT design assumes that the putative exposure does not depend on the behavior of both parents—an assumption that is probably violated in the case of dyadic behaviors like marital conflict, in which case modeling spousal characteristics potentially mitigates the problem (Eaves, Silberg, & Maes, 2005). The ECoT design is limited by the requirement that the combined samples be comparable in terms of size, participant characteristics, and measured constructs (Narusyte et al., 2008). Without longitudinal data on offspring behavior, the Twins and Parents design is likely to violate the assumption that genetic factors that influence the phenotype in the parent generation are the same as those that influence the phenotype in the offspring generation (Eaves et al., 2010).

Statistical Innovations: Methods to Statistically Match “Exposed” and “Unexposed” Individuals

Propensity Score Models

Propensity score models were designed to mirror RCTs by matching groups of individuals on a range of characteristics that predated their exposure to a given risk factor or “treatment” (D’Agostino & D’Agostino, 2007). Although individuals within these analyses have not received treatment in the traditional sense, we use the term *treatment* throughout our discussion of propensity score modeling to be consistent with Rosenbaum and Rubin’s (1983) definition of propensity scores as a function of probability of assignment to a particular treatment, conditional on observed covariates. More specifically, multivariate logistic regression models are used to estimate the propensity (or probability) of exposure or treatment on the basis of a set of measured covariates. The propensity score indexes the probability (from 0 to 1) that an individual would have been “treated” (or exposed to the risk factor) based on their background characteristics. For example, an individual with a history of peer problems, neighborhood and family poverty, and poor academic performance will have a much higher estimated propensity to join a gang (where joining a gang is considered the treatment or exposure) than an individual who has had more advantages in life, positive relations with peers, and positive experiences at school. Each individual is assigned a propensity score and matching algorithms are then used to match “treated” and “untreated” individuals with similar propensity scores (Rosenbaum & Rubin, 1985). The success of propensity score modeling hinges on whether treated and untreated matched groups are equivalent on pre-exposure characteristics, some of which may not have been observed.

As an example, Boutwell and Beaver (2010) showed that mothers in the Early Childhood Longitudinal Study birth cohort who smoked during pregnancy were more socially disadvantaged, engaged in more health-risk behaviors, and more likely to have partners who engaged in health-risk behaviors than mothers who did not smoke during pregnancy. After estimating the propensity to smoke during pregnancy (based on the measured covariates), they were able to match mothers who had similar propensities to smoke during pregnancy, but who differed as to whether they actually smoked. Matching smokers and nonsmokers on the propensity to smoke during pregnancy reduced pre-existing differences between the two groups and allowed for clearer causal inference about the relationship between smoking during pregnancy and offspring outcomes (as reviewed in the section Smoking During Pregnancy).

Group-Based Trajectory Modeling

Propensity score analysis has been combined with group-based trajectory modeling to re-create the desirable features of experimental designs in nonexperimental longitudinal data by approximating balance between treated versus untreated groups that have formed naturally over time (Haviland & Nagin, 2005; Haviland, Nagin, & Rosenbaum, 2007; Haviland, Rosenbaum, Nagin & Tremblay, 2008). The basic idea behind these efforts is to integrate a developmental view of matched treated versus untreated individuals who have followed a similar developmental pathway for the behavior under study prior to treatment. With respect to anti-

social behavior, creating matches among children who are following the same antisocial behavior trajectory may increase the quality of potential matches as the individuals share a developmental history and associated risks for the disorder. This approach also provides the secondary benefit of testing whether treatment effects differ across developmental subtypes. For example, a factor may play a causal role for one subgroup of adolescents but exert a confounding effect or a null effect within another group.

Summary of RCTs, Natural Experiments, and Statistical Matching Methods

Our discussion of methods for achieving stronger causal inference is not exhaustive. For example, we do not describe regression discontinuity, instrumental variable, migration, interrupted time series, or Mendelian randomization designs in detail because they have not typically been used to identify the causal status of risk factors for antisocial behavior. Excellent descriptions of these methods are available elsewhere (Davey Smith & Ebrahim, 2003; Gennettian, Magnuson, & Morris, 2008; Shadish, Cook, & Campbell, 2002). In the Conclusion section of this article, we encourage researchers to consider how these methods to facilitate causal inference used commonly in other disciplines could be applied to advance our understanding of causal risk factors for antisocial behavior.

It bears repeating that all research designs—quasi-experimental or otherwise—have limitations and are premised on assumptions that may or may not be justifiable. Each design carries its own threats to internal and external validity. Progress is achieved when different studies, using different designs with different limitations, converge on similar findings. It is also important to acknowledge that the answer to whether a risk factor exerts a causal effect may be conditional on characteristics of the individual or on the broader social context. As reviewed in the following section, the notion that individuals are differentially susceptible to risk exposures is premised on the assumption that the risk exposure is causal, at least for some individuals.

Differential Susceptibility to the Environment

The identification of causal risk exposures using the methods and designs described previously should inform investigations of whether individuals are differentially susceptible to risk exposures. Differential susceptibility and diathesis stress models imply that some individuals are more strongly affected than others by the presence or absence of risk exposures (Belsky & Pluess, 2009). There is mounting evidence that genetic differences among individuals moderate effects of risk exposures like maltreatment and harsh parenting on risk for antisocial behavior (Bakermans-Kranenburg & van IJzendoorn, 2011; Caspi et al., 2002; Jaffee et al., 2005; Kim-Cohen et al., 2006). However, this field of inquiry has developed with relatively little discussion of whether the environmental risks implicated in studies of Gene \times Environment ($G \times E$) interaction are actually environmentally mediated, causal risk exposures (but see Moffitt, Caspi, & Rutter, 2005, for an exception). Clearly, the plausibility of $G \times E$ models depends as much on there being a causal effect of the “E” risk as it does on the biological plausibility of the “G” risk.

Equally, the prospect of differential susceptibility has implications for methods to facilitate causal inference. The fact that a given risk factor may be a cause of antisocial behavior for some individuals but not others implies that the main effects of risk exposures may be relatively small in magnitude. Thus, the kinds of methods reviewed in the following sections—which are primarily designed to capture main effects of risk exposures—must be adequately powered to detect relatively small main effects and sufficiently flexible to consider the influence of key moderators. Otherwise, they may lead to inaccurate conclusions about the causal status of risk exposures. We encourage researchers to consider how quasi-experimental designs and statistical innovations can be applied to move beyond the traditional focus on main effects models in psychology and facilitate causal inference in models that assume differential effects across a population, developmental period, and/or setting.

Review of Risk Factors for Antisocial Behavior

The following sections synthesize findings from studies designed to facilitate causal inference for eight robust risk factors for antisocial behavior: smoking during pregnancy, harsh discipline, maltreatment, divorce, teen parenthood, parental psychopathology (including depression, antisocial behavior, and alcohol use problems), peer deviance, and social disadvantage (including poverty and neighborhood disadvantage). The order in which these risk factors are presented reflects their proximity to the child (Table 1). Although these are not the only risk factors for antisocial behavior to be identified consistently in longitudinal, epidemiological studies, others—such as parental monitoring and supervision, father absence, domestic violence, and bullying victimization—are not reviewed because we identified few, if any, studies of these that allowed for strong causal inference. Risk factors like violent television program or video game viewing (Anderson et al., 2003, 2010) were not reviewed even though they have been subject to experimental manipulation because those studies tend to measure aggression in the short term.

Special attention is paid to quasi-experimental designs that are able to separate variables that usually “go together in life” (e.g., single motherhood and teenage motherhood) and confound the interpretation of casual effects in this field and statistical methods that attempt to mimic the conditions of random assignment. For each section, we briefly review the evidence of an association from observational studies. We next identify interpretive challenges to these observed associations and then describe results of studies that allow for stronger causal inference. Although none of the research designs by themselves permit definitive causal claims, the overall pattern of findings from these studies is interpreted as lending or not lending support for a causal role of the risk factor in youth antisocial behavior.

Smoking During Pregnancy

Smoking during pregnancy has been shown to be a robust risk factor for offspring conduct problems, particularly for boys, in studies that adjust for a broad array of potential confounding variables (Wakschlag, Pickett, Cook, Benowitz, & Leventhal, 2002). Women who quit smoking during pregnancy have children with lower levels of conduct problems than women who do not quit smoking (Robinson et al., 2010), and there is evidence of a dose–response relationship between exposure to tobacco smoke in utero and offspring conduct problems (Brennan, Grekin, Mortensen, & Mednick, 2002; D’Onofrio et al., 2010). Smoking during pregnancy is predictive of offspring conduct problems even in countries such as Brazil where smoking is relatively normative and less confounded with social class than in higher income countries (Brion et al., 2010).

