Development and Psychopathology, **11** (1999), 101–126
Copyright © 1999 Cambridge University Press
Printed in the United States of America

Developmental pathways to antisocial behavior: The delayed-onset pathway in girls

PERSEPHANIE SILVERTHORN AND PAUL J. FRICK
University of Alabama

Abstract

Recent research has suggested that there are two distinct trajectories for the development of antisocial behavior in boys: a childhood-onset pathway and an adolescent-onset pathway. After reviewing the limited available research on antisocial girls, we propose that this influential method of conceptualizing the development of severe antisocial behavior may not apply to girls without some important modifications. Antisocial girls appear to show many of the correlates that have been associated with the childhood-onset pathway in boys, and they tend to show impaired adult adjustment, which is also similar to boys in the childhood-onset pathway. However, antisocial girls typically show an adolescent-onset to their antisocial behavior. We have proposed that these girls show a third developmental pathway which we have labeled the "delayed-onset" pathway. This model rests on the assumption that many of the putative pathogenic mechanisms that contribute to the development of antisocial behavior in girls, such as cognitive and neuropsychological deficits, a dysfunctional family environment, and/or the presence of a callous and unemotional interpersonal style, may be present in childhood, but they do not lead to severe and overt antisocial behavior until adolescence. Therefore, we propose that the delayed-onset pathway for girls is analogous to the childhood-onset pathway in boys and that there is no analogous pathway in girls to the adolescent-onset pathway in boys. Although this model clearly needs to be tested in future research, it highlights the need to test the applicability of current theoretical models for explaining the development of antisocial behavior in girls.

There is no shortage of statistics to underscore the pressing need to gain a better understanding of the development of severe antisocial behavior in youth. The Office of Juvenile Justice and Delinquency Prevention (OJJDP) reports that juvenile violent crime arrests have increased 100% between 1983 and 1992, and the OJJDP estimates that juvenile crime will more than double again by the year 2010 (Snyder & Sickmund, 1995). In general, more boys than girls commit crimes at a ratio of about 3.9 : 1 (Butts et al., 1995). Boys outnumber girls within the psychiatric literature as well, with a male:female ratio of Conduct Disorder of about 4 : 1 (Cohen et al., 1993; Shaffer et al., 1995). Despite the unequal rate at which boys and girls commit crimes, both sexes are contributing to the escalating crime rate. Between 1988 and 1992, the number of juvenile offenders seen in court for all delinquent acts rose 26% for boys and 27% for girls. For status offenses, the increase was 17% for boys and 20% for girls (Butts et al.). Therefore, while clearly remaining below the prevalence found in boys, the absolute number of severely antisocial girls is rapidly increasing, along with the associated costs to society which result from the behaviors of these girls. Unfortunately, despite the increasing rate of severe antisocial behavior in girls, much of the research in both the delinquency and psychiatric literature on severe antisocial behavior has focused almost exclusively on boys. Little attention has been paid to the similarities and differences in the development of antisocial behavior in girls and boys.

Recently, a compelling theoretical model

Persephanie Silverthorn is now with the Department of Psychology, University of North Texas.

Address correspondence and reprint requests to: Persephanie Silverthorn, Department of Psychology, University of North Texas, P.O. Box 311280, Denton, TX 76203; E-mail: psilver@unt.edu.

https://doi.org/10.1017/S0954579499001972 Published online by Cambridge University Press

for explaining the development of antisocial behavior in boys has been developed, which proposes that antisocial behavior in boys emerges from distinctive combinations of individual characteristics and environmental factors, known as pathways. In this article, we start by providing a brief overview of this multiple pathway model. We believe that this will offer a theoretical framework within which to interpret girls' antisocial behaviors. We then review the available, albeit quite limited, literature on antisocial behavior in girls. Based on this review, we suggest that there is sufficient reason to question the applicability of the multiple pathway model in explaining the development of antisocial behavior in girls. We propose that several important modifications in the theoretical model need to be considered to explain the existing research on antisocial girls and to guide future research on the development of severe antisocial behavior in girls.

Developmental Trajectories to Antisocial Behavior in Boys

Overview of the theoretical model

There have been several highly influential articles that have described the development of severe antisocial behavior through at least two distinct developmental trajectories: one in which the onset of severe antisocial behavior begins in childhood and the second in which the onset of severe antisocial behavior coincides with the onset of adolescence (Hinshaw, Lahey, & Hart, 1993; Moffitt, 1993a). To illustrate the strong influence that this method of conceptualizing antisocial behavior has had on the field, the most recent version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994) has adopted this approach as part of its nomenclature for distinguishing subtypes of Conduct Disorder (i.e., Childhood-Onset Type, Adolescent Onset Type). One of the major reasons that this method of conceptualizing severe antisocial behavior has been so influential is that it has proven to have great predictive utility. That is, longitudinal studies have consistently shown that one of the best predictors of which children with severe antisocial behavior are most likely to continue to show antisocial behavior into adulthood is the onset of severe conduct problems prior to adolescence (e.g., Frick & Loney, in press; Loeber, 1991; Robins, 1966).

However, in addition to the predictive utility of this model, it has the potential for guiding our understanding of different causal pathways to the development of severe antisocial behavior. This relevance to a causal theory is exemplified by the work of Moffitt and colleagues (Moffitt, 1993a; Moffitt, Caspi, Dickson, Silva, & Stanton, 1996). These authors have outlined numerous differential correlates to the two patterns of behavior, and they have weaved these divergent sets of correlates into a theoretical model that proposes separate causal mechanisms for the two developmental pathways. In Table 1, we summarize the results of Moffitt's work, highlighting both the divergent correlates and the hypothesized divergent causal mechanisms that have been proposed to account for these correlates.

One difference between children in the two pathways is that children in the childhood-onset group are characterized by markedly more aggression than children in the adolescent-onset group, which may be a central factor leading to the continuity of the antisocial behavior for children in the former pathway (Roff & Wirt, 1984; Stattin & Magnusson, 1984). Also, children with the childhood-onset pattern of behavior tend to follow a particular developmental progression of conduct problem behaviors in which less severe oppositional, negative, and argumentative behaviors are present very early in a child's development, preceding the more severe aggressive and antisocial behaviors (see also Hinshaw et al., 1993; Lahey & Loeber, 1994). In contrast, this developmental sequence does not appear to be as common for boys with an adolescent-onset to their antisocial behavior (Moffitt et al., 1996). Furthermore, the childhood-onset group is characterized by having higher rates of cognitive/neuropsychological dysfunction, such as having low intelligence (especially on measures of verbal intelligence and indices of executive functioning and planning abilities) and having a much higher rate of Attention-

https://doi.org/10.1017/S0954579499001972 Published online by Cambridge University Press

Table 1. *Summary of Moffitt's theoretical model to explain differential correlates to two developmental trajectories to antisocial behavior*

Developmental Trajectory	Differential Correlates	Proposed Mechanism
Childhood-onset	High rates of physical aggression, early onset of negative and argumentative behavior that precedes more severe antisocial behavior, high rates of neuropsychological dysfunction, a cold and callous interpersonal style, and high rates of family dysfunction	Transactional process of child with difficult temperament evoking series of failed parent–child encounters that prevent child from learning prosocial interactional skills and leads child to become ensnared in consequences of his/her antisocial behavior
Adolescent-onset	Endorses a rebellious personality style that rejects traditional status hierarchies and religious values and endorses acceptance of experimentation with drugs and alcohol	Exaggeration of natural rebellious process set up by maturity gap between biological/cognitive maturity and societal acceptance of adult status

Note: This summary is based on the work of Moffitt and colleagues (Moffitt, 1993a; Moffitt et al., 1996).

Deficit/Hyperactivity Disorder (ADHD) than children with the adolescent-onset pattern of behavior (see also Moffitt, 1993b).

The two patterns of antisocial behavior also appear to be associated with different personality traits. The childhood-onset group shows a personality profile characterized by impulsive and impetuous behavior and a cold, callous, alienated, and suspicious interpersonal style (Moffitt et al., 1996). In contrast, children showing the adolescent-onset pattern seem to desire more close relationships with others, yet tend to reject traditional status hierarchies and religious rules (Moffitt et al.). In addition, children with the childhood-onset pattern of antisocial behavior seem to come from much more dysfunctional family environments, characterized by a high rate of parental psychopathology, a high rate of family conflict, and the use of dysfunctional parenting practices than children with an adolescent-onset (see also Frick, 1994; Loeber & Stouthamer–Loeber, 1986).

As illustrated in Table 1, Moffitt and colleagues have taken these divergent sets of correlates and proposed the operation of two distinct causal mechanisms to account for these correlates. In the childhood-onset group, the process involves the "juxtaposition of a vulnerable and difficult infant with an adverse rearing context that initiates . . . a transactional process in which the challenge of coping with a difficult child evokes a chain of failed parent–child encounters" (Moffitt, 1993a, p. 682). This transactional process leads a child to "miss out on opportunities to acquire and practice prosocial patterns of behavior" (p. 683), which in turn leads him to "become ensnared by the consequences of a lifelong pattern of antisocial behavior (e.g., teenage parenthood, drug abuse, school drop out, poor work histories, criminal record) which further narrow the options for conventional behavior" (Moffitt, p. 683).

This transactional model outlined by Moffitt (1993a) to explain the childhood-onset pattern of behavior is not very different from the transactional models used by other authors to explain the development of antisocial behavior in children (e.g., Patterson, Reid, & Dishion, 1992). What is unique to this theoretical model, however, is the clear specification of *different* processes involved in the adolescent-onset pattern of antisocial behavior. Moffitt reasons that some level of antisocial behavior is almost normative in adolescence. In a community sample of youth in New Zealand, only 7% of males reported engaging in *no* delinquent or illegal activities, including relatively minor status offenses such as drinking alcohol, using a fake ID, etc. (Krueger et al., 1994). This normative pattern of behavior is viewed as being a reaction to the "maturity gap" that has been created in many industrial-

https://doi.org/10.1017/S0954579499001972 Published online by Cambridge University Press

ized societies in which there is a 5 to 10 year span between biological/cognitive maturity and socially accepted adult status. Specifically, teens develop fully physically mature bodies as early as ages 12 or 13 years, yet are denied adult status and activities until age 18 or 21 years. Engaging in status offenses, which by definition are illegal only because they are committed by a minor, engenders feelings of independence and "adulthood" for the average adolescent.

It is clear that the rate and severity of the antisocial behavior exhibited by children in the adolescent-onset pattern of behavior is not normative. This group shows a high rate of severe antisocial behavior that operates at a high cost to society and results in significant impairment to the adolescents showing this behavior (e.g., Moffitt et al., 1996). However, adolescents with this pattern of behavior are likely to have a rebellious personality style that makes them more likely to have an exaggeration of the normal development process outlined above. The severity of their behavior is partly a result of this personality predisposition and partly a form of "social mimicry" in which the antisocial behavior mimics the behavior of adolescents from the childhood-onset group in a misguided attempt to gain a sense of maturity (Moffitt, 1993a). This process provides a rationale for why this group may be less likely to continue their antisocial behavior into adulthood. Once societal acceptance of adult status is achieved, the major motivation underlying the antisocial behavior is no longer present.

Extensions of the basic model

As with any theoretical model, this two pathway model has both strengths and weaknesses in its ability to explain the development of severe antisocial behavior. One of its clear strengths is that it explicitly recognizes the possibility that there may be multiple causal pathways to the development of severe patterns of antisocial behavior. Also, it incorporates the important predictive utility of this distinction, and it explains the many divergent correlates to the two patterns of behavior. One of its weaknesses, however, is that it treats the childhood-onset pathway as a homogeneous group, and there is growing evidence that this may not be warranted. For example, in a longitudinal study of a birth cohort from New Zealand, Moffitt and colleagues found that only 54% of the 59 children who met criteria for a childhood-onset pattern of antisocial behavior showed "persistent antisocial behavior," which spanned across the childhood and adolescent years (Moffitt et al., 1996). This suggests that even within the childhood-onset group, there may be important and meaningful subgroups.

Although Moffitt alluded to subgroups within the childhood-onset pathway based on executive function deficits and attention problems (Moffitt, 1994), it is Lynam (1996) who clearly suggested that it may be the inattentive, impulsive, and hyperactive behaviors, the symptom domains associated with a diagnosis of ADHD, that may designate an important subgroup of children within the childhood-onset group. Lynam reviewed research showing that the presence of these behaviors predicted a more serious and chronic pattern of antisocial behavior. Furthermore, he proposed that these children may show certain cognitive (e.g., frontal lobe deficits) and motivational (e.g., poor response modulation) deficits that suggest that the etiology of their conduct problems may be different from other children and may make them more analogous to adults with Antisocial Personality Disorder (APD). In addition to accounting for the higher rate of ADHD in children with a childhood-onset pattern of antisocial behavior, this extension of the model could also account for the higher rates of family dysfunction in children with a childhood-onset of antisocial behavior. Specifically, Colder, Lochman, and Wells (1997) reported that highly active children were more susceptible to the influences of dysfunctional parenting practices than low active children. For example, poor parental monitoring was more strongly associated with the development of aggressive behavior in highly active children than in children with a low activity level.

Moffitt et al.'s (1996) longitudinal study suggests the possibility of another unique subgroup of children who show the childhood-

https://doi.org/10.1017/S0954579499001972 Published online by Cambridge University Press

onset pattern of antisocial behavior. In this study, one of the ways in which children who exhibited persistent antisocial behavior differed from other children in the childhood-onset group was on the presence of a cold and callous interpersonal style. This finding is analogous to studies of antisocial adults in which there is a subgroup of adults with APD who show "psychopathic traits" which are characterized by such personality features as egocentricity, lack of guilt, lack of empathy, and shallow emotions (Cleckley, 1964; Hare, 1993). Importantly, studies in adults have suggested that the presence of these traits in antisocial adults lead to a more violent and persistent pattern of antisocial behavior and a group of antisocial adults who have unique correlates (e.g., lower anxiety, poor response modulation, abnormal processing of affective stimuli) that could suggest a separate etiology (see Hare, Hart, & Harpur, 1991; Newman & Wallace, 1993).

Frick and colleagues have reported a series of studies that suggest an analogous subgroup of children may exist within children who show the childhood-onset pattern of conduct problems that are distinguished by the presence of callous and unemotional traits (Frick, in press). In a clinic-referred sample of children ($n = 120$; ages 6–13 years), almost all of whom had a substantial number of ADHD symptoms, Christian, Frick, Hill, Tyler, and Frazer (1997) identified a cluster of children who had severe conduct problems who also showed high rates of callous unemotional traits and a cluster of children with severe conduct problems without these traits. Children with conduct problems who also showed callous unemotional traits exhibited greater numbers of conduct problems, exhibited more varied conduct problems, had more contact with the police, and had a stronger family history of parental APD, all factors that have been associated with poor long term outcome in past longitudinal research (see Frick & Loney, in press; Lahey et al., 1995; Loeber, 1991). In other studies from this research group, the presence of callous unemotional traits have designated children with conduct problems who show a reward dominant response style (O'Brien & Frick, 1996) and in whom their antisocial behavior is unrelated to intelligence (Christian et al., 1997) and dysfunctional parenting practices (Wootton, Frick, Shelton, & Silverthorn, 1997), suggesting that it may designate children with different causal factors underlying their antisocial behavior. Specifically, in these children, conduct problems seem to be related to a deficit in behavior inhibition that makes them susceptible to the development of a callous and unemotional interpersonal style (Kochanska, 1993) which in turn leads them to ignore societal and/or parental norms and makes them more likely to violate the rights of others (Frick, in press).

The paper by Lynam (1996) and the series of studies by Frick and colleagues (Christian et al., 1997; Frick, in press; Wootton et al., 1996) suggest that even within the childhood-onset trajectory to antisocial behavior there may be multiple causal pathways. Although Moffitt and colleagues have acknowledged the presence of callous and unemotional traits in adulthood for those in the childhood-onset group (e.g., Moffitt, 1994; Moffitt et al. 1996), they have not emphasized the presence of these traits in childhood, nor have they elucidated the presence of subgroups in the childhood-onset group. Thus, it is important to note that the research by Lynam (1996) and Frick and colleagues have attempted to extend the typology of Conduct Disorder beyond the basic distinction between the childhood-onset and adolescent-onset trajectories.

Although these proposals are in their infancy and require much further testing, they serve to illustrate two important points. First, they demonstrate the great influence that the two-trajectory model has had in guiding research on the development of conduct problems. The two-trajectory model has provided an important starting point for refining our study of the development of antisocial behavior by distinguishing between two somewhat distinct causal pathways. This distinction sets the stage for even more refined analyses *within* these pathways. Second, these extensions of the basic model highlight some of the key correlates to the childhood-onset pathway that are likely to be important in any adequate causal theory. These include the presence of

https://doi.org/10.1017/S0954579499001972 Published online by Cambridge University Press

cognitive/neuropsychological dysfunction (e.g., poor impulse control, cognitive impairments), the presence of dysfunctional family environments, and the presence of a cold and callous interpersonal style. However, what has been missing to this point in the basic two-trajectory model and the attempts to refine and extend it, is an answer to the question of whether these lines of research would apply equally well to the development of antisocial behavior in girls. There is a general assumption that it would (e.g., Caspi, Lynam, Moffitt, & Silva, 1993; Caspi & Moffitt, 1991; Moffitt, 1994). However, much of the research on which this model was developed (Moffitt, 1993a) and extended (Frick, in press; Lynam, 1996) has been based on exclusively or predominantly male samples. In the next section, we review the available literature on antisocial behavior in girls with the goal of determining whether or not the literature supports extending this theoretical framework to girls.

Prevalence of Antisocial Behavior in Girls

Developmental variations in the gender ratio of severe antisocial behavior

The main reason usually cited for the scarcity of studies investigating antisocial behavior in girls is that there are fewer delinquent and antisocial girls than boys (e.g., Robins, 1986). As noted in the beginning of the paper, the juvenile delinquency literature and the psychiatric literature both report that the ratio of severely antisocial boys to girls is approximately 4 : 1 (Butts et al., 1995; Cohen et al., 1993; Shaffer et al., 1995). However, this overall ratio hides several important developmental differences in the sex ratio. A recent review paper (Keenan & Shaw, 1997) reported that during the first 5 years of life, there are almost no sex differences between boys and girls in most types of behavioral dysfunction. However, after age 4 years, the rate of girls' behavior problems decreases while the rate of behavioral problems for boys either increases or stays at the same rate, leading to the male predominance of behavioral problems (including conduct problems) throughout much of childhood.

Numerous studies have also noted that the sex ratio between girls and boys narrows in adolescence (e.g., American Psychiatric Association, 1994). This decrease appears to be due to the marked increase in the number of girls engaging in antisocial behaviors in adolescence combined with a much less striking increase in the rate of antisocial behavior in boys. For example, in a large epidemiological study in Canada, the reported rate for Conduct Disorder in children ages 4–11 years was 6.5% for boys and 1.8% for girls; however, for children ages 12–16 years, the rate was 10.4% for boys and 4.1% for girls (Offord, Adler, & Boyle, 1986; Offord, Boyle, & Racine, 1991). Interestingly, a more detailed picture of this change in prevalence is obtained when the different types of antisocial behaviors were studied. In the young age group, boys showed significantly higher rates of both nonaggressive and aggressive antisocial behaviors. In the adolescent age group, however, the rate of nonaggressive symptoms was almost indistinguishable between boys and girls, whereas the rate of aggressive symptoms remained much greater in boys (Offord et al., 1986, 1991). These results suggest that the reason for the decreased sex ratio in adolescence is primarily due to a large increase in nonaggressive antisocial behaviors by girls. Similar results were obtained by McGee and colleagues, who reported on a large birth cohort followed longitudinally in New Zealand (McGee, Feehan, Williams, & Anderson, 1992). The male : female ratio for severe conduct problems decreased from 2.6 : 1 at age 11 years to 0.7 : 1 at age 15 years. This striking change in the sex ratio was mainly due to a "marked increase in . . . nonaggressive conduct and oppositional disorders" for girls (McGee et al., 1992, p. 57).

Other researchers have found that the decrease in the male : female ratio for antisocial behaviors in adolescence appears to be due to girls beginning their offending at an older age. For example, Robins (1966) located adults who had been referred to a child guidance clinic in the late 1920's to determine

https://doi.org/10.1017/S0954579499001972 Published online by Cambridge University Press

their adult outcome. She found that females with APD as adults had a modal age-of-onset of 14 years or older, whereas males with adult APD had a modal age-of-onset of 9 years of age. Warren and Rosenbaum (1986) found that girls committed to the California Youth Authority between the years 1961 and 1969 had an average age-of-onset of 14 years, with approximately 66% having had their first recorded offense between 13 and 15 years (Warren & Rosenbaum). In addition, 1992 data on delinquency rates across the United States show that the rate of female delinquency jumps dramatically from 1.9/1000 at age 10 years to 9.9/1000 at age 12 years (Butts et al., 1995). These ratios can be compared to the rate for boys at 10.5/1000 at age 10 years and 36.1/1000 at age 12 years (Butts et al.). Finally, Stattin and Magnusson (1984), in their longitudinal study of Swedish children, found that delinquent girls did not differ from controls on measures of aggression at age 10 years; however, the aggressiveness scores did differ at age 13 years. Furthermore, scores at age 13 years, but not age 10 years, predicted adult criminality. They concluded that "the difference . . . suggests that aggressive behavior starts to become predictive not until girls generally reach puberty" (Stattin & Magnusson, 1984, p. 16).

The studies of the prevalence of antisocial behavior in girls can be summarized as follows. First, there appears to be few sex differences in behavioral problems during the first 5 years of life. Second, during childhood, the rate of behavioral problems in general, and conduct problems in particular, decreases for girls but not for boys, leading to a markedly discrepant male : female ratio through much of this developmental period. Third, severe conduct problems increase for both boys and girls in adolescence, but the increase is most striking for girls and for the prevalence of nonaggressive conduct problems. This leads to a notable narrowing of the male : female ratio in adolescent samples. These developmental changes in the prevalence rates of conduct problems for boys and girls need to be explained in any theoretical model of the development of conduct problems for girls. Such an explanation does not follow directly from the two-trajectory model or its extensions that were discussed previously.

Explanations for changes in prevalence across development

There have been numerous attempts to explain the finding that girls tend to exhibit lower rates of antisocial behavior in the elementary school-age years. One common explanation is that this finding is an erroneous conclusion drawn from inadequate and inappropriate measurement. Specifically, this explanation proposes that, although girls show an "antisocial trait" at the same rate as boys, the manifestation of this trait differs in either type of rate of behaviors compared to boys. For example, Crick and colleagues have found that boys tend to show much more verbal (e.g., threatening others) and physical (e.g., hitting or pushing others) aggression than girls, whereas girls show more relational aggression (e.g., excluding children from play groups, spreading rumors about children to have them rejected by others) (Crick, 1995; Crick & Grotpeter, 1995). Other authors have proposed even more dramatic differences in how antisocial tendencies may be manifested in girls, such as through the display of somatization symptoms (Lillienfeld, 1992). This possibility is based on research showing that somatization disorder and APD have a familial link, with men who show APD having a high rate of somatization in their female children and women with somatization disorder having a high rate of APD or Conduct Disorder in their male children (Cloninger & Gottesman, 1987; Frick, Kuper, Silverthorn, & Cotter, 1995). Still other authors have proposed that it may not be the type of behaviors that are different in boys and girls, but it may simply be the rate and severity of behaviors that differentiate boys and girls with the antisocial trait, which have led to arguments for the use of same-sexed norms in judging the severity of antisocial behaviors (Zoccolillo, 1993).