Despite consistency across studies, most findings are challenged by threats to causal interpretation. Mothers who smoke during pregnancy are different in many other respects from nonsmoking mothers: they tend to have less income, less education, and lower occupational status and to experience more stress in pregnancy than mothers who do not smoke (Kodl & Wakschlag, 2004; Maughan, Taylor, Caspi, & Moffitt, 2004; Pickett, Wilkinson, &

Table 1
Summary of Causal Status of Risk Factors for Youth Antisocial Behavior

Categories of risk factors	Types of methods	No. of studies	Environmentally mediated effects?
Smoking during pregnancy	Cross-fostering, FE, propensity	7	No
Harsh, coercive discipline	Adopt, CoT, discord MZ, FE, RCT, Twin	17	Yes, but also reverse causation and familial confounding
Maltreatment	TaP, Twin	3	Yes, but also familial confounding
Divorce	Adopt, CoT, FE	4	Yes, but also familial confounding
Adolescent motherhood	CoT, FE	5	Yes, but also familial confounding
Parental psychopathology			
Depression	Adopt., ECoT, RCT, timing	7	Yes, for mothers but not fathers; some familial confounding
Antisocial behavior	CoT, TaP	4	Yes, but also genetically mediated effects
Alcohol use	CoT	4	No
Peer deviance	Discord MZ, FE, propensity, RCT, twin	9	Yes, but also selection effects
Social disadvantage			
Poverty	FE, IV, RCT, UI	9	Yes, but also familial confounding
Neighborhood disadvantage	RCT	2	No

Note. FE = fixed effects; propensity = propensity score model; adopt = adoption; CoT = Children of Twins design; discord MZ = discordant monozygotic twins design; ECoT = Extended Children of Twins design; IV = instrumental variable; RCT = randomized control trial; TaP = Twins and Parents; twin = standard twin design; UI = universal intervention.

Wakschlag, 2009; Roza et al., 2009). Smokers are also more likely to have a history of antisocial behavior compared with nonsmokers (Kodl et al., 2004; Maughan et al., 2004; Roza et al., 2009). In addition, it is known that smoking during pregnancy is heritable (Agrawal et al., 2008), and genes common to the mother and child could account for the association with offspring conduct problems (D'Onofrio et al., 2008). These factors (rather than prenatal smoking per se) could be the true causal influences on children's antisocial behavior (Maughan, 2009).

Does the mother's background confound the relationship between maternal smoking and offspring antisocial behavior? Sibling comparisons, propensity score models, and studies measuring the father's smoking in pregnancy have been used to test whether the mother's background characteristics account for observed associations between her smoking in pregnancy and offspring antisocial behavior.

In four different studies of siblings (in which mother's characteristics were held constant for siblings in a pair), there was a dose-response relationship in the population between tobacco exposure in utero and offspring antisocial behavior, even after adjusting for an extensive array of covariates. However, fixed effects analyses restricted to siblings showed that siblings who differed in their exposure to tobacco smoke in utero did not differ in terms of externalizing behavior problems in middle childhood (Gilman, Gardener, & Buka, 2008; Hao & Matsueda, 2006) or adolescence (D'Onofrio et al., 2008). In addition, there were no differences in rates of violent or nonviolent offending in adulthood (D'Onofrio et al., 2010). Thus, all four studies point to familial confounding of the relationship between smoking during pregnancy and offspring antisocial behavior.

In another study, propensity score models were used to statistically equate smoking and nonsmoking mothers on a range of background characteristics (Boutwell & Beavers, 2010). Once mothers had been matched for their propensity to smoke during pregnancy, observed associations between maternal smoking and offspring antisocial behavior at age 4 years were no longer significant.

A final approach to testing whether smoking during pregnancy has effects on offspring conduct problems that are mediated via the intrauterine environment is to compare the relative strength of the effect of mothers' versus the fathers' smoking during pregnancy. If smoking during pregnancy is hypothesized to have a direct effect on the fetus, then the mother's smoking during pregnancy should be more strongly associated with offspring antisocial behavior than the father's smoking over the same period. Consistent with this hypothesis, mothers' smoking during pregnancy was significantly associated with offspring conduct problems in a sample of Brazilian 4-year-olds, but fathers' smoking during pregnancy was not (Brion et al., 2010). However, this finding was not replicated in a large sample of U.K. 4-year-olds (Brion et al., 2010) or in a Dutch sample (Roza et al., 2009), suggesting that familial confounding could have accounted for the relationship.

Does genetic risk confound the relationship between maternal smoking and offspring antisocial behavior? One approach to dealing with genetic confounds is to compare maternal-fetal pairs that are genetically related versus genetically unrelated pairs (e.g., the mother was the recipient of a donor egg, or the fetus was carried by a surrogate). One study found that children whose mothers smoked during pregnancy engaged in more antisocial

behavior in middle childhood than children whose mothers did not smoke during pregnancy, but this association was only observed among genetically related mother-offspring pairs. Thus, the findings suggest that genes common to the mother and child accounted for the relationship between smoking during pregnancy and offspring antisocial behavior, although it bears noting that the number of smokers was small, particularly in the group who were not genetically related to the fetus (Rice et al., 2009).

In summary, a range of research designs including sibling comparisons, propensity score models, designs that measure fathers' smoking, and "cross-fostering" designs converge in showing that smoking during pregnancy is not likely to be a cause of children's antisocial behavior. Rather, pre-existing differences between mothers who do versus those who do not smoke during pregnancy confound the apparent relationship. These are likely to include genetically based differences in the propensity to engage in antisocial behavior as well as other familial confounders. These studies use data from different countries, different measures of antisocial behavior (e.g., parental reports versus criminal records), and assess antisocial behavior from childhood to adulthood.

Although smoking during pregnancy may not be a causal risk factor for offspring conduct problems, it is clearly a cause of poor neonatal health (Shah & Bracken, 2000). For example, observational, quasi-experimental, and experimental human and animal studies agree that smoking during pregnancy is a cause of low birth weight (Kramer, 2003), including reduced fetal head growth (Roza et al., 2007). Thus, smoking during pregnancy may have indirect effects on antisocial behavior via effects on neurocognitive functioning. In addition, although smoking may not have main effects on youth antisocial behavior, it may have interactive effects, with some children more susceptible than others (Wakschlag et al., 2010).

Harsh, Coercive, and Inconsistent Discipline

Harsh, coercive, and inconsistent discipline comprises a constellation of parenting behaviors, including physically and verbally harsh discipline (e.g., corporal punishment, shouting, swearing, threatening) and inconsistent discipline of children's misbehavior (Reid, Patterson, & Snyder, 2002). A meta-analysis of 88 studies found that the more corporal punishment children experienced, the more aggressive they were and the more they engaged in antisocial behavior in childhood and adulthood, with effect sizes (*d*) ranging from .36 to .57 (Gershoff, 2002). This association among antisocial behavior, corporal punishment, and harsh discipline more broadly has been identified in studies that include rigorous controls for early-emerging antisocial behavior or difficult temperament as well as family characteristics including parental antisocial behavior (Lansford et al., 2002; Taylor, Manganello, Lee, & Rice, 2010).

There are two main interpretive challenges to causal inference in studies of harsh discipline and children's antisocial behavior. The first is reverse causation. Because harsh discipline is often a response to a child's perceived misbehavior (Anderson, Lytton, & Romney, 1986), children who experience harsh discipline may already be highly aggressive and oppositional for reasons that have nothing to do with how they have been disciplined. The second challenge is that familial confounding—including genetic confounding—accounts for the association between harsh discipline and children's antisocial behavior.

In the case of harsh discipline, there is evidence from RCTs that interventions designed to reduce (or prevent) children's antisocial behavior are successful because they cause decreases in harsh, inconsistent parenting and promote positive parenting. These include the Incredible Years Program (Beauchaine, Webster-Stratton, & Reid, 2005; Gardner, Hutchings, Bywater, & Whitaker, 2010), parent training programs for children with conduct or oppositional defiant problems (Fossum, Morch, Handegard, Drugli, & Larsson, 2009), and parent training programs for recently-divorced or separated mothers (Forgatch, Patterson, DeGarmo, & Beldavs, 2009; McClain et al., 2010).

Although RCTs to reduce antisocial behavior can be successful in identifying factors that account for the maintenance or stability of antisocial behavior over time—and this is a critical question in its own right—they do not necessarily identify the factors that caused the behavior to emerge in the first place. Moreover, although participants are randomly assigned to intervention and control conditions, the individual elements of the intervention (e.g., promoting positive parenting, reducing harsh discipline) are not randomized. Thus, it is not clear which specific elements of the intervention are responsible for observed changes in child behavior. Would, for example, promoting positive parenting without simultaneously reducing the use of harsh discipline result in similarly large changes in antisocial behavior? To be fair, attempts to disentangle intervention elements may be valid from an experimental design perspective, but they may not capture conditions in the real world where changes in one aspect of parenting will naturally lead to changes in other forms of parenting and will accumulate to have a positive impact on children's antisocial behavior.

Do children evoke harsh discipline? Twin and adoption designs have consistently found that genetically influenced characteristics of children evoke physically punitive responses from adults. For example, in studies of child twins, genetic factors account for between approximately one fifth and one third of the variation in physical discipline (Jaffee, Caspi, Moffitt, Polo-Tomas, et al., 2004; Wade & Kendler, 2000) and parental harsh discipline (including harsh verbal as well as physical discipline; Button, Lau, Maughan, & Eley, 2008; Pike, McGuire, Hetherington, Reiss, & Plomin, 1996). In addition, the covariation between harsh physical and verbal discipline and children's antisocial behavior is largely accounted for by a genetic factor common to both phenotypes (Button et al., 2008; Jaffee, Caspi, Moffitt, Polo-Tomas, et al., 2004; Neiderhiser, Reiss, Hetherington, & Plomin, 1999). Studies of adoptees have also found that those who are at genetic risk for antisocial behavior (based on their biological parents' history of externalizing problems) experience more harsh and coercive discipline than adoptees who are not at genetic risk (Ge et al., 1996; O'Connor, Deater-Deckard, Fulker, Rutter, & Plomin, 1998; Riggins-Caspers, Cadoret, Knutson, & Langbehn, 2003).

Does familial confounding account for the relationship between harsh discipline and children's antisocial behavior? Comparisons of siblings (nontwin and MZ twins) and children of twins have been used to test whether familial confounding—including genetic confounding—accounts for the relationship between children's antisocial behavior and harsh discipline. These studies show that harsh discipline has an environmentally mediated effect on children's antisocial behavior. For example, data

from studies of MZ twins have shown that the twin who experienced more harsh discipline also had more conduct problems (Asbury, Dunn, Pike, & Plomin, 2003; Asbury, Dunn, & Plomin, 2006; Pike et al., 1996). One study of nontwin siblings showed that children who were spanked more often than their siblings also had more externalizing problems than their siblings, even after controlling for a host of other child and family factors that varied among siblings in a family (Hao & Matsueda, 2006). However, the magnitude of the effect decreased substantially from models that controlled for unobserved family-specific heterogeneity to models that estimated stable effects of children's externalizing behavior on spanking, suggesting that the association between spanking and children's externalizing problems reflected both parent- and child-driven effects.

Using the CoT design, Lynch, Turkheimer, D'Onofrio, Mendle, and Emery (2006) also showed that, within a nuclear family, children who experienced more harsh physical discipline than their siblings had more externalizing problems than their siblings. In addition, children who experienced more harsh physical discipline than their cousins had more externalizing problems than their cousins and the magnitude of this effect was no larger in DZ twin families (where genetic confounds are uncontrolled) than in MZ twin families (where genetic confounds are automatically controlled). Thus, findings were consistent with a causal effect of harsh physical discipline on antisocial behavior.

Finally, we note that there are many genetically-informative studies of parent-child relations, comprising a heterogeneous group of constructs that includes parents' negative feelings about children (Caspi et al., 2004; Larsson, Viding, Rijdsdijk, & Plomin, 2008), parental criticism and preferential treatment of one sibling versus another (Carbonneau, Eaves, Silberg, Simonoff, & Rutter, 2002), parent-child conflict (Burt, McGue, Krueger, & Iacono, 2005; Burt, McGue, Iacono, & Krueger, 2006; Klahr, McGue, Iacono, & Burt, 2011; Klahr, Rueter, McGue, Iacono, & Burt, 2011), parent-child dyadic mutuality (Deater-Deckard & Petrill, 2004), negative means of controlling child behavior other than physical discipline (O'Connor et al., 1998), and composites of various parenting practices (Ge et al., 1996; Neiderhiser et al., 1999; Pike et al., 1996). Because of our desire to focus on a relatively homogeneous construct (harsh, coercive discipline) that has been consistently identified as a robust correlate of child antisocial behavior, we did not include these other studies of parenting in our review.

In summary, data from a variety of research designs are consistent with the hypothesis that there are reciprocal effects of harsh discipline and children's antisocial behavior. That is, although child effects are clearly present, harsh discipline nevertheless leads to increases in children's antisocial behavior. Thus, successfully reducing harsh disciplinary practices should prevent the emergence of or result in reductions in children's antisocial behavior. Intervening as early as possible to teach parents how to handle children's oppositional behavior should reduce the likelihood of coercive cycles developing.

Maltreatment

Results from prospective, longitudinal studies—including those involving demographically-matched controls—have shown that victims of child maltreatment have elevated rates of antisocial

behavior in childhood and adolescence compared with nonmaltreated youth (Dodge, Bates, & Pettit, 1990; Jaffee, Caspi, Moffitt, & Taylor, 2004; Smith & Thornberry, 1995; Widom, 1989). A recent meta-analysis identified a moderate to large effect size of .61 between experiencing violence (largely in the form of maltreatment) and youth antisocial behavior (Wilson, Stover, & Berkowitz, 2009).

Although maltreatment could be a causal risk factor for antisocial behavior, two alternative explanations are possible. As with harsh discipline, the relationship may reflect familial confounding, including genetic confounding (DiLalla & Gottesman, 1991). Parents who have a history of antisocial behavior are at elevated risk of maltreating their children (Brown, Cohen, Johnson, & Salzinger, 1998; Dinwiddie & Bucholz, 1993; Walsh, McMillan, & Jamieson, 2002), and antisocial behavior is moderately heritable (Burt, 2009; Rhee & Waldman, 2002). Alternatively, physical abuse in particular may be provoked by oppositional, aggressive behavior on the part of the child.

Do children evoke maltreatment? Two studies have used the twin design to test whether evocative gene–environment correlations account for the relationship between maltreatment and children’s antisocial behavior. Although one study relied on maternal reports of young children’s experiences of maltreatment (Jaffee, Caspi, Moffitt, & Taylor, 2004) and the other used data from young adult twins and nontwin siblings who provided retrospective reports of childhood maltreatment (Schulz-Heik et al., 2009), both showed that genetic factors accounted for only 6%–7% of the variation in children’s experiences of maltreatment. Thus, genetically influenced characteristics of children accounted for a small and statistically nonsignificant portion of the variance in maltreatment.

Does familial confounding account for the relationship between maltreatment and children’s antisocial behavior? Using the LTaP design, Eaves et al. (2010) found that nongenetic effects of parent antisocial behavior on offspring antisocial behavior were largely mediated by adverse parenting (i.e., parental neglect, exposure to interparental violence, and parents’ inconsistent discipline of children) that directly accounted for approximately 30% of shared environmental effects on offspring antisocial behavior. Moreover, effects of the shared environment (including adverse parenting) on offspring antisocial behavior were evident when youth were adolescents and adults. Very small passive gene–environment correlations were detected.

In summary, the two studies that estimated genetic influences on child maltreatment (Jaffee, Caspi, Moffitt, Polo-Tomas, et al., 2004; Schulz-Heik et al., 2009) showed that additive genetic influences on child maltreatment were small, accounting for only 6%–7% of the variance. Alone, this finding suggests that genetically influenced characteristics of children (including their antisocial behavior) do not provoke adults to abuse them, although it bears noting that assortative mating for psychiatric disorder (and, particularly, for antisocial behavior) could have artifactually deflated observed heritability estimates. In addition, Eaves et al. (2010) showed that the relationship between maltreatment-related childhood adversity and offspring antisocial behavior was not accounted for merely by gene variants common to parents and children; rather, childhood adversity was shown to have direct, environmentally mediated effects on youth antisocial behavior, and passive gene–environment correlations were shown to be

small. Together, these findings provide stronger support for the hypothesis that maltreatment is a cause of youth antisocial behavior than for alternative hypotheses involving passive or evocative gene–environment correlation.