Each of these arguments makes the basic assumption that if sex-specific criteria were used to assess for the presence of the antiso-

https://doi.org/10.1017/S0954579499001972 Published online by Cambridge University Press

cial trait, there would be an equal prevalence of the trait for boys and girls across development. Unfortunately, there are three important issues that limit the viability of this basic assumption. First, this assumption fails to adequately explain why some girls do engage in similar antisocial behaviors as boys. A variety of studies using court records, arrests, and self-report measures indicates that juvenile crime is increasing proportionately for both boys and girls. In fact, with the exception of violent crime, the most common types of offenses are very similar between boys and girls. For example, in 1992 the most common reasons for referral to juvenile court for female offenders were property offenses (57%), person offenses (23%), public order disturbances (18%), and drug offenses (3%) (Butts et al., 1995). The most common reasons for referral were almost identical for boys: property offenses (57%), person offenses (20%), public order disturbances (17%), and drug offenses (5%). Similarly, Robins (1986) using self-report data, found that although girls engaged in antisocial behaviors less often than boys, the rank ordering of the most common antisocial behaviors was nearly identical for boys and girls.

A second limitation with the explanation that the lower prevalence of antisocial behavior in childhood is solely due to incorrect measurement practices is that there are very limited data to suggest that the use of other measurement approaches, such as use of relational aggression or measurements based on sex-specific norms, would tap the same construct or trait currently measured as antisocial behavior in boys. For example, one would expect that girls who show relational aggression or who show non-normative levels of antisocial behavior relative to other girls but not relative to boys would show similar correlates (e.g., family histories of antisocial behavior, dysfunctional family backgrounds) as boys with severe conduct problems. These findings would support the contention that they are measuring a similar trait. There is some preliminary evidence that, at least in adults, women with somatization symptoms show high rates of sensation seeking behavior similar to men with antisocial behavior patterns (Frick et al., 1995). However, with this notable exception, the construct validity of alternative methods of assessing the antisocial trait in girls has not been well established.

Third, the explanation that the male predominance of antisocial behaviors during much of childhood is solely due to inadequate measurement also does not explain well the *changes* in prevalence rates across development. As we attempt to do in the model outlined below, any explanation for the differences in prevalence of antisocial behavior between girls and boys through childhood must also explain why the ratio is different at various stages of development, such as in preschool and again in adolescence. While the notion of "heterotypic continuity" (Rutter, 1990) describes how an underlying trait may be manifested in different overt behaviors at different stages of development, for example, "biting and hitting at age four, shoplifting and truancy at ten, selling drugs and stealing cars at 16, robbery and rape at 22, and fraud and child abuse at 30" (Moffitt, 1994, p. 12), this concept does not explain the presence of antisocial behaviors in girls at age 4 years, the absence of these behaviors at age 8 years, and the apparent re-emergence of these behaviors at age 14 years.

The explanations for the male predominance of antisocial behaviors through much of childhood discussed thus far have focused on the possibility that differences in prevalence rates are largely due to inappropriate measurement of these behaviors in girls. However, because of the limitations of this explanation, some researchers have proposed that there are true differences in the prevalence of the antisocial trait in girls and boys (e.g., Eme, 1992; Gualtieri & Hicks, 1985; Keenan & Shaw, 1997; Zahn–Waxler, 1993). Both biological factors and socialization factors have been proposed to explain these differences. As examples of biological explanations, Eme summarized two theoretical models that can explain male-dominated psychiatric disorders, such as conduct disorders. Both models assume that, when a girl has a predominately male disorder, she has a more severe manifestation of the disorder. However, the two models pose different reasons as to why this might

https://doi.org/10.1017/S0954579499001972 Published online by Cambridge University Press

occur. The first model, the polygenetic multiple-threshold model, posits that genetic (and environmental) factors combine to form a "liability," and a disorder is only shown when the liability crosses a certain threshold. Those who show the disorder more often, in this case boys, have a lower threshold requiring less liability, whereas those who manifest the disorder less often, in this case girls, require a greater liability to cross the threshold. As a result, girls would be predicted to show a higher genetic loading for the disorder and a more severe manifestation of the disorder when it is shown (Cloninger & Gottesman, 1987). The second model, the constitutional variability model, posits that during prenatal development, boys develop more slowly than girls, making them biologically immature longer and more susceptible to biological insults, and allowing for a greater amount of genetic information to be transcribed (Gualtieri & Hicks, 1985; Ounsted & Taylor, 1972; Taylor & Ounsted, 1972). Girls, on the other hand, are less susceptible to minor biological trauma in early development, develop more quickly than boys, and have less genetic material transcribed. As a result, a disorder may be less prevalent in girls because the trait is subject to more genetic variability in boys. When it does occur in girls, it would be more likely to be associated with severe neurological insult leading to a more severe manifestation of the disorder. Thus, although these two models offer different explanatory mechanisms to explain the differential prevalence in psychiatric disorders, they both assume that the differences do in fact exist.

There have also been numerous explanations for the male predominance of antisocial behavior in childhood that have posited a major role of socialization (e.g., Keenan & Shaw, 1997; Zahn–Waxler, 1993). For example, Zahn–Waxler points out that in most cultures, girls are not expected to engage in aggression and antisocial behavior, and in fact, are actively discouraged from behaving against societal norms (see also Maccoby, 1986). In addition, girls reportedly suffer from more "aggression anxiety" and guilt when they do behave aggressively (e.g., Bettencourt & Miller, 1996; Frodi, Macaulay, & Thome, 1977; Parke & Slaby, 1983). Keenan and Shaw proposed that differences in socialization could account for some of the changes in prevalence in conduct problems across development for girls. Specifically, they proposed that the decline in externalizing behavior problems after the preschool years for girls could be because such behavior is "channeled into predominately internalizing problems as a result of socialization" (p. 101). In support of this proposal, they reviewed, among other data, several studies that reported that mothers encouraged more prosocial and internalizing behaviors (e.g., shyness) in their school-aged daughters but not in their sons.

This model proposed by Keenan and Shaw (1997) is the first model which has attempted to account for some of the changes in prevalence rates in conduct problems across development for girls. However, it primarily accounts for the changes in prevalence from the preschool to school-aged years and does not provide a compelling rationale for why the prevalence and sex ratio again changes in adolescence. Therefore, to date there has not been a compelling model to explain both the male predominance of severe antisocial behaviors throughout much of childhood and the changes in the sex ratio across development. In the model we outline below, we offer such a developmental explanation. In this model, we draw on many features of the models discussed in this section that, in isolation, have proven inadequate to explain the change in overt antisocial behaviors for girls over the course of their development. Also, in developing this model, we attempt to integrate findings from the existing literature on the individual and environmental characteristics of girls who show severe antisocial behavior. Some key findings from this research are reviewed in the following section.

Characteristics of Girls with Severe Conduct Problems

There are two primary bodies of research that provide data on the characteristics of severely antisocial girls: the juvenile delinquency literature and the psychiatric literature. These two lines of research have tended to follow some-

https://doi.org/10.1017/S0954579499001972 Published online by Cambridge University Press

what divergent paths. The juvenile delinquency literature has been mostly a presentation of delinquency statistics, descriptive profiles, and/or summaries of adult outcomes, whereas the psychiatric literature has been mostly epidemiological and adolescent/adult outcome studies. Together, however, they provide useful information on the characteristics of girls with severe conduct problems. Unfortunately, nearly all of the relevant studies are flawed in some way, whether it be inadequate sample size, inadequate measures, or inadequate comparison groups. Therefore, the information must be interpreted very cautiously and much more rigorous research is needed on girls with conduct disorders. However, a review of this literature suggests that four main themes consistently emerge from these studies that are relevant for developing a model to explain the development of antisocial behavior in girls.

Family dysfunction and severely antisocial girls

The first consistent theme in this literature is that antisocial girls, despite their later age of onset, come from very adverse and dysfunctional familial backgrounds (e.g., Viale–Val & Sylvester, 1993; Warren, 1986). The most frequent finding for female delinquents is that they tend to originate from non-intact families with a history of numerous parental changes (e.g., Calhoun, Jurgens, & Chen, 1993; Henry, Moffitt, Robins, Earls, & Silva, 1993; Offord, Abrams, Allen, & Poushinsky, 1979; Rosenbaum, 1989). One study in particular found that 97% of incarcerated female delinquents came from non-intact families (Rosenbaum).

In addition, other more serious family dysfunction has been reported for antisocial girls. For example, in a small sample of incarcerated female delinquents ($n = 21$), a full 90% came from violent and abusive households (Lewis et al., 1991). Of 159 adolescent girls who were committed to the California Youth Authority in the 1960s and for whom records were available (out of 240 originally placed), 37% had mothers who were charged with abuse and neglect and 67% of these girls were removed from their homes and placed in foster care (Rosenbaum, 1989). Recorded reports from parole officers indicated that 53% of the fathers (when present) and 47% of the mothers were viewed as "rejecting." A review of the relationship between child abuse and neglect and adolescent delinquency found that among incarcerated girls, as many as 86% had suffered from severe physical punishment (Widom, 1989). In a study comparing assaultive female offenders ($n = 23$), nonassaultive female offenders ($n = 27$), and female nonoffenders ($n = 23$), both offender groups scored lower on measures of family cohesion and family adaptability than the nonoffender group (Sprengelmeyer & Borduin, 1995).

Family psychopathology also appears to be high for girls with antisocial behavior. In Rosenbaum's (1989) investigation of incarcerated female delinquents, she found a noticeable presence of familial criminality: A startling 76% had at least one family member with a previous arrest, 30% had a biological father with an arrest record, and 32% had a biological mother who has been arrested. Similar findings were reported by Bergsmann (1989), with 64% of female delinquents reporting that a relative had been incarcerated. Other studies have found that the rate of parental mental illness is higher in the families of antisocial girls. For example, one study of female delinquents on probation found that, regardless of the type of crime committed, they had mothers with more maternal psychiatric illnesses than did nondelinquent girls matched for age, IQ, and school achievement (Offord et al., 1979). Overall, 54% of girls had at least one parent diagnosed with a psychiatric disorder and only 20% of girls came from homes that were "clear" from mental illness or family dysfunction. For girls committed to the California Youth Authority, 34% of biological fathers were reported to be alcoholic and 29% were "neurotic or psychotic"; rates for mothers were 31% and 27%, respectively (Rosenbaum, 1989).

Many researchers have concluded that not only do antisocial girls, irrespective of the type of antisocial behaviors exhibited, tend to have extremely negative family histories, but also that the backgrounds of female delin-

https://doi.org/10.1017/S0954579499001972 Published online by Cambridge University Press

quents are much worse than the backgrounds of male delinquents (e.g., Calhoun et al., 1993; Eme, 1992; Viale–Val & Sylvester, 1993; Warren, 1986). For example, Henggeler, Edwards, and Borduin (1987) compared 32 male and 32 female adolescents from "intact" families matched on demographic variables and divided them into 4 groups: male delinquent, male well-adjusted, female delinquent, and female well-adjusted ($n = 8$ per group). Henggeler et al. (1987) found that there was more mother–child conflict/hostility in the families of female delinquents than in those of male delinquents. This is weak support given the small number of subjects and the limited number of significant findings. More importantly, however, this and other studies which report that the families of female delinquents are more dysfunctional than the families of male delinquents used samples comprised largely of adolescents. As a result, adolescent girls are often compared to heterogeneous male samples, which according to Moffitt (1993a), are likely comprised of boys with a childhood-onset to their antisocial behavior (who tend to have greater familial dysfunction) and boys with an adolescent-onset to their behavior (who tend to have less family dysfunction). When preadolescent samples are used, few differences in family dysfunction are found between girls and boys with conduct problems (e.g., Webster–Stratton, 1996).