A limitation of these studies is that none involved documented cases of maltreatment. Jaffee, Caspi, Moffitt, and Taylor (2004) relied on maternal reports of maltreatment, and these may have been biased by underreporting. The others (Eaves et al., 2010; Schulz-Heik et al., 2009) relied on retrospective recall of parenting behaviors, only some of which could actually be described as abusive. This limitation may be pervasive in genetically informative studies. For example, although studies of adoptees neatly eliminate the biological relationship between parents and offspring (and, hence, passive genotype–environment correlation), adoptive families are screened to have low rates of maltreatment. Although studies of twins (and children of twins) tend not to have information about substantiated maltreatment, it is possible (but difficult) to obtain that information (as demonstrated by Jonson-Reid et al., 2010). One way forward may be to combine child protective service records with parental reports of children’s experience of abuse or neglect. Other possibilities for quasi-experimental and experimental designs include propensity score analyses (using samples that include nonmaltreated children and maltreated children that overlap in their likelihood of experiencing maltreatment) or experimental prevention studies to prevent maltreatment.

Divorce

There is a large body of literature showing that the children of divorced parents have elevated rates of antisocial behavior (Emery, 1999), although effect sizes are modest in magnitude (Amato, 2001). As with other risk factors for antisocial behavior, these associations are potentially confounded by co-occurring genetic and familial risk factors.

Although 43% of first marriages end in divorce (Bramlett & Mosher, 2002), families in which couples subsequently divorce differ from intact families in at least three ways. First, they are disadvantaged in terms of education, occupational status, and income (Kiernan & Mueller, 1998; Teachman, 2002). Second, they are more likely to have histories of antisocial behavior (Emery, Waldron, Kitzmann, & Aaron, 1999; Fomby & Cherlin, 2007; Lahey et al., 1988). Third, families in which parents subsequently divorce are more dysfunctional than families in which parents do not divorce, with elevated rates of child problem behaviors, parent–child conflict, and marital conflict (Block, Block, & Gjerde, 1986; Cherlin et al., 1991; Doherty & Needle, 1991; Elliott & Richards, 1991; Peris & Emery, 2004; Shaw, Winslow, & Flanagan, 1999; Sun, 2001), although not all studies have identified these predivorce differences (Forehand, Armistead, & David, 1997; Shaw, Emery, & Tuer, 1993). In some cases, such predivorce differences in child and family functioning have been shown to account for differences in children’s antisocial behavior following a divorce (Cherlin et al., 1991; Peris & Emery, 2004), but this has not always been found (Morrison & Cherlin, 1995; Shaw et al., 1999).

It is also possible that features of the family environment correlated with divorce rather than divorce per se are the true causal risk factors for youth antisocial behavior. In practice, identifying

these features and determining whether they give rise to, stem from, or simply co-occur with divorce is difficult.

Three different designs have been used to test whether familial confounding accounts for the association between divorce and youth antisocial behavior. First, an adoption study found that divorce was associated with increased antisocial behavior regardless of whether parents and children were biologically related. Moreover, children had to have been exposed to the divorce in order for it to have the predicted effect (Burt, Barnes, McGue, & Iacono, 2008). Another study identified the expected differences in antisocial behavior in children from divorced versus intact families, but only if parents and children were biologically related (O'Connor, Caspi, DeFries, & Plomin, 2000). However, this study included relatively few children who were adopted and whose adoptive parents subsequently divorced.

Second, data from a high-risk sample of Australian twins and their young adult offspring showed that youth whose parents had divorced—particularly before the youth had reached the age of 16—had more behavior problems than their cousins whose parents had not divorced. The magnitude of this difference did not depend on whether the youth's parents and their aunts and uncles were MZ or DZ twins, providing evidence that neither genetic nor parental family background factors confounded the association between divorce and offspring behavior problems (D'Onofrio et al., 2005).

Third, within-individual fixed effects methods have been used to estimate trajectories of externalizing problems extending from 1 year prior to a divorce to 3 years following the divorce (Lansford et al., 2006). This study showed that differences between children who experienced divorce (particularly prior to adolescence) and those who did not were greatest in the year following divorce and remained significant 3 years postdivorce. Effects of divorce in adolescence on antisocial behavior were negligible, however. In summary, a range of research designs converge in showing that divorce is likely to play a causal role in increasing risk for youth antisocial behavior. Although these designs help to rule out genetic and nongenetic familial confounding, different designs are required to determine whether the effects of divorce are misidentified—that is, whether experiences that co-occur with divorce, but are not caused by it, are the true causal influences on youth antisocial behavior and whether the effects of divorce are felt more strongly during childhood.

Adolescent Motherhood

Children born to adolescent mothers are at risk for a range of adverse outcomes, including antisocial behavior (Coley & Chase-Lansdale, 1998; Jaffee, Caspi, Moffitt, Belsky, & Silva, 2001). However, pre-existing differences in the abilities, behaviors, and family backgrounds of younger versus older first-time mothers could account for differences in their children's behavior (Jaffee, 2002).

Findings from quasi-experimental studies are mixed as to whether adolescent motherhood is a causal risk factor for youth antisocial behavior. Two studies have used data from the Children of the National Longitudinal Survey of Youth (CNLSY) to compare cousins, some of whom were discordant for having been born to an adolescent mother (Geronimus, Korenman, & Hillemeier, 1994; Turley, 2003). Neither study found that youth born to

adolescent mothers had more behavior problems—a composite of internalizing and externalizing problems—than their cousins who were born to older mothers. Thus, these studies suggest that (extended) families in which some women become adolescent mothers differ from families in which women delay childbearing beyond adolescence, and these differences account for children's outcomes. A limitation of these studies is that they were conducted at a point when many women in the NLSY had not yet become mothers, and estimates may have been biased by restricted variability in maternal age (D'Onofrio et al., 2009b).

Because cousin comparisons provide incomplete control for family background factors, other researchers have used data from the CNLSY to compare siblings from the same family, some of whom may have been born when their mother was a teenager and others of whom may have been born when their mother was older. Findings from these studies are also mixed. One group found that siblings had equally high levels of externalizing problems even if their mother was no longer a teenager by the time the younger siblings were born (Hao & Matsueda, 2006). However, another study using a larger sample of CNLSY participants found that older siblings had more antisocial behavior (as reported by mothers and youth themselves) than younger siblings (D'Onofrio et al., 2009b). Differences between these comparisons of NLSY siblings (D'Onofrio et al., 2009b; Hao & Matsueda, 2006) could have arisen from measuring mother's age on a continuum rather than measuring whether the mother was an adolescent at the birth of each child. The studies also comprised different waves of data, with Hao & Matsueda (2006) including data up to 1996 and D'Onofrio et al. (2009b) including data up to 2004, with the result being that the latter sample was larger and included a wider range of maternal ages.

Finally, data from the Australian Children of Twins sample (Harden, Lynch, et al., 2007) showed that children who were born when their mother was an adolescent had significantly more antisocial behavior than their siblings who were born when their mother was no longer an adolescent. They also found that youth who came from extended families in which one or both twins made a transition to parenthood in adolescence had more antisocial behavior than youth from extended families in which neither twin had become an adolescent parent. In general, these results are consistent with the possibility that adolescent motherhood is causally related to youth antisocial behavior.

In summary, although the evidence is mixed, the best controlled quasi-experimental studies using the most representative samples provide evidence of a causal effect of adolescent motherhood on offspring antisocial behavior. Although this causal effect can be identified, all the studies reviewed showed that failing to account for genetic and nongenetic confounders resulted in overstatement of the effects of adolescent motherhood on youth antisocial behavior.

Parental Psychopathology

Decades of high-risk designs have observed that children whose parents have histories of depression, antisocial behavior, and substance use problems are at elevated risk for antisocial behavior. As noted earlier, the fact that psychopathology runs in families could reflect the transmission of a genetic liability for antisocial behavior; equally, it could reflect a socially transmitted process.