Adult outcome of severely antisocial girls

A second common theme in the literature on antisocial girls is the very negative outcome in late adolescence and adulthood for these girls, including arrests, psychiatric illness, drug and alcohol addiction, and the presence of numerous behaviors characteristic of an unstable and chaotic lifestyle. For example, Zoccolillo and Rogers (1991) presented outcome data for a sample of girls with Conduct Disorder 2 to 4 years after their admission to a psychiatric hospital. The girls had been admitted to the hospital when they were aged 13 to 16 years. Two to 4 years later, 50% had been arrested or were on probation, 50% had been pregnant, 41% had dropped out of school permanently, and 22% had attempted suicide. Kovacs, Krol, and Voti (1994) presented data from a longitudinal study of primary depression in outpatient clinic-referred girls. Although the initial contact occurred when the girls were between the ages of 8 and 13 years, almost no girls were diagnosed with conduct problems (including Conduct Disorder and substance abuse) prior to age 12 years (mean age of diagnosis = 12.4 years). However, of the girls that were diagnosed with conduct problems prior to age 18 years, nearly 60% experienced a teenage pregnancy. The authors report that Conduct Disorder predated the pregnancy in every case. Werner (1987), using data from a large longitudinal study, found that at age 18 years, more than half of the girls with a history of conduct problems had a psychiatric illness which necessitated inpatient or outpatient treatment.

Similarly, Bardone, Moffitt, and Caspi (1997) reported that girls diagnosed with Conduct Disorder at age 15 years were 2.6 times more likely to have a diagnosable disorder at age 21 years than were controls without a diagnosis at age 15 years. In addition, girls with Conduct Disorder had higher rates of APD symptoms and self-reported illegal behavior scores than did girls with no diagnosis at age 15 or girls diagnosed with depression at age 15 years. Stattin and Magnusson (1984) found that 50% of highly aggressive Swedish girls at age 13 years had been registered for at least one offense at age 26 years. In addition, 75% of repeat offenders had been highly aggressive at age 13 years. In contrast, if a girl was rated as normally aggressive at age 13 years, there was only a 5% chance that she would offend in adulthood (Stattin & Magnusson, 1984). Robins' (1966) longitudinal study of adults who had been referred to a child guidance clinic in the late 1920s found that an adult diagnosis of Sociopathic Personality (SO) was found in 17% of the girls referred for conduct problems, and 40% of these girls had been arrested as adults. Also, antisocial girls were at high risk for being diagnosed with Hysteria (similar to the current definition of Somatization Disorder). For girls, even as few as two antisocial symptoms in childhood were associated with a negative

https://doi.org/10.1017/S0954579499001972 Published online by Cambridge University Press

psychiatric outcome in adulthood, with only 17% of these girls being considered "well" at the follow-up assessment (Robins, 1966). Similar findings were reported in a more recent retrospective study, with 83% of women who retrospectively reported having severe antisocial behavior in adolescence reporting some type of adult diagnosis, including 39% with an externalizing disorder diagnosis (Robins, 1986). In addition, women with a juvenile history of conduct problems had a larger number of recent (within the last 6 months) adverse life events, including losing a job, having something repossessed or being sued for debts, breaking up with a lover, ending a relationship with a best friend, and moving, than did men with a childhood history of antisocial behavior (Robins, 1986). These negative adult outcomes were found whether the woman had engaged in "more masculine" (e.g., vandalism, fighting, stealing) or "less masculine" (lying, runaway, substance abuse) conduct problems (Robins).

Even more distressing findings are available in the juvenile delinquency literature. In a 7-year follow-up study of 21 matched male and female delinquents who were 14.9 years old at the time of initial assessment, 71% of the girls had been arrested with a mean number of 3.8 adult offenses, 71% had serious drug and alcohol problems, and 90% had attempted suicide, with over half of those girls attempting on more than one occasion (Lewis et al., 1991). Warren and Rosenbaum (1986) gathered follow-up information for 159 girls committed to the California Youth Authority between the years 1961 and 1969. During the 205 months after the girls were released, only 5 of the 159 girls (3%) had no further arrests following their release. The mean number of arrests for these girls was 7.2, and the mean number of convictions was 3.5 (Warren & Rosenbaum).

Thus, the available findings suggest that girls with a history of conduct problems in adolescence almost invariably have poor adult outcomes, regardless of whether the outcomes are measured as adult arrests, diagnoses of APD or other psychiatric disorders, alcohol and substance abuse, or other behaviors characteristic of an unstable and chaotic lifestyle, including losing a job, having something repossessed or being sued for debts, breaking up with a lover, ending a relationship with a best friend, and/or moving. Whether the adult outcome falls on the internalizing or externalizing disorder spectrum, these symptoms are associated with negative social and economic consequences (e.g., Moffitt, 1994) and significantly impair the functioning of these women (Robins, 1986).

Cognitive and neurological dysfunction in severely antisocial girls

A third theme in the literature on antisocial girls is that there seems to be a high rate of cognitive and/or neuropsychological dysfunction in female delinquents. For example, Phifer (1992) found that the average female delinquent had a low average IQ and had failed at least one grade, and Werner (1987) found that an IQ below 80 was the strongest predictor of adolescent delinquency ($r = .38$) in girls. Tremblay et al. (1992) found a modest negative correlation between girls' math achievement at age 10 years and aggressive ($r = -.21$) and delinquent ($r = -.20$) behavior at age 14 years. Lewis et al. (1991) reported that 43% of their sample had neurological impairment and 14% had cognitive impairment. There is also some evidence that antisocial girls may have high rates of ADHD, perhaps even higher than rates for antisocial boys. For example, Zoccolillo (1993) reported that delinquent girls have a higher rate of ADHD than delinquent boys, even though in the general population, boys have higher rates of ADHD. Loeber and Keenan (1994) in a review of gender and disruptive disorders reported a higher overlap between ADHD and CD for girls, although this overlap was moderated by the presence of developmental delays. Kovacs, Paulauskas, Gatsonis, and Richards (1988) found that in a group of outpatient clinic-referred girls, girls with ADHD were at a higher risk of developing CD than girls without ADHD. Studies of neurological dysfunction in girls with ADHD generally have been mixed, with some researchers finding that girls with ADHD have higher rates of neurological problems compared to the rates for

https://doi.org/10.1017/S0954579499001972 Published online by Cambridge University Press

ADHD boys and other researchers finding that girls have lower rates (e.g., Seidman et al., 1997; see also Silverthorn, Frick, Kuper, & Ott, 1996). Unfortunately, few studies looking at neurological dysfunction in girls with ADHD have included adolescent girls, and almost none have determined whether conduct problems were present, limiting the applicability of these studies to the question at hand.

The available data are limited at this time and interpretations must be made with caution. Nevertheless, preliminary data suggest that girls with antisocial behavior may be characterized by having high rates of school failure and lower IQs. In addition, there is some evidence that girls with antisocial behavior are more likely to have ADHD than are boys with antisocial behavior.

Suicide attempts and histories of abuse in severely antisocial girls

The findings reviewed thus far suggest that, despite the typical adolescent-onset of conduct problems for girls, antisocial girls show many characteristics that make them similar to boys with childhood-onset patterns of conduct problems; namely, high rates of family dysfunction and family psychopathology, poor adult outcomes, and high rates of cognitive and neuropsychological dysfunction (see Moffitt, 1993a). However, a fourth theme from the literature on severely antisocial girls is that there are two factors related to female antisocial behavior that do not appear as frequently in the literature on antisocial boys. The first factor that was evident from the previous discussion of poor outcomes is that there is a high rate of suicide attempts among female delinquents. For example, 19 of the 21 subjects in Lewis et al.'s (1991) study of female juvenile offenders had attempted suicide, 10 on more than one occasion. Other authors have reported attempted suicide rates ranging from 22% to 50% in samples of girls with conduct problems (Bergsmann, 1989; Zoccolillo & Rogers, 1991). A second apparently unique factor is the high rate of physical and sexual abuse among antisocial girls. Studies have suggested that between 43% and 75% of antisocial girls had been sexually abused, compared to the general population rate of 12%, and between 42% to 62% of antisocial girls had been physically abused (Bergsmann, 1989; Bureau of Justice Statistics, 1997; Calhoun et al., 1993; Lewis et al., 1991; Snell, 1994; Viale–Val & Sylvester, 1993). These rates of abuse are higher than for samples of adolescent male offenders, where 1.8% had been sexually abused and 22.5% had been physically abused (Viale–Val & Sylvester).

A Delayed-Onset Pathway in the Development of Antisocial Behavior in Girls

Research on severely antisocial girls and the two-trajectory model

We feel that the literature on severely antisocial girls that we have reviewed, both research showing changes in prevalence of severe antisocial behavior with concomitant changes in the male : female ratio across development and research on individual and environmental characteristics of girls who exhibit severe antisocial behavior, call into question the applicability of the two-trajectory model which has been proposed to explain the development of conduct problems in boys. Two key issues need to be addressed if the model is to fit the existing data on girls. First, the onset of antisocial behavior and the adult outcome of antisocial girls seems to be much more homogeneous than is found in samples of antisocial boys. That is, most antisocial girls start showing severe antisocial behavior in adolescence and the vast majority of these girls have negative outcomes in adulthood, including psychiatric illness and unstable, chaotic lifestyles. As a result, one of the primary reasons for developing the two-trajectory model, its ability to predict differential outcomes, does not appear to be applicable to girls. To reiterate, the two-trajectory model for boys was developed in part to explain the paradoxical findings that many adolescent antisocial boys do not become adult offenders, but nearly all antisocial adults were antisocial as children. No such paradoxical data have been found for

https://doi.org/10.1017/S0954579499001972 Published online by Cambridge University Press

girls; almost no girls are antisocial in childhood, and of those who engage in antisocial behaviors in adolescence, nearly all have negative adult outcomes, including arrests, psychiatric diagnoses (externalizing and/or internalizing), and/or chaotic, unstable lifestyles.

The above discussion could help to explain why many longitudinal studies of girls have failed to find a relationship between early childhood conduct problems and later adult antisocial behaviors, whereas most longitudinal studies of boys, often within the same investigation, have found a strong relationship between childhood conduct problems and adult antisocial behaviors (e.g., Achenbach, Howell, McConaughy, & Stanger, 1995; Bates, Bayles, Bennett, Ridge, & Brown, 1991; Kratzer & Hodgins, 1997; Pulkkinen & Pitkanen, 1993; Sanson, Prior, Smart, & Oberklaid, 1993; Tremblay et al., 1992). Briefly, regardless of the behavioral variable used, for example, aggression (e.g., Pulkkinen & Pitkanen, 1993) or disruptiveness (e.g., Tremblay et al., 1992), measures obtained prior to adolescence fail to correlate with adolescent and adult behaviors for girls, whereas significant correlations are typically found for boys. However, when adolescent scores are used, investigators more often find significant correlations for girls between disruptive, delinquent behaviors and antisocial behaviors in later adolescence and adulthood (e.g., Bardone et al., 1997; Kovacs et al., 1994; Lewis et al., 1991; Robins, 1966, 1986; Zoccolillo & Rogers, 1991). Interestingly, although measures of individual conduct problems obtained in childhood do not correlate with later antisocial behaviors in girls (Caspi & Moffitt, 1991), measures of family dysfunction have been found to be the strongest predictors of later antisocial behavior (Roff & Wirt, 1984).

The second key issue in developing a model of delinquency in girls is that despite the later onset of antisocial behavior for girls, the backgrounds and outcomes of antisocial girls are much more consistent with the findings for childhood-onset conduct problems in boys. For example, antisocial girls have high rates of family dysfunction (e.g., Lewis et al., 1991; Offord et al., 1979; Rosenbaum, 1989), significantly impaired adult outcomes (e.g., Robins, 1986), and high rates of cognitive and neurological deficits (e.g., low average IQ, school failure) and ADHD (e.g., Werner, 1987; Zoccolillo, 1993). These correlates are similar to those found in boys with a childhood-onset of antisocial behavior but not in boys with an adolescent-onset to their conduct problems (e.g., Hinshaw et al., 1993; Moffitt, 1993a). It is important to note that there is to date no direct comparison of antisocial girls specifically with childhood-onset boys to test this apparent similarity. However, the potential similarity in backgrounds clearly suggests that some modifications in the two-trajectory model are needed before it can be extended to girls.