Maternal depression. Children whose mothers are currently depressed or have a lifetime history of depression are at elevated risk for antisocial behavior (Goodman, 1997). Although there is evidence that the relation between maternal depression and child problem behaviors is bidirectional (Feske et al., 2001; Ge, Conger, Lorenz, Shanahan, & Elder, 1995; Hammen, Burge, & Adrian, 1991; Jaffee & Poulton, 2006; Kim, Conger, Elder, & Lorenz, 2003), studies that model this bidirectionality consistently identify effects of the mother's depression on her child's behavior. Nevertheless, very few studies of maternal depression have been designed to disentangle whether maternal depression is an environmentally mediated risk factor i.e., the rearing environment provided by depressed mothers is criminogenic or, given that depression is moderately heritable (Sullivan, Neale, & Kendler, 2000), whether depressed mothers transmit a genetic liability for problem behaviors to their children.

Evidence from four different research designs is consistent with the possibility that maternal depression has environmentally mediated effects on offspring antisocial behavior. First, maternal depression is associated with an increased risk for offspring antisocial behavior if the mother was depressed after her child was born, but not if she was only ever depressed before her child was born (Kim-Cohen, Moffitt, Taylor, Pawlby, & Caspi, 2005). Thus, exposure to a mother's depression was necessary for it to increase risk for youth antisocial behavior. Second, one study of adoptees showed that adolescents' risk of disruptive behavior disorders was elevated if their mothers had a lifetime history of major depressive disorder, regardless of whether youth were adopted or nonadopted (Tully, Iacono, & McGue, 2008), and another found that more depressive symptomatology among adoptive mothers was associated with more externalizing problems in their toddlers, even controlling for the birth mother's depressive symptomatology (Pemberton et al., 2010). Thus, youth need not have been genetically related to their mother in order for her depression to be associated with their behavior. Third, successful treatment of a mother's depression is associated with declines in her children's symptoms of antisocial behavior (Weissman et al., 2006). Fourth, one study using the ECoT model showed that although passive gene-environment correlation accounted for a small portion of the variation in adolescent conduct problems, there were also small, statistically significant effects of parental depression that were environmentally mediated (Silberg, Maes, & Eaves, 2010).

There is weak evidence that fathers' depression has environmentally-mediated effects on youth antisocial behavior. One study found that adoptive fathers' symptoms of depression when their infants were age 9 months (but not at later points) were predictive of elevated externalizing problems in their children at age 27 months, controlling for adoptive mothers' and birth mothers' depression (Pemberton et al., 2010). However, Ramchandani et al. (2008) failed to find that fathers' depression was more strongly associated with youth antisocial behavior when fathers were depressed after rather than before their children were born, particularly after controlling for family demographic variables.

In summary, five studies using different research designs converge in showing that although children clearly inherit a genetic liability to depression, maternal depression is also likely to be an environmentally-mediated causal risk factor for children's conduct problems. Findings from the small pool of studies on fathers' depression offer weaker support for causal claims. These findings

reinforce efforts to identify how a mother's depression impinges on her relationship with her child or her relationship with other family members, thereby increasing the child's risk for antisocial behavior. In addition, exposure to a mother's depression may occur in the prenatal as well as the postnatal period (Hay, Pawlby, Waters, Perra, & Sharp, 2010). More research is needed to identify how the intrauterine environment changes as a function of a mother's depression and how these changes could produce a liability to antisocial behavior later in the child's life.

Parental antisocial behavior and substance use. Antisocial behavior tends to run in families (Farrington, Jolliffe, Loeber, Stouthamer-Loeber, & Kalb, 2001; Smith & Farrington, 2004; Thornberry, 2009). However, most research designs have been unable to determine whether the transmission of antisocial behavior from parents to children is genetically or environmentally mediated (Blazei, Iacono, & Krueger, 2006; Moffitt, 2005).

There is little question that individual differences in children's antisocial behavior are partly accounted for by genetic factors. Twin and adoption studies show that genetic factors account for approximately 43%–46% of the variation in antisocial behavior in childhood and adolescence (Burt, 2009; Rhee & Waldman, 2002). More recently, however, quasi-experimental designs have been used to estimate the nongenetic transmission of antisocial behavior from parents to children.

Findings have been mixed. On the one hand, findings from the Australian CoT sample support an environmentally mediated effect; the association between parents' and sons' conduct problems was significant in the sample overall and the within-twin family effect was equally strong in MZ- versus DZ-twin-families (i.e., the extent to which within-pair differences in antisocial behavior in the parent generation were associated with differences in antisocial behavior among cousins; D'Onofrio et al., 2007). Similarly, studies using two different samples found that although there was a clear association between fathers' antisocial behavior and children's antisocial behavior in the sample overall, the offspring of antisocial men who lived with their children had more antisocial behavior problems than the offspring of antisocial men who did not live with their children (Blazei, Iacono, & McGue, 2008; Jaffee, Moffitt, Caspi, & Taylor, 2003), again suggesting that the intergenerational transmission of risk for antisocial behavior was environmentally as well as genetically mediated.

In contrast, other studies have not found support for the hypothesis that the intergenerational transmission of risk for antisocial behavior is environmentally mediated. One study using the Twins and Parents design found that although shared environmental factors accounted for approximately one fifth of the variation in adolescent conduct problems, parental factors represented a small and nonsignificant portion of this effect. The nongenetic pathway by which parent antisocial behavior was associated with offspring conduct problems was small and statistically nonsignificant, although power to detect this pathway was relatively low (Maes, Silberg, Neale, & Eaves, 2007). Similarly, data from the Australian CoT sample (D'Onofrio et al., 2007) showed that—in contrast to boys—girls had as many conduct problems as their cousins, regardless of whether the twin parents were concordant or discordant for conduct problems.

In summary, the bulk of evidence suggests that the transmission of antisocial behavior from parents to children appears to be both genetically and environmentally mediated, with one study suggest-

ing different pathways for sons versus daughters (D'Onofrio et al., 2007). More research is needed to understand the role of mothers' antisocial behavior and to test whether there are truly sex differences in the transmission of antisocial behavior across generations.

Parental substance use. Several longitudinal studies have established that the children of alcoholics are at elevated risk of antisocial behavior (Chassin, Rogosch, & Barrera, 1991; Hill & Muka, 1996; Sher, Walitzer, Wood, & Brent, 1991). There is some evidence that parental alcoholism is a unique predictor of childhood externalizing problems after controlling for parental depression and antisocial behavior (Loukas, Zucker, Fitzgerald, & Krull, 2003). However, the high heritability of behaviors across the externalizing spectrum (Hicks, Krueger, Iacono, McGue, & Patrick, 2004) raises questions as to whether the relationship between parental substance use and child antisocial behavior is driven by a shared genetic risk for externalizing problems versus exposure to parental alcoholism per se. Although small in number, the majority of studies that have employed statistical controls have shown that a history of alcohol dependence in the absence of other co-occurring disorders or environmental stressors is not sufficient to confer risk to offspring (see, for example, Chassin et al., 1991; Ohannessian et al., 2005; Schuckit, Smith, Radzimirski, & Heyneman, 2000). Given the limited body of research on other types of substances, our review focuses primarily on parental alcohol problems.

A handful of studies have leveraged genetically informative research designs to test how parental alcoholism may influence problem behaviors among offspring (Haber et al., 2010; Haber, Jacob, & Heath, 2005; Waldron, Martin, & Heath, 2009). Using data from the Vietnam Era Twin Registry, Haber and colleagues (Haber et al., 2005, 2010) showed that the offspring of nonalcoholic fathers had as many symptoms of conduct problems as their cousins whose fathers had a lifetime history of alcohol abuse or dependence. Thus, genes common to alcoholism and conduct problems are likely to explain the intergenerational relation between the two. In contrast to the findings for parental alcoholism, paternal drug dependence was shown to have an environmentally mediated effect on offspring antisocial behavior (Haber et al., 2010).

Waldron and colleagues (2009) also found little evidence of environmental transmission of risk for antisocial behavior from parental alcoholism in two ongoing CoT studies in Australia. Consistent with a genetically mediated pathway, they reported that offspring at high genetic risk (i.e., children born to MZ twins, regardless of whether their own father or their uncle had alcohol use disorder) exhibited more problem behaviors than those at intermediate genetic risk (i.e., children born to DZ twins whose uncles had an alcohol use disorder) or at low genetic risk (i.e., children born to DZ twins where neither the uncle nor the father had an alcohol use disorder).

If parent substance use problems have environmentally mediated effects on youth antisocial behavior, then changes over time in parents' substance use symptomatology should be associated with increases or declines in children's antisocial behavior over the same period. Limited support for this hypothesis was found in one study showing that children's symptoms of antisocial behavior were elevated during developmental periods when parents' symptoms of substance use problems were similarly elevated (Hussong, Huang, Curran, Chassin, & Zucker, 2010). However, this within-

individual effect was observed only when youth antisocial behavior was assessed by fathers' report (marginally significant) and in the matched control sample when youth reported on their own antisocial behavior.

In sum, evidence to date has primarily supported a strong genetic transmission of the effects of parental alcoholism to offspring conduct problems for both males and females. Future research is required to address whether environmentally mediated effects are more likely to be present at different stages of development or for different types of children. Moreover, there is a need to address whether the transmission of risk for parental substance use may differ for dependence on alcohol versus illicit substances.

Peer Deviance

Peer deviance can take the form of antisocial behavior enacted within the context of dyadic friendships, informal peer networks, and organized gangs (Thornberry & Krohn, 1997; Warr, 1996). Youth who associate with deviant peers are more likely than youth who do not to engage in violent and nonviolent antisocial behavior and other risky behaviors (Elliot & Huizinga, 1985; Thornberry & Krohn, 1997).

The association between peer deviance and youth antisocial behavior may reflect a causal process wherein deviant peers model and reinforce antisocial behavior (Burgess and Akers, 1966; Deater-Deckard, 2001; Dishion et al., 1999; Sutherland, 1936) or a youth-driven process wherein those who are prone to antisocial behavior selectively affiliate with deviant peers (Glueck and Glueck, 1950; Hirschi, 1969; Kendler, Jacobson, Myers, & Eaves, 2008). Indeed, these processes are likely to interact reciprocally over time (Elliot & Huizinga, 1985; Elliot & Menard, 1996; Thornberry, 1987).

Do deviant peers model and reinforce antisocial behavior? Some evidence for causal effects of peer deviance on youth antisocial behavior comes—paradoxically—from randomized interventions designed to reduce antisocial behavior. In one RCT in which the groups were parent treatment, adolescent group treatment (with or without parent treatment), and controls, youth who participated in the adolescent treatment groups had significantly more teacher-reported antisocial behavior problems compared with control youth at 1-year follow-up (Dishion & Andrews, 1995), although by 2- and 3-year follow-ups, iatrogenic effects on teacher-reported antisocial behavior were only marginally significant (Poulin, Dishion, & Burraston, 2001). Subsequent work by Dishion and colleagues has suggested that deviant peer groups provide opportunities for youth to model and positively reinforce one another's antisocial behavior and that deviant talk leads to increases in antisocial behavior (Dishion, Spracklen, Andrews, & Patterson, 1996; Patterson, Dishion, & Yoerger, 2000). There is, however, disagreement in the field about the strength and consistency of iatrogenic effects of group treatment for antisocial behavior, with a meta-analysis showing that peer group interventions have beneficial effects that do not differ depending on whether groups are homogeneous versus heterogeneous for antisocial behavior (Weiss et al., 2005).

Do youth selectively affiliate with deviant peers? Genetically-informative designs provide evidence that youth who engage in antisocial behavior selectively affiliate with deviant peers. For example, using data from a sample of MZ twins who

reported sharing few of their friends, Burt, McGue, and Iacono (2009) found that, within-pairs, the twin who had higher levels of antisocial behavior in early adolescence also had more deviant peers later in adolescence. Similarly, in a sample of male adolescent twin pairs, Kendler et al. (2008) found that youth antisocial behavior was predictive of peer deviance through processes of social selection. However, unlike Burt et al. (2009), they also found that peer deviance was associated with antisocial behavior through processes of social causation (Kendler et al., 2008).

Because joining a gang is a discrete event that can be located at a particular point in time, a number of studies have used longitudinal data and fixed effects methods (Gordon et al., 2004) to test whether youth who join gangs were already engaging in higher levels of antisocial behavior than nonjoiners, whether gang membership is associated with subsequent increases in antisocial behavior, and whether exiting a gang is associated with declines in antisocial behavior. These studies are generally consistent in showing evidence of selection effects: youth who go on to join gangs tend to engage in higher levels of antisocial behavior than youth who do not join gangs (Esbensen & Huizinga, 1993; Gordon et al., 2004; Thornberry, Krohn, Lizotte, Smith, & Tobin, 2003). However, these same studies also show that antisocial behavior increases after youth join gangs (Esbensen et al., 1993; Gordon et al., 2004) and that it falls to pre-gang levels when youth have exited gangs (Gordon et al., 2004).

Haviland, Nagin, and Rosenbaum (2007) combined group-based trajectory methods with propensity score matching to estimate the causal impact of gang membership on youth antisocial behavior. This analysis identified trajectories of boys' antisocial behavior from ages 11 to 13 and, within trajectories, matched boys on their propensity to join a gang. Boys who joined gangs at age 14 engaged in significantly higher levels of violence over the next 2 years compared with boys who did not join gangs, although group differences dissipated by age 16 due to movement into and out of gangs.

In summary, there is substantial evidence from longitudinal, epidemiological, and genetically informative studies that both social selection and social causation are operative in the relationship between peer deviance and antisocial behavior. Although youth who engage in antisocial behavior selectively affiliate with delinquent peers, associating with deviant peers provides new opportunities to engage in delinquency.

Social Disadvantage

Several risk factors associated with social disadvantage have been shown to predict youth antisocial behavior independent of measured individual and family factors, including neighborhood poverty, low levels of neighborhood collective efficacy (Odgers et al., 2009; Sampson, Raudenbush, & Earls, 1997), and family poverty (Duncan & Brooks-Gunn, 1997). Nevertheless, one challenge to causal inference is the possibility of unobserved heterogeneity: poor families may differ from nonpoor families in ways that may explain not only their circumstances but also their children's antisocial behavior. The social selection versus social causation debate has a long and rich history within the neighborhood effects literature. Experimental and quasi-experimental methods are required to identify cause-effect relations and these designs

have been embraced by investigators working on this issue from across a number of disciplines.

Poverty. At least two studies have used comparisons of siblings and cousins from the CNLSY to test for income effects on youth externalizing problems (D'Onofrio et al., 2009a; Hao & Matsueda, 2006). Using fixed effects methods, Hao and Matsueda (2006) showed that sibling differences in the experience of poverty in early childhood were associated with sibling differences in antisocial behavior, controlling for a range of child and family characteristics that also varied among siblings. D'Onofrio et al. (2009a) estimated hierarchical linear models using data from siblings and cousins and showed that within-family (extended and nuclear family) differences in income were associated with intra-family differences in conduct problems, particularly for boys.

Another study took advantage of a natural experiment in which Native American families participating in a longitudinal study of youth development saw radical increases in income due to the opening of a casino on their reservation approximately 4 years after the study started (Costello, Compton, Keeler, & Angold, 2003). Because all Native American families benefited from casino profits, this study eliminated the possibility that income gains were associated with individual differences among families that could potentially confound observed associations between income and children's antisocial behavior. Among the Native American children whose families transitioned out of poverty, levels of antisocial behavior dropped significantly from the 4 years prior to the 4 years following the casino opening. Moreover, levels of antisocial behavior were as low in this group as in the group of children whose families were never poor. In contrast, levels of antisocial behavior did not change significantly among the group whose families were never poor or among the group whose families remained poor even after the casino opened.

A follow-up of this sample has shown that the benefits of the income supplement extended into young adulthood. The Native Americans who benefited from the casino profits had lower arrest rates for minor crimes (but not more serious crimes) by age 21 than the non-Native American population (Akee, Copeland, Keeler, Angold, & Costello, 2010) and lower rates of substance use disorders (Costello, Erkanli, Copeland, & Angold, 2010), which are closely linked with earlier antisocial behavior.

Two studies have used within-family changes in income over time to estimate effects of income on youth antisocial behavior. Strohschein (2005) used seven waves of data from the CNLSY extending from early childhood into early adolescence and showed that children had significantly fewer antisocial behavior problems during periods when their families had relatively higher versus lower income. A similar pattern of results was obtained in a study that used data from the National Institute of Child Health and Human Development Study of Early Child Care and Youth Development and estimated effects of income change within families from the time children were 2 years to approximately 6 years old (Dearing, McCartney, & Taylor, 2006), although they found the effects of income change were strongest for the chronically poor group (i.e., children whose family income fell below the federal poverty threshold at three out of five assessments).