We propose that these data support the presence of only one developmental trajectory for antisocial girls, a trajectory that has many commonalities with the childhood-onset trajectory in boys but for girls, typically has an adolescent-onset. The characteristics of this model and the similarities and differences between this trajectory and the two-trajectories proposed for boys are summarized in Table 2. We have labeled this trajectory the "delayed-onset" pathway. We have chosen this particular label indicate that there is a relationship between this pathway and the two pathways for boys, but that for girls (a) antisocial behaviors generally do not emerge until adolescence, (b) the underlying predisposing factors to adolescent antisocial behavior (e.g., dysfunctional family environments, cognitive and/or neuropsychological dysfunction) are likely to be present in childhood, and (c) these predisposing factors are most likely to be similar to the underlying factors in male childhood-onset CD.

Similarity in mechanisms between delayed-onset girls and childhood-onset boys

In the previous discussion of likely mechanisms that underlie the behaviors of boys in the childhood-onset group, Moffitt (1993a, 1994) focused on temperamental vulnerabilities in the child which interact with a dysfunctional family environment. In the extensions of this theory by Lynam (1996) and Frick (in press), two potential vulnerabilities that have

https://doi.org/10.1017/S0954579499001972 Published online by Cambridge University Press

Table 2. *Comparison of shared characteristics, shared mechanisms, and points of divergence between delayed-onset pathway in girls and childhood-onset and adolescent-onset trajectories in boys*

	Delayed-Onset Trajectory		
	Shared Characteristics	Shared Mechanisms	Points of Divergence
Childhood-onset trajectory	Often come from dysfunctional family backgrounds, have high rates of cognitive/ neuropsychological deficits, often show callous-unemotional traits and/or poor impulse control, and have poor adult outcome	Difficult temperaments (e.g., poor impulse control, low behavioral inhibition) interact with adverse rearing environments that lead to enduring individual vulnerabilities	Mechanisms occurring during middle childhood suppress overt antisocial behaviors in childhood, and biological and social changes during puberty encourage manifestation of these behaviors
Adolescent-onset trajectory	Onset of severe antisocial behavior is typically in adolescence and antisocial behavior is typically less aggressive/violent in nature	Changes in biological maturation (e.g., sexual and cognitive maturity) and in social milieu (e.g., greater peer acceptance of antisocial behavior, less parental supervision) lower constraints on antisocial behavior	Antisocial behavior is associated with more enduring vulnerabilities that interact with changes in biological and social milieu as child approaches adolescence

been proposed are the presence of poor impulse control which interacts with problematic parenting practices (see also Colder, Lochman, & Wells, 1997) and low behavioral inhibition which can lead to the development of a callous and unemotional interpersonal style (see also Kochanska, 1993). Although the evidence is far from definitive, we hypothesize that similar underlying factors are present in preadolescent girls who later show severe antisocial behavior in adolescence.

This contention is based on several studies that have provided at least preliminary supportive data. Christian et al. (1997) investigated the relation between callous and unemotional traits and conduct problems in a clinic-referred sample of preadolescent children ages 6 to 13 years. Cluster analyses resulted in the emergence of four clusters of children. One cluster had high rates of conduct problems without high rates of callous and unemotional traits (CP-only), another group was high on callous and unemotional traits only (CU-only), one group was high on both dimensions (CU + CP), and a clinic control group was low on both dimensions (CC). Although more males than females were found in all four categories (only 19% of the sample was female), the largest percentage of girls was found in the CU-only group (33.3%), and the smallest percentage of girls was found in the CU + CP group (11.1%). Similar results were found by Silverthorn, Hannahan, and Frick (1995) who reported that in both a clinic and community sample of preadolescent children, sex was *not* associated with scores on a measure of callous and unemotional traits. Boys and girls had similar means for the callous and unemotional measure and for each of the six items in the scale. In contrast, sex was significantly related to conduct problems with boys having more conduct problems prior to adolescence.

Also consistent with our proposed model, Moffitt and colleagues reported that childhood-onset boys and delinquent girls both exhibited personality traits that are somewhat analogous to callous and unemotional traits,

https://doi.org/10.1017/S0954579499001972 Published online by Cambridge University Press

labeled "social closeness" (Krueger et al., 1994; Moffitt et al., 1996). These authors reported that childhood-onset boys scored lower on measures of social closeness than did adolescent-onset boys, whereas girls who engaged in a variety of adolescent crimes (called versatile delinquents) had scores similar to childhood-onset boys. Similar findings emerged on measure of poor impulse control, with childhood-onset boys and delinquent girls showing more impulsivity than adolescent-onset boys. These data provide the most direct evidence that, as would be expected by the delayed-onset model, antisocial girls show callous and unemotional traits and poor impulse control which is similar to childhood-onset boys. However, what is clearly lacking in this research is a test of the key assumption of this model that callous and unemotional traits and impulsivity were present in these girls prior to adolescence and predict the development of severe antisocial behaviors in adolescence.

Why are antisocial behaviors in girls delayed until adolescence?

Our model posits that in general, the mechanisms involved in delayed-onset pathway are analogous to the processes involved in the childhood-onset pathway for boys and are illustrated in Figure 1. Similar to the childhood-onset boys, we propose that girls in the delayed-onset pathway begin life with a difficult temperament which interacts with a dysfunctional family environment, and this transactional process sets into motion a chain of failed parent–children interactions. While this process would explain the high rate of behavioral problems found in girls in infancy and early childhood, which is similar to the rate found in preschool boys (Keenan & Shaw, 1997), and the presence of antisocial behavior in adolescence and adulthood, it has thus far failed to explain, if indeed prevalence rates of antisocial behavior are lower for school-age girls compared to boys, why this shift occurs. We believe that separate mechanisms which occur during the transition to middle childhood and during the onset of puberty help to explain the change in the rates of antisocial behaviors for girls. In addition, we believe that these hypothesized mechanisms are unique to the delayed-onset model, and could not be accounted for by the two-trajectory model used for boys.

Developmental changes during the transition to middle childhood. There are several processes which may occur at the time children enter elementary school which may explain the decrease in the rate of conduct problems for girls but not boys. The low rate of serious antisocial behaviors during middle childhood may be due in part to an increase in parental socialization practices which encourage girls to express their temperament and behavioral symptoms through primarily internalizing behaviors (Keenan & Shaw, 1997). Teachers also contribute to this socialization process, attending to and reinforcing aggressive behavior in boys but not girls (Keenan & Shaw; Orenstein, 1994; Sadker & Sadker, 1994). In addition, starting around age 5 years and increasing throughout the elementary school years, peer relationships become more important and peer approval becomes increasingly desired (Serbin, Powlishta, & Gulko, 1993). Also beginning around kindergarten, children begin to adhere to gender stereotypes much more strongly than they did when they were younger, identifying themselves as either "masculine" or "feminine," and engaging in school and play activities typical of their gender (Mann, 1994; Serbin et al.). They tend to have fairly inflexible sex-role definitions, which may contribute to children expecting their peers to behave in gender appropriate ways. Thus, it may be the increasing pressure from parents, teachers, and peers for children to conform to typical gender stereotyped behaviors and the increasing desire of children to please their peers that may partially explain the childhood decrease in antisocial behaviors for girls and why there is no concomitant decrease for boys.

It is also possible that girls, but not boys, experience a number of *protective* factors in childhood, which leads to a decrease in antisocial behaviors during the elementary school years. In fact, elementary school itself may serve to buffer girls from a variety of negative

https://doi.org/10.1017/S0954579499001972 Published online by Cambridge University Press

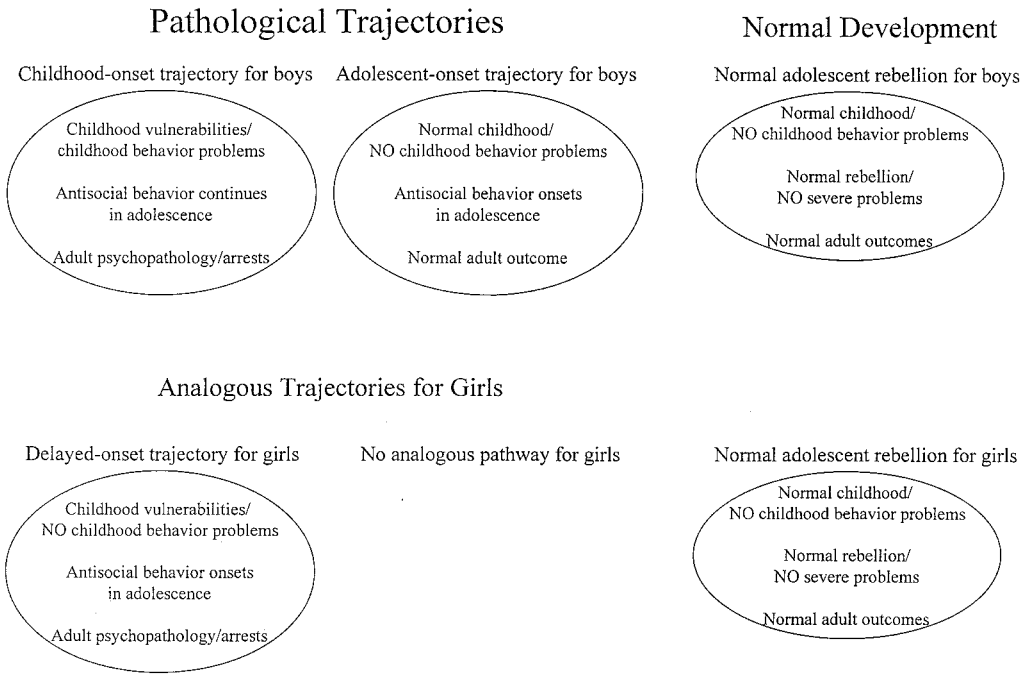


Figure 1. Pathological trajectories and normal development for boys and their analogous trajectories for girls.

effects. For example, numerous studies have shown that in elementary school, girls tend to function very well, earning higher grades, receiving more praise for passive and dependent behaviors, and receiving less negative attention from teachers, despite the fact that overall, boys receive more teacher attention (Keenan & Shaw, 1997; Mann, 1994; Orenstein, 1994; Sadker & Sadker, 1994). In addition, girls are typically seen as more socially and academically competent than boys according to both parent and teacher raters (Hetherington & Clingempeel, 1992). It also appears that preadolescent girls show fewer negative reactions than boys when faced with negative and/or aversive childhood events, such as divorce, family discord, maternal employment, and day care (Eme & Kavanaugh, 1995). In reaction to negative events such as divorce, boys tend to show problem behaviors that are more severe and more sustained than girls (Hetherington & Clingempeel).

A provocative explanation as to why this might occur comes from neuroendocrinological work with primates (Plant, 1991, 1994). Recent research suggests that the prepubertal and pubertal experiences may be very different for males and females. Simplistically presented, in males, the hypothalamic gonadotropin-release hormone (GnRH) pulse generator (which secretes the gonadotropic, or sex, hormones) is capable of functioning at an adult level in infancy (Plant, 1994). However, the GnRH pulse generator does not being to operate at an adult level until after puberty due to an active restraint on the system's ability to produce the hormones. In contrast, it appears that during infancy and childhood, females have an "immature" pulse generator which is incapable of performing at an adult level until after additional maturational changes occur as a result of the initial onset of puberty, which occurs between ages 8 and 10 years. During the prepubertal period, it takes a less intense effort to restrain the production of gonadotropic hormones for girls than for boys (Plant, 1994), presumedly because of the immaturity of the female pulse generator. These findings indicate that two important differences are evident between males and females. First, due to the more active restraint system, pre-pubertal males are in extreme hypogonadic state com-

https://doi.org/10.1017/S0954579499001972 Published online by Cambridge University Press

pared to prepubertal females (Plant, 1991, 1994). Second, during the transition to puberty, females have the added developmental task of sexual maturation of the GnRH pulse generator, whereas the pulse generator has been fully mature in males since infancy (Plant, 1994). In addition, once puberty is initiated, for males, hormone pulses occur at invarying intervals, whereas for females, hormone pulses change regularly based on ovulation cycles (Marshall, Dalkin, Haisenleder, Griffin, & Kelch, 1992).

These findings suggest that males may have a much more tumultuous time during childhood, when hormonal production is actively and strongly restrained. However, the transition to puberty should be less difficult for males, since it generally consists of the "reawakening" of the GnRH pulse regulator. In contrast, childhood may be much easier for females, since the apparent less active restraint on the hormonal system presumedly leads to a less severe hypogonadal state. However, the transition to puberty would be expected to be much more difficult, since it involves the sexual maturation of the pulse generator (Plant, 1994). Data with humans suggest additional sex differences during puberty, with girls experiencing rapid physical maturation at the same time as extreme and rapid endocrine changes and boys completing the majority of their endocrinological changes prior to external physical maturation (Angold & Worthman, 1993; Petersen, Sarigani, & Kennedy, 1991).

While the following discussion was necessarily simplistic, the differential endocrinological pattern for males and females during childhood and adolescence, if extrapolated to humans, may help to explain the changing pattern of antisocial behaviors for girls, and in general, may help to explain the changing gender patters of psychiatric disorders. The more severe reaction to prepuberty for boys might explain why boys have higher rates of psychiatric illness prior to age 10 years, and the more complex shift to puberty for girls might explain the general increase of psychiatric illness in adolescence. In addition, the less severe reaction to pre-pubertal hormones for girls, coupled with the putative effects of gender-specific socialization efforts and school-based protective factors during middle childhood, might help to explain why, despite coming from homes with severe family dysfunction, delayed-onset girls typically do not manifest severe antisocial behaviors during childhood.

These hypotheses are speculative at this point, particularly since they rely partially on the extrapolation of primate endocrinology to humans and because the data available for girls is limited. However, these speculations illustrate how the delayed-onset model, in contrast to the two trajectory model used for boys, attempts to explain the change in prevalence of antisocial behavior over time, specifically the decrease of antisocial behaviors during childhood for these at-risk girls who previously engaged in disruptive behaviors. To improve upon the existing theories, however, the delayed-onset model must also explain the apparent reemergence of antisocial behavior in puberty.

The importance of puberty. In addition to the proposing the presence of factors which may suppress the manifestation of antisocial behaviors during middle childhood for girls, we propose that there are factors unique to puberty which cause the reemergence of antisocial behaviors in these at-risk girls. Given the many changes that occur at puberty in "hormonal status, physical form, cognition, and family and peer relationships" (Costello & Angold, 1993, p. 93), the factors involved are most likely numerous and complex.

Typically, the onset of puberty is defined for research purposes as the onset of menarche (Brooks–Gunn & Warren, 1985), despite menarche occurring between 2.37 to 3.38 years, on average, after the initiation of endocrinological changes which lead to breast and/or public hair development (Herman–Giddens et al., 1997; Plant, 1994). There tends to be a difference in how boys and girls view puberty, with girls viewing menarche and associated body changes (i.e., round bodies, increase in fatty tissue) as extremely negative (Greif & Ulman, 1982; Petersen et al., 1991).

https://doi.org/10.1017/S0954579499001972 Published online by Cambridge University Press

Boys, on the other hand, tend to have a more positive view of the physical changes of puberty (Gaddis & Brooks–Gunn, 1985).

Although puberty is characterized by a significant increase in hormone levels, there is little evidence to suggest that in girls, the hormones themselves play a significant direct role in negative affect such as aggression (Brooks–Gunn & Warren, 1989). In fact, data suggest that there is a relatively weak relationship between hormones and behavior for girls, whereas this relationship was much stronger for boys (Angold & Worthman, 1993; Nottlemann, Ingoff–Germain, Susman, & Chrousos, 1990). In contrast, there is a growing boys of evidence that suggests that for girls more so than for boys, hormonal changes may be more influenced by stressful environmental factors (Angold & Worthman; Belsky, Steinberg, & Draper, 1991; Nottlemann et al., 1990). Research suggests that the presence of stressors during childhood, including family conflict and father absence, is associated with earlier menarche (Belsky et al., 1991; Moffitt, Caspi, Belsky, & Silva, 1992). Interestingly, these findings may help to explain the apparent relationship between early onset of menarche and antisocial behaviors in girls (see also Silverthorn, Green, & Loeber, 1998). Roff and Wirt (1984) found that measures of family dysfunction have been found to be the strongest predictors of later antisocial behavior for girls. Moffitt, Caspi, and colleagues (Caspi et al., 1993; Caspi & Moffitt, 1991) have reported that girls with an early-onset of menarche engage in more antisocial behaviors at ages 13 and 15. However, at age 7 and 9 years, "no significant difference between the girls who were later subdivided into four menarcheal groups" was found on measures of behavior problems (Caspi & Moffitt, p. 162), and behavior problems at age 7 years was *not* associated with early menarche (Moffitt et al., 1992). In contrast, in the same sample, family conflict and father absence at age 7 years were both significantly correlated with early menarche. Unfortunately, despite finding that family factors are related to early menarche (Moffitt et al.) and early menarche is associated with adolescent antisocial behavior for girls (Caspi & Moffitt), in none of the published research from the New Zealand data does there appear to be a direct test of a relationship between family factors and adolescent behavior problems for girls (e.g., Bardone et al., 1997; Caspi et al., 1993; Caspi & Moffitt, 1991; Henry et al., 1993; Moffitt et al., 1992; White, Moffitt, Earls, Robins, & Silva, 1990). Nevertheless, the findings encourage the speculation that for girls, a dysfunctional family environment may be directly linked to antisocial behaviors in adolescence for girls with no apparent behavior problems in childhood, and these family factors may also be related to the early onset of menarche in these girls.

The transition to puberty also involves a number of psychosocial disruptions. Studies have suggested that although boys' self-esteem tends to generally increase throughout adolescence, girls' self-esteem drops at puberty, particularly when puberty occurs during the transition to junior high (Simmons & Blyth, 1987). Junior high differs from elementary school in a variety of ways, including an increased emphasis on teacher control and discipline, less positive teacher–student relationships, and an increased emphasis on group work and public evaluation (Eccles et al., 1993), all of which tend to be more problematic for girls than for boys (Orenstein, 1994; Pipher, 1994). In addition, students earn lower grades in the first year of junior high, despite the academic work actually being easier than it was during the last year of elementary school. Petersen et al. (1991) found that when peak puberty occurred within 6 months of the transition to junior high, both boys and girls had negative reactions to the school change. However, girls tend to be more vulnerable to these negative effects since they are more likely than boys to experience puberty simultaneously with school change (43% for girls vs. 12% for boys in Petersen et al.'s study). And, as noted in the above section, evidence from primates suggests that the transition to adolescence may be hormonally more disruptive for females than males (Plant, 1994). Plus, research in humans has found that girls experience endocrinological changes in con-

https://doi.org/10.1017/S0954579499001972 Published online by Cambridge University Press

cert with physical maturation, while boys experience physical maturation after endocrinological changes (Angold & Worthman, 1993; Petersen et al., 1991). Thus, it is possible that physical and hormonal changes associated with puberty, in concert with the difficult transition to junior high school and marked decrease in self-esteem, may lead to the emergence of antisocial behaviors in at-risk girls.

Finally, there are a number of changes in a girl's social milieu that may cause a girl with a callous and unemotional interpersonal style and/or impulse control begin a pattern of antisocial behavior in adolescence. At the same time that girls are experiencing the tumultuous shifts associated with puberty, there is an increase in the overall rate of antisocial behavior in their social milieu, since both childhood-onset and adolescent-onset boys are committing delinquent acts (Moffitt, 1993a; Moffitt et al., 1996). The increase in antisocial behaviors in general could provide a vulnerable girl with greater peer modeling of and peer support for antisocial behavior than was present prior to adolescence (e.g., Caspi et al., 1993; Caspi & Moffitt, 1991; Rutter, 1990). This prevalence shift could also provide somewhat of a decrease in societal constraints against antisocial behavior because it becomes more normative, although these constraints are probably still greater for girls than boys, meaning only "vulnerable" girls should show severe antisocial behavior. Additionally, there is generally a decrease in the amount of parental supervision that occurs in adolescence that may allow for greater opportunities for vulnerable girls to act in antisocial ways (Frick, Christian, & Wootton, in press; Paikoff & Brooks–Gunn, 1991).

Continuities between adolescent and adult behaviors. Moffitt (1993a) argues that for males with a childhood-onset of delinquency, antisocial behaviors are maintained into adulthood as a result of both cumulative consequences (the combined effects of a lifetime of antisocial behaviors) and contemporary consequences (the individual characteristics which cause problems in current circumstances) (see also Moffitt, 1994). Although girls in the delayed-onset pathway typically did not engage in antisocial behaviors during childhood, the putative preadolescent correlates, including family dysfunction and a history of physical and/or sexual abuse, in combination with individual correlates, including poor impulse control, a callous and unemotional personality style, and cognitive and/or neurological deficits, would have led to these girls to have experienced at least some negative interactions during this period. In addition, by the end of adolescence, these girls would have had ample time to engage in antisocial behaviors, despite their later age-of-onset. Furthermore, we argue that antisocial girls are viewed as aberrant, even with the increase of antisocial and delinquent behaviors among boys in adolescence.

If our model outlined in the above sections is correct, then it would appear that delayed-onset girls would have had the opportunity to experience a long history of negative cumulative consequences (cf. Moffitt, 1994). In addition, individual variables such as poor impulse control and callous interpersonal traits could be associated with negative contemporary consequences, such as teen pregnancy and transitory relationships. These cumulative and contemporary consequences could very well lead to the continuation of antisocial behaviors. However, not every woman may have the opportunity (or desire) to engage in illegal acts in adulthood, although due to the net effect of these cumulative and contemporary consequences, it is hypothesized that they will experience other negative consequences, such as increased rates of internalizing disorders or alcohol and/or drug abuse. In addition, if these women are unable to control their environment or obtain desired objects via antisocial means (e.g., aggression, intimidation, stealing), they may begin using somatization symptoms to achieve the same end result (see also Frick et al., 1995; Lillienfeld, 1992). Again, although these proposals are speculative, they would help to explain the findings that delinquent adolescent girls tend to have a wide variety of negative adult outcomes, not only antisocial outcomes.

Proposed mechanisms of the delayed-onset model. The delayed-onset model combines a

https://doi.org/10.1017/S0954579499001972 Published online by Cambridge University Press

number of aspects of previous explanations that have been developed to account for the discrepant sex ratio for antisocial disorders into a comprehensive theory that we believe explains the changing prevalence of antisocial behaviors for girls from the preschool years through adulthood. Specifically, the actual mechanism of the delayed-onset model is hypothesized to occur in the following manner. First, girls in the delayed-onset pathway begin life with individual vulnerabilities, such as a difficult temperament, poor impulse control, and cognitive and/or neurological deficits which interact with a dysfunctional family environment to produce a series of failed parent–children interactions. As preschoolers, girls initially show overt behavior problems as a result of their temperament and these interactional difficulties, which is similar to the process in young childhood-onset boys. However, delayed-onset girls fail to follow the same behavioral progression as childhood-onset boys, and apparently exhibit few severe antisocial behaviors in childhood. It is possible that a combination of socialization pressures from parents, teachers, and peers for girls to engage in stereotypically female behaviors, presence of school-related protective factors, and putative positive effects of a less intense restraining system on prepubertal hormones, leads to the suppression of overt manifestation of antisocial behaviors in childhood. This lasts through the first decade of life, until puberty, when delayed-onset girls begin exhibiting overt and severe antisocial behaviors. The change in behavioral manifestation could be due to the convergence of physical, hormonal, and psychosocial changes which occur during puberty, which potentially includes a strong, negative reaction to the hormonal changes of puberty, negative psychological reactions to the physical changes of puberty, decreased self-esteem, shifts in school structure, increased prevalence of antisocial behavior by male peers, and decreased parental supervision, which encourages these already vulnerable girls to begin to manifest severe antisocial behaviors. The consequences of these cumulative effects, combined with the proximal consequences of their behaviors and interpersonal style, leads to these girls having a negative outcome in adulthood. Some of these girls may continue to manifest antisocial behaviors, but for many, the consequences of previous and current behaviors will be internalizing disorders, substance abuse, and/or somatization behaviors. In this way, the single trajectory proposed in the delayed-onset model attempts to explain not only the development of antisocial behaviors for girls, but also the changes in prevalence of antisocial behaviors over a girl's lifespan.