Experimental studies of antipoverty policies have demonstrated some effects of income on children's externalizing behavior. For example, in the Minnesota Family Investment Partnership, families were randomized to a control condition (in which they re-

ceived standard welfare benefits) or one of two treatment conditions in which they either received enhanced earnings disregards (i.e., earned income was partially discounted when a family's welfare benefits were calculated) or enhanced earnings disregards plus a work mandate (i.e., individuals were required to participate in employment or employment-related activities to be eligible for welfare assistance). Families in both treatment conditions had significantly higher incomes than families in the control condition and children in both treatment conditions had significantly lower levels of antisocial behavior than control children (Gennetian & Miller, 2002).

In a related vein, other researchers have used naturally occurring variation in child benefit policy (i.e., cash transfers to families with children) to estimate income effects on children's antisocial behavior. Using data from the Canadian National Longitudinal Study of Children and Youth, Milligan and Stabile (2008) showed that children whose families were receiving relatively more benefits (because of differences across provinces and over time in benefits policy) had significantly lower levels of antisocial behavior; this effect was especially pronounced for girls.

In summary, data from a variety of quasi-experimental designs converge in identifying a causal role for family income in children's antisocial behavior. This is notable seeing as how many of the studies of the CNLSY involved a restricted range of income due to the relatively young age of mothers whose children were old enough to be assessed for conduct problems. These studies differ in terms of whether income effects are stronger for boys versus girls, whether income in early childhood has stronger effects than income at later stages of development, and whether income effects are stronger at the lower versus higher end of the income distribution.

Neighborhood disadvantage. Two quasi-experimental studies have been designed to test whether neighborhood conditions have causal effects on individual well-being (i.e., social causation) or whether neighborhood conditions arise from the characteristics of individuals who opt to reside in them or reside in them due to downward social mobility (social selection). In *Moving to Opportunities (MTO)*, families who were living in individual and neighborhood poverty were randomly assigned to receive housing vouchers to move to a more affluent neighborhood (experimental group), to move to any neighborhood, or to receive no vouchers. Thus, random assignment to experimental and control conditions was applied to eliminate the possibility that social selection could account for neighborhood effects on youth outcomes.

It was found that 4–7 years postrandomization, girls in the experimental group had significantly lower arrest rates for violent and property crime, but no differences among the groups were found in terms of parent-reported antisocial behavior. Although boys in the experimental group initially had lower levels of violent crime compared with control boys, arrests for property crimes and self-reported behavior problems were higher among boys in the experimental group versus those in the control group by the end of the follow-up period (Kling, Ludwig, & Katz, 2005). Thus, moving out of neighborhood poverty was beneficial for girls, but not for boys, with one explanation being that boys were more likely than girls to capitalize on newfound opportunities to commit property crimes because they were less attached to school and more likely to affiliate with a deviant peer group (Kling et al., 2005).

Another randomized intervention followed two groups in the wake of a 1985 court-ordered desegregation program in Yonkers, New York. A 7-year follow-up of the sample found no differences in parent- or youth-reported antisocial behavior between the adolescent offspring of families who were randomly selected to relocate to publicly funded housing in middle-class neighborhoods versus demographically similar youth whose families were not selected to move (Fauth, Leventhal, & Brooks-Gunn, 2007).

In combination, the results of these quasi-experimental studies suggest that, at best, relocating from poor to more affluent neighborhoods has small effects on reducing girls' antisocial behavior and, at worst, it facilitates antisocial behavior for some youth. We note that these conclusions are based on a small body of research. There is considerable controversy in the field of sociology as to how these findings from experimental interventions should be interpreted, with some concluding that individual behavior is more strongly influenced by characteristics of individuals than by characteristics of neighborhoods (Clampet-Lundquist & Massey, 2008) and others arguing that these RCTs do not provide a fair test of neighborhood effects because they involve individual- rather than neighborhood-level interventions (Sampson, 2008). Yet another possibility relates to the timing of the intervention in the lives of the children under study, where the detrimental effects of disadvantage may have already been transmitted to the children and set into motion a developmental progression of antisocial behavior that a later move to a low-poverty neighborhood was not able to reverse.

Conclusions

We identified eight categories of risk factors for children's antisocial behavior that have been identified consistently in longitudinal, epidemiological studies. We reviewed data from studies using quasi-experimental designs and statistical matching methods to evaluate whether the effects of these risk factors on children's antisocial were likely to be causal. Researchers interested in learning more about methods designed to facilitate causal inference will benefit from recent articles aimed at psychologists (D'Onofrio & Lahey, 2010; Foster, 2010; Foster & Kalil, 2008; Lahey & D'Onofrio, 2010; Rutter, 2007; Shadish, 2010; West & Thoemmes, 2010).

For some of these categories, there was little support for causal effects. In the case of smoking during pregnancy, studies using a range of research designs showed that children whose mothers smoked during pregnancy had elevated levels of antisocial behavior compared with children whose mothers did not smoke during pregnancy because these groups differed on correlated family and genetic risks for antisocial behavior. Similarly, the bulk of evidence indicated that children born to alcoholics are at elevated risk for antisocial behavior because they inherit a genetic liability for externalizing spectrum problems. For research in these areas to remain productive, investigators will need to use quasi-experimental or experimental designs to show that these are likely to be causal risk factors after all or to show that there are causal effects for particular subgroups or at particular ages. It is important for the field to not lose sight of the fact that although these risk factors may not be causes of youth antisocial behavior, they may cause other undesirable outcomes. For example, there is substantial evidence from experimental and nonexperimental designs that

smoking during pregnancy is a cause of poor neonatal health, including increased rates of infant mortality (Salihu & Wilson, 2007). To conclude from this review that mothers should not be discouraged from smoking during pregnancy would be the wrong conclusion.

The literature on neighborhood disadvantage was much smaller than the literature on smoking during pregnancy, but RCTs showed that youth who were randomly selected to move from disadvantaged to more affluent neighborhoods engaged in as much antisocial behavior as youth who were randomly selected to remain in disadvantaged neighborhoods. It is unclear whether RCTs to change characteristics of neighborhoods (e.g., an intervention to improve collective efficacy within a neighborhood rather than an RCT to move individual families from one neighborhood to another) would have stronger causal effects on antisocial behavior among youth living within those neighborhoods.

For the majority of risk categories, including maltreatment, harsh and coercive discipline, divorce, maternal depression, parental antisocial behavior, adolescent motherhood, peer deviance, and poverty, there was some evidence of social causation and some evidence for alternative hypotheses involving familial confounding, reverse causation, and social selection. We note that the research base for some of these risk factors was relatively small (e.g., parental antisocial behavior, divorce). These findings have two implications. First, because the relationship between risk exposures and children's antisocial behavior is likely to be reciprocal over time (i.e., social causation and selection are implicated), this implies that interventions can be effectively directed at multiple targets, such as those involving the child's family and those involving the youth's own behavior. These interventions will benefit from being developmentally informed. As illustrated in work carried out by Gerald Patterson and the team at the Oregon Social Learning Center (Reid et al., 2002), there is a developmental progression by which antisocial behavior emerges in childhood through hostile and coercive interactions among family members that place children at risk for affiliating with delinquent peer groups (Snyder, 2002). Although it may be effective to encourage adolescents to disassociate themselves from friends who are involved in delinquency, it is likely to be more effective to intervene earlier in the process, before behavioral tendencies are firmly established.

The fact that social causation, social selection, reverse causation, and familial confounding are all at play in the development of antisocial behavior also has implications for how effective one can expect interventions to be. Children whose parents divorce or whose parents made an early transition to parenthood are likely to have more antisocial behavior problems than children whose parents did not do those things. Reducing divorce and teen birth rates is likely to reduce rates of children's antisocial behavior, but intervention effects may be relatively modest. Nevertheless, even modest intervention effects are likely to have substantial public health significance if they are cost-effective and targeted at risk factors that affect a large number of people (Rosenthal, 1994).