Normal-adolescent rebellion

A key assumption in the delayed-onset model, one that clearly differentiates it from the two-trajectory model proposed for boys, is the assumption that there is not a trajectory analogous to the adolescent-onset trajectory in girls. That is, we propose that there is not a subgroup of adolescent girls with few individual vulnerabilities and/or adverse rearing contexts who, as a function of the biological and social milieu of adolescence, begin to show a severe and impairing pattern of antisocial behavior. However, this does not suggest that typical adolescent girls are immune to developmental issues related to autonomy and identity formation, or that they do not show a normal pattern of adolescent rebellion. Like the two-trajectory model outlined for boys, the delayed-onset model for girls only focuses on explaining severe and impairing patterns of antisocial behavior (Moffitt et al., 1996). Thus, a complete classification of adolescent boys would include two pathological groups, those boys in the childhood-onset pathway and those in the adolescent-onset pathway, and one normal group comprised of normal rebellious teenagers (Moffitt, 1993a; Moffitt et al.) (see Figure 1). Like adolescent boys, typical adolescent girls are likely to engage in minor, non-serious, and non-frequent normal teenage rebellious activities that would not be classified as particularly deviant or delinquent (Krueger et al., 1994).

Results from a recent nationwide study of the prevalence of a wide range of emotional and behavioral problems support this notion (McDermott, 1996). Specifically, in younger school-age children (ages 5 to 8 years), girls

https://doi.org/10.1017/S0954579499001972 Published online by Cambridge University Press

showed much less oppositional, negative, and rebellious behaviors than boys. However, by ages 15 to 17, girls were equally likely to show these behaviors. Interestingly, this equalization of the sex ratio was not found for many other types of behaviors, such as inattentive, impulsive, and overactive behaviors. Further support for the idea that normal adolescent girls may engage in minor rebellious behaviors and, in the absence of other significant predisposing factors, will not engage in severe antisocial behavior, comes from official data for alcohol and illicit drug use. Whereas male and female rates of alcohol consumption (75.9% and 76.0%, respectively) and reports of being drunk in the past year (53.4% and 46.1%, respectively) were quite similar, a much higher rate of males than females reported using harder drugs, such as marijuana, inhalants, LSD, cocaine, and heroin (Snyder & Sickmund, 1995).

Thus, as shown in Figure 1, this group of girls should be considered normal rebellious adolescents and should not be confused as having an adolescent-onset of antisocial behavior. Similar to normal rebellious adolescent boys, neither group engages in the types of behavior which would contribute to a diagnosis of Conduct Disorder or adjudication as a delinquent. In addition, these teens do not engage in these rebellious behaviors to any significant extent. This latter group is discussed not to confuse the issue or inexplicitly suggest something other than a one-trajectory model for girls, but to make clear that we do not believe that girls are idle during adolescence or that they fail to strive for independence and autonomy. We do believe, however, that severe antisocial behavior is so aberrant for girls that only those with preexisting individual and environmental vulnerabilities will engage in these behaviors.

Summary

In conclusion, our main thesis is that without some important modifications, the emerging conceptual model of two developmental pathways in the development of severe patterns of antisocial behavior may not apply to the development of antisocial behavior in girls. In boys, there appears to be two distinct developmental pathways. One pathway onsets in early childhood, is associated with a number of pathogenic mechanisms (e.g., neuropsychological dysfunction, adverse family backgrounds, callous and unemotional interpersonal style), and is often associated with a lifelong pattern of antisocial behavior. A second pattern of severe antisocial behavior in boys onsets in adolescence, and although not normative, it seems to be an exaggeration of a normal developmental process that is partly a function of a maturity gap that is created in many industrialized societies.

This two trajectory model has been developed largely from research on boys, but there is an implicit assumption in the research that it applies equally well to the development of antisocial behavior in girls. Our review of the available research on antisocial girls calls this assumption into question. Girls typically do not begin showing severe patterns of antisocial behavior until adolescence. However, these antisocial girls appear to show many of the same pathogenic mechanisms that were associated with the childhood-onset pathway in boys and which are hypothesized to make them vulnerable to developing severe antisocial behavior. We have proposed that these girls show a third developmental pathway which we have labeled the "delayed-onset" trajectory. In this pathway, girls are hypothesized to share many of the vulnerabilities of the early-onset boys but do not manifest severe antisocial behavior until adolescence when there are significant changes in girls' biological and social milieu. In contrast to the two-trajectory model, the delayed-onset model explicitly offers hypotheses to explain the changing manifestation of antisocial behaviors in girls from preschool to adulthood.

This model, which we have labeled as the "delayed-onset" model, was designed to offer a conceptual model with many clear and testable predictions (see Table 2; Figure 1) that can guide research on the development of antisocial behavior in girls, a sorely neglected area of research. For example, prepubertal girls who show the pathogenic factors out-

https://doi.org/10.1017/S0954579499001972 Published online by Cambridge University Press

lined in this paper, such as poor impulse control or callous unemotional traits, could be followed into adolescence to determine if these factors are associated with the development of severe antisocial behavior in adolescence. In addition, tests can be made to determine if girls with a difficult temperament at age 3 years decrease their antisocial behaviors during childhood and then subsequently increase their rate of antisocial behaviors in adolescence. These are just two examples of the many predictions that follow from this conceptual model but are quite different from the predictions that would be made from the unmodified two-trajectory model. We recognize that our model is in its early stage of development, and it is based on a limited and methodologically flawed literature. However, it offers many testable predictions to guide future research, and its ability to withstand these tests will ultimately determine its scientific merit.

Even with the many likely revisions that will be needed as the data accumulate, this model will hopefully spur further research on the development of antisocial behavior in girls. It clearly calls into question the practice of using mixed adolescent samples when conducting research based on the two-trajectory model. If our model is correct, much clearer support of the model would be obtained in samples limited to boys (see Moffitt et al., 1996). The way groups are typically formed in this research is based solely on the age of the sample. In mixed adolescent samples, this would combine adolescent-onset boys with delayed-onset girls, who we propose are much more like the childhood-onset boys on most background factors and on measures of negative outcomes. Given how influential the two-trajectory model has become to the field, this is a critical methodological issue for future studies to consider.

References

Achenbach, T. M., Howell, C. T., McConaughy, S. H., & Stanger, C. (1995). Six-year predictors of problems in a national sample of children and youth: I. Cross-informant syndromes. *Journal of the Academy of Child and Adolescent Psychiatry, 34,* 336–347.

American Psychiatric Association. (1994). *The Diagnostic and Statistical Manual of Mental Disorder* (4th ed.). Washington, DC: Author.

Angold, A., & Worthman, C. W. (1993). Puberty onset of gender differences in rates of depression: A developmental, epidemiologic and neuroendocrine perspective. *Journal of Affective Disorders, 29,* 145–158.

Bardone, A. M., Moffitt, T. E., & Caspi, A. (1997). Adult mental health and social outcomes of adolescent girls with depression and conduct disorder. *Development and Psychopathology, 8,* 811–829.

Bates, J. E., Bayles, K., Bennett, D. S., Ridge, B., & Brown, M. M. (1991). The origins of externalizing behavior problems at eight years of age. In D. J. Pepler & K. H. Rubin (Eds.), *The development and treatment of childhood aggression* (pp. 93–120). Hillsdale, NJ: Erlbaum.

Belsky, J., Steinberg, L., & Draper, P. (1991). Childhood experience, interpersonal development, and reproductive strategy: An evolutionary theory of socialization. *Child Development, 62,* 647–670.

Bergsmann, I. R. (1989). The forgotten few: Juvenile female offenders. *Federal Probation, 12,* 73–78.

Bettencourt, B. A., & Miller, N. (1996). Gender differences in aggression as a function of provocation: A meta-analysis. *Psychological Bulletin, 119,* 422–447.

Brooks–Gunn, J., & Warren, M. P. (1985). Measuring physical status and timing in early adolescence: A developmental perspective. *Journal of Youth and Adolescence, 14,* 163–189.

Brooks–Gunn, J., & Warren, M. P. (1989). Biological and social contributions to negative affect in young adolescent girls. *Child Development, 60,* 40–55.

Bureau of Justice Statistics. (1997). *Privacy and juvenile justice records: A mid-decade status report* (BJS Publication No. NCJ-161255). Annapolis Junction, MD: Author.

Butts, J. A., Snyder, H. N., Finnegan, T. A., Aughenbaugh, A. L., Tierney, N. J., Sullivan, D. P., & Poole, R. S. (1995). *Juvenile Court Statistics: 1992*. Washington, DC: Office of Juvenile Justice and Delinquency Prevention.

Calhoun, G., Jurgens, J., & Chen, F. (1993). The neophyte female delinquent: A review of the literature. *Adolescence, 28*(110), 461–471.

Caspi, A., Lynam, D., Moffitt, T. E., & Silva, P. A. (1993). Unraveling girls' delinquency: Biological, dispositional, and contextual contributions to adolescent misbehavior. *Developmental Psychology, 29,* 19–30.

Caspi, A., & Moffitt, T. E. (1991). Individual differences are accentuated during periods of social change: The sample case of girls at puberty. *Journal of Personality Psychology, 61,* 157–168.

Christian, R., Frick, P. J., Hill, N., Tyler, A. L., & Frazer, D. (1997). Psychopathy and conduct problems in chil-

https://doi.org/10.1017/S0954579499001972 Published online by Cambridge University Press