Experimental, quasi-experimental, and statistically innovative designs are crucial for identifying whether risk exposures have causal effects. Once causal effects have been identified, the next step is to identify the mechanisms by which they operate. Take, for example, parental divorce. Findings from a range of research designs suggest that experiencing a divorce causes increases in

children's antisocial behavior. But this is merely a starting point. Does divorce lead to increases in youth antisocial behavior because of the accompanying drop in income that many divorced parents experience? As reviewed earlier, low income is likely to have causal effects on youth antisocial behavior, making it a plausible mediator. Does divorce lead to changes in parenting? RCTs demonstrate that in the absence of targeted interventions, harsh and coercive discipline increase and parental warmth decreases and these changes are associated with increases in youth antisocial behavior (Forgatch et al., 2009). However, more research is needed to look at other aspects of parenting that are likely to change following a divorce, including monitoring and supervision. Can the adverse effects of divorce be explained by high levels of interparental conflict that either predate or follow from the divorce? More research is needed to establish that interparental conflict has causal effects on youth antisocial behavior. Although causally informative studies exist, it is difficult to integrate the findings because each study measures marital conflict in a different way (Cummings, Faircloth, Mitchell, Cummings, & Schermerhorn, 2008; Harden, Turkheimer, et al., 2007; Meyer et al., 2000). Moreover, these studies involve marital conflict in intact families, and the effects of conflict between married parents may differ from the effect of conflict among divorced or separated parents.

Some of the methods we have reviewed are informative about differential susceptibility. For example, the analytic approach that combines group-based trajectory and propensity score models is capable of testing whether effects of exposures on children's antisocial behavior vary across different developmental trajectories. For example, affiliating with deviant peers may have a stronger adverse effect on the antisocial behavior of boys who, to that point, have been engaging in low to moderate levels of antisocial behavior compared with boys who are already engaging in very high levels of antisocial behavior. In theory, this hypothesis could be tested by modeling trajectories of antisocial behavior through childhood, measuring the propensity to affiliate with deviant peers in adolescence, and estimating the effect of deviant peer affiliations on adolescent antisocial behavior in matched groups within each behavioral trajectory (e.g., Odgers, Caspi, et al., 2008). In practice, there may be constraints on which hypotheses can be tested given properties of specific trajectory groups. For example, in their work on gang affiliations, Haviland et al. (2007) were unable to estimate effects of joining a gang among boys who had followed a trajectory of chronic, highly antisocial behavior since childhood because the distribution of propensity scores among the gang joiners versus nonjoiners was substantially dissimilar and suitable matches could not be identified.

Identifying mechanisms by which risk factors lead to increases in children's antisocial behavior may ultimately lead to investigations of how psychosocial risk factors "get under the skin." Recent models of the neurobiology of children's antisocial behavior highlight the additive and interactive effects of genetic factors and early childhood adversity on serotonergic and stress response systems, with downstream effects on children's antisocial behavior (Susman, 2006; van Goozen, Fairchild, Snoek, & Harold, 2007). More research is needed that crosses multiple levels of analysis—"from neurons to neighborhoods" in popular parlance (Shonkoff & Phillips, 2000)—in order to identify neurobiological factors that are implicated in the emergence and persistence of children's antisocial behavior. Identification of neurobiological mechanisms

involved in children's antisocial behavior may inform pharmacological treatments (e.g., those targeting the hypothalamic–pituitary–adrenal axis) that, in conjunction with psychosocial interventions involving children and their families, may be optimally effective in reducing antisocial behavior (van Goozen & Fairchild, 2008).

Understanding the causal pathways by which risk factors lead to increases in children's antisocial behavior may also require research that captures children's contexts and behaviors with higher resolution, particularly around critical turning points in development. Such research could provide the best of all worlds by using standard longitudinal designs to capture long-term trends in development and embedding within these designs finer grained measures of processes that could be informative about causal chains. These types of measurement burst designs (Nesselroade, 1991) have been applied in other areas of psychology and health research (for review, see Sliwinski, 2008) to capture short-term changes and dynamic processes within individuals, but have not yet been applied to the study of antisocial behavior. Such a research effort would require careful consideration of key developmental periods in which antisocial behavior develops, the specific risk factors involved in the emergence (or maintenance) of antisocial behavior during those periods, and how densely risk factors should be assessed.

The quasi-experimental and statistically innovative methods we reviewed each have their strengths and weaknesses. Research is a cumulative enterprise, and findings that converge across different samples, methods, and measures are likely to be robust. For example, a recent article examined the relationship among stressful life events, prior depressive episodes, and major depressive disorder (MDD) in a sample of adult twins (Kendler & Gardner, 2010). There is considerable controversy in the literature about the causal relations among these variables. The authors used both co-twin control and propensity score matching to estimate the causal impact of stressful life events on MDD. Both methods match individuals who are exposed versus those who are unexposed to stressful life events on a range of covariates, but they make different assumptions that are often violated in practice. Although propensity score methods explicitly match exposed and unexposed individuals on a set of measured covariates and test whether “balance” on relevant covariates has been achieved, they assume—unrealistically—that all relevant confounders have been included in the estimate of the propensity score. In contrast, the co-twin control method automatically matches discordant pairs for genetic factors (completely in the case of MZ twins) and shared environmental factors, but does not automatically match discordant pairs for nonshared experiences nor does it test whether balance is achieved within pairs on those nonshared measures. Together, these methods offer complementary approaches to testing whether stressful life events play a causal role in increasing risk for depression.

Finally, we note that the quasi-experimental and statistically innovative methods we have described could be usefully applied to studies of adult antisocial behavior and desistance from crime. Illustrations of this application can be found in the literature on marriage as promoting desistance from antisocial behavior (Burt et al., 2010; Horney, Osgood, & Marshall, 1995; Sampson, Laub, & Wimer, 2006). Indeed, sibling studies could be particularly informative in this vein as the environments siblings inhabit increas-

ingly diverge as they get older, but family-of-origin effects and—in the case of MZ twins—genetic factors will be shared.

We also encourage researchers to consider methods that are commonly employed in other disciplines but that have been rarely applied to facilitate causal inference in studies of antisocial behavior. These include the use of instrumental variables as well as regression discontinuity and other longitudinal change models that leverage the power of estimating within individual effects. The use of instrumental variables involves an approach to the statistical analysis of a particular type of natural experiment that capitalizes on situations where external forces, such as policy changes or forces of nature, create conditions that mimic a randomized experiment. For example, “instruments” such as changes in policy (e.g., increase in minimum wage within a state) are often used because they are unrelated to the risk factor under study (e.g., change in family income) and unrelated to factors that tend to be associated with family income (e.g., parental education). These types of events (or *exogenous shocks*) provide an opportunity to disentangle the effects of a risk factor from other factors that are typically interwoven with it in life. Although approaches using instrumental variables are not without their limitations (see Foster, 2010 and Gennetian et al., 2008, for a fuller discussion), they are currently underutilized in psychology and, under the right conditions, can be valuable tools for facilitating causal inference.

Similarly, regression discontinuity and longitudinal change models that estimate the effect of a given risk factor on an individual's trajectory of antisocial behavior or on moment-to-moment changes in his or her antisocial behavior can also be leveraged to provide compelling tests of causal theories (Collins, 2006; Shadish et al., 2002). For example, researchers interested in studying the effects of familial conflict on children's antisocial behavior may want to observe whether the antisocial behavior of a given child is higher on days when the child is, versus is not, exposed to conflict at home. Using each child in the study as his or her own control to estimate within-individual effects of conflict on antisocial behavior can be a powerful method for understanding how, when, and for whom high levels of familial conflict may matter.

Antisocial behavior that emerges in childhood often persists into adulthood and is associated with poor educational, employment, interpersonal, and physical health outcomes across the life course (Moffitt, 2006; Odgers, Moffitt, et al., 2008). Effective interventions to prevent the emergence of antisocial behavior or to deflect youth who are on an antisocial trajectory are dependent on empirical research that can identify causal impacts of psychosocial (and biological) risk factors for antisocial behavior. The quasi-experimental designs and statistically innovative methods reviewed here—with their strengths and weaknesses—can be combined with careful longitudinal, epidemiological research to further advance our knowledge about key risk factors for antisocial behavior. Findings from these studies will provide a springboard for intervention research and for basic research into the mechanisms by which these risk factors exert their effects.

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Received April 28, 2011

Revision received September 15, 2011

Accepted September 16, 2011 ■

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