dren: II. Implications for subtyping children with conduct problems. *Journal of the American Academy of Child and Adolescent Psychiatry, 36,* 233–241.
Cleckley, H. (1964). *The mask of sanity* (3rd ed.). St. Louis, MO: Mosby.
Cloninger, C. R., & Gottesman, I. I. (1987). Genetic and environmental factors in antisocial behavior disorder. In S. A. Mednick, T. E. Moffitt, & S. A. Stack (Eds.), *The causes of crime* (pp. 92–109). Cambridge: Cambridge University Press.
Cohen, P., Cohen, J., Kasen, S., Velez, C. N., Hartmark, C., Johnson, J., Rojas, M., Brook, J., & Streuning, E. L. (1993). An epidemiological study of disorders in late childhood and adolescence: I. Age and gender specific prevalence. *Journal of Child Psychology and Psychiatry, 34,* 851–867.
Colder, C. R., Lochman, J. E., & Wells, K. C. (1997). The moderating effects of children's fear and activity level on relations between parenting practices and childhood symptomology. *Journal of Abnormal Child Psychology, 25,* 251–263.
Costello, E. J., & Angold, A. (1993). Toward a developmental epidemiology of the disruptive behavior disorders. *Development and Psychopathology, 5,* 91–101.
Crick, N. R. (1995). Relational aggression: The role of intent attributions, feelings of distress, and provocation type. *Development and Psychopathology, 7,* 313–322.
Crick, N. R., & Grotpeter, J. K. (1995). Relational aggression, gender, and social-psychological adjustment. *Child Development, 66,* 710–722.
Eccles, J. S., Midgley, C., Wigfield, A., Buchanan, C. M., Reuman, D., Flanagan, C., & Iver, D. M. (1993). Development during adolescence: The impact of stage–environment fit on young adolescents' experiences in schools and in families. *American Psychologist, 48,* 90–101.
Eme, R. F. (1992). Selective female affliction in the developmental disorders of childhood: A literature review. *Journal of Clinical Child Psychology, 21,* 354–364.
Eme, R. F., & Kavanaugh, L. (1995). Sex differences in Conduct Disorder. *Journal of Clinical Child Psychology, 24,* 406–426.
Frick, P. J. (1994). Family dysfunction and the disruptive behavior disorders: A review of recent empirical findings. In T. H. Ollendick & R. J. Prinz (Eds.), *Advances in clinical child psychology* (Vol. 16, pp. 203–226). New York: Plenum.
Frick, P. J. (in press). Callous-unemotional traits and conduct problems: A two-factor model of psychopathy in children. In R. D. Gares, D. J. Cooke, & A. Forthe (Eds.), *Psychopathy: Theory, research, and implications for society*. Dordresch, Netherlands: Kluwer.
Frick, P. J., Christian, R. E., & Wootton, J. M. (in press). Age trends in the association between parenting practices and conduct problems. *Behavior Modification.*
Frick, P. J., Kuper, K. K., Silverthorn, P., & Cotter, M. (1995). Antisocial behavior, somatization, and sensation-seeking behavior in mothers of clinic-referred children. *Journal of the American Academy of Child and Adolescent Psychiatry, 34,* 805–812.
Frick, P. J., & Loney, B. R. (in press). Outcomes of children and adolescents with oppositional defiant disorder and Conduct Disorder. In H. C. Quay & A. E. Hogan (Eds.), *Handbook of disruptive behavior disorders.* New York: Plenum.
Frodi, A., Macaulay, J., & Thome, P. R. (1977). Are women always less aggressive than men? A review of the experimental literature. *Psychological Bulletin, 84,* 634–660.
Gaddis, A., & Brooks–Gunn, J. (1985). The male experience of pubertal change. *Journal of Youth and Adolescence, 14,* 61–69.
Greif, E. B., & Ulman, K. J. (1982). The psychological of menarche on early adolescent females: A review of the literature. *Child Development, 53,* 1413–1430.
Gualtieri, T., & Hicks, R. E. (1985). An immunoreactive theory of selective male affliction. *Behavioral and Brain Sciences, 8,* 427–441.
Hare, R. D. (1993). *Without conscience: The disturbing world of psychopaths around us.* New York: Pocket.
Hare, R. D., Hart, S. D., & Harpur, T. J. (1991). Psychopathy and the DSM-IV criteria for antisocial personality disorder. *Journal of Abnormal Psychology, 100,* 391–398.
Henggeler, S. W., Edwards, J., & Borduin, C. M. (1987). The family relations of female juvenile delinquents. *Journal of Abnormal Child Psychology, 15,* 199–209.
Henry, B., Moffitt, T., Robins, L., Earls, F., & Silva, P. (1993). Early family predictors of child and adolescent antisocial behaviour: Who are the mothers of delinquents? *Criminal Behaviour and Mental Health, 3,* 97–118.
Herman–Giddens, M. E., Slora, E. J., Wasserman, R. C., Bourdony, C. J., Bhapkar, M. V., Koch, G. G., & Hasemeier, C. M. (1997). Secondary sexual characteristics and menses in young girls in office practice: A study from the pediatric research in office settings network. *Pediatrics, 99,* 505–512.
Hetherington, E. M., & Clingempeel, W. G. (1992). Coping with marital transitions. *Monographs for the Society for Research in Child Development, 57,* (2–3, Serial no. 227).
Hinshaw, S. P., Lahey, B. B., & Hart, E. L. (1993). Issues of taxonomy and comorbidity in the development of conduct disorder. *Development and Psychopathology, 5,* 31–49.
Keenan, K., & Shaw, D. (1997). Development and social influences on young girls' early problem behavior. *Psychological Bulletin, 121,* 95–113.
Kochanska, G. (1993). Toward a synthesis of parental socialization and child temperament in early development of conscience. *Child Development, 64,* 325–347.
Kovacs, M., Krol, R. S. M., & Voti, L. (1994). Early onset psychopathology and the risk for teenage pregnancy among clinically referred girls. *Journal of the American Academy of Child and Adolescent Psychiatry, 33,* 106–113.
Kovacs, M., Paulauskas, S., Gatsonis, C., & Richards, C. (1988). Depressive disorders in childhood III. A longitudinal study of comorbidity with and risk for conduct disorders. *Journal of Affective Disorders, 15,* 205–217.
Kratzer, L., & Hodgins, S. (1997). Adult outcomes of child conduct problems: A cohort study. *Journal of Abnormal Child Psychology, 25,* 65–81.
Krueger, R. F., Schmutte, P. S., Caspi, A., Moffitt, T. E., Campbell, K., & Silva, P. A. (1994). Personality traits are linked to crime among men and women: Evidence from a birth cohort. *Journal of Abnormal Psychology, 103,* 328–338.
Lahey, B. B., & Loeber, R. (1994). Framework for a developmental model of Oppositional Defiant Disorder

https://doi.org/10.1017/S0954579499001972 Published online by Cambridge University Press

and Conduct Disorder. In D. K. Routh (Ed.), *Disruptive behavior disorders in childhood.* New York: Plenum.

Lahey, B. B., Loeber, R., Hart, E. L., Frick, P. J. Applegate, B., Zhang, Q., Green, S. M., & Russo, M. F. (1995). Four-year longitudinal study of conduct disorders: Patterns and predictors of persistence. *Journal of Abnormal Psychology, 104,* 83–93.

Lewis, D. O., Yeager, C. A., Cobham–Portorreal, C. S., Klein, N., Showalter, C., & Anthony, A. (1991). A follow-up of female delinquents: Maternal contributions to the perpetuation of deviance. *Journal of the American Academy of Child and Adolescent Psychiatry, 30,* 197–201.

Lillienfeld, S. O. (1992). The association between antisocial personality and somatization disorders: A review and integration of theoretical models. *Clinical Psychology Review, 12,* 641–662.

Loeber, R. (1991). Antisocial behavior: More enduring than changeable? *Journal of the American Academy of Child and Adolescent Psychiatry, 30,* 393–397.

Loeber, R., & Keenan, K. (1994). Interaction between Conduct Disorder and its comorbid conditions: Effects of age and gender. *Clinical Psychology Review, 14,* 497–523.

Loeber, R., & Stouthamer–Loeber, M. (1986). Family factors as correlates and predictors of juvenile conduct problems and delinquency. In M. Tonry & N. Morris (Eds.), *Crime and justice* (Vol. 7, pp. 29–149). Chicago: University of Chicago Press.

Lynam, D. R. (1996). Early identification of chronic offenders: Who is the fledgling psychopath? *Psychological Bulletin, 120,* 209–234.

Maccoby, E. E. (1986). Social groupings in childhood: Their relationship to prosocial and antisocial behavior in boys and girls. In D. Olweus, J. Block, & M. Radke–Yarrow (Eds.), *Development of antisocial and prosocial behavior: Research, theories, and issues* (pp. 263–284). Orlando, FL: Academic Press.

Mann, J. (1994). *The difference: Growing up female in America*. New York: Warner.

Marshall, J. C., Dalkin, A. C., Haisemleder, D. J., Griffin, M. L., & Kelch, R. P. (1992). GnRH Pulses: The regulators of human reproduction. *Transactions of the American Clinical and Climatological Association, 104,* 31–46.

McDermott, P. A. (1996). A nationwide study of developmental and gender prevalence for psychopathology in childhood and adolescence. *Journal of Abnormal Child Psychology, 24,* 53–66.

McGee, R., Feehan, M., Williams, S., & Anderson, J. (1992). DSM-III disorders from age 11 to age 15 years. *Journal of the American Academy of Child and Adolescent Psychiatry, 31,* 50–59.

Moffitt, T. E. (1993a). Adolescence-limited and life-course-persistent antisocial behavior: A developmental taxonomy. *Psychological Review, 100,* 674–701.

Moffitt, T. E. (1993b). The neuropsychology of conduct disorder. *Development and Psychopathology, 5,* 135–151.

Moffitt, T. E. (1994). Natural histories of delinquency. In E. G. M. Weitekamp & H. J. Kerner (Eds.), *Cross-national longitudinal research on human development and criminal behavior* (pp. 3–61). Netherlands: Kluwer.

Moffitt, T. E., Caspi, A., Belsky, J., & Silva, P. (1992). Childhood experience and the onset of menarche: A test of a sociobiological model. *Child Development, 63,* 47–58.

Moffitt, T. E., Caspi, A., Dickson, N., Silva, P., Stanton, W. (1996). Childhood-onset versus adolescent-onset antisocial conduct problems in males: Natural history from ages 3 to 18 years. *Development and Psychopathology, 8,* 399–424.

Newman, J. P., & Wallace, J. F. (1993). Diverse pathways to deficient self-regulation: Implications for disinhibitory psychopathology in children. *Clinical Psychology Review, 13,* 699–720.

Nottleman, E. D., Inoff–Germain, G., Susman, E. J., & Chrousos, G. P. (1990). Hormones and behavior at puberty. In J. Bancroft & J. M. Reinsch (Eds.), *Adolescence and puberty* (pp. 88–123). New York: Oxford University Press.

O'Brien, B. S., & Frick, P. J. (1996). Reward dominance: Associations with anxiety, conduct problems, and psychopathy in children. *Journal of Abnormal Child Psychology, 24,* 223–240.

Offord, D. R., Abrams, N., Allen, N., & Poushinsky, M. (1979). Broken homes, parental psychiatric illness, and female delinquency. *American Journal of Orthopsychiatry, 49,* 252–264.

Offord, D. R., Adler, R. J. M., & Boyle, M. H. (1986). Prevalence and sociodemographic correlates of Conduct Disorder. *The American Journal of Social Psychiatry, 6,* 272–278.

Offord, D. R., Boyle, M. H., & Racine, Y. A. (1991). The epidemiology of antisocial behavior in childhood and adolescence. In D. J. Pepler & K. H. Rubin (Eds.), *The development and treatment of childhood aggression* (pp. 31–54). Hillsdale, NJ: Erlbaum.

Orenstein, P. (1994). *SchoolGirls: Young women, self-esteem, and the confidence gap.* New York: Doubleday.

Ounsted, C., & Taylor, D. C. (1972). The Y chromosome message: A point of view. In C. Ounsted & D. Taylor (Eds.), *Gender differences: Their ontogeny and significance* (pp. 241–262). London: Churchill Livingstone.

Paikoff, R. L., & Brooks–Gunn, J. (1991). Do parent–child relationships change during puberty? *Psychological Bulletin, 110,* 47–66.

Parke, R. D., & Slaby, R. G. (1983). The development of aggression. In P. H. Mussen (Series Ed.) & E. M. Hetherington (Vol. Ed.), *Handbook of child psychology: Volume 4. Socialization, personality, and social development* (4th ed., pp. 548–641). New York: Wiley.

Patterson, G. R., Reid, J. B., & Dishion, T. J. (1992). *Antisocial boys.* Eugene, OR: Castalia.

Petersen, A. C., Sarigiani, P. A., & Kennedy, R. E. (1991). Adolescent depression: Why more girls? *Journal of Youth and Adolescence, 20,* 247–271.

Phifer, J. D. (1992). *Family stress, antisocial behavior, and the behaviorally/emotionally disturbed girl.* Unpublished doctoral dissertation.

Pipher, M. (1994). *Reviving Ophelia: Saving the selves of adolescent girls.* New York: Ballentine.

Plant, T. M. (1991). The neuroendocrine regulation of the onset of puberty in primates. In A. Negro–Vilar & Perez–Palacios (Eds.), *Serono symposium publications: Vol. 71. Reproduction, growth, and development* (pp. 75–79). New York: Rowen.

Plant, T. M. (1994). Puberty in primates. In E. Knobil & J. D. Neill (Eds.), *The physiology of reproduction* (2nd ed., pp. 453–485). New York: Raven.

https://doi.org/10.1017/S0954579499001972 Published online by Cambridge University Press

Pulkkinen, L., & Pitkanen, T. (1993). Continuities in aggressive behavior from childhood to adulthood. *Aggressive Behavior, 19,* 249–263.

Robins, L. (1966). *Deviant children grown up: A sociological and psychiatric study of sociopathic personality*. Baltimore, MD: Williams and Wilkins.

Robins, L. (1986). The consequence of Conduct Disorder in girls. In D. Olweus, J. Block, & M. Radke–Yarrow (Eds.), *Development of antisocial and prosocial behavior: Research, theories, and issues* (pp. 382–414). Orlando, FL: Academic.

Roff, J. D., & Wirt, R. D. (1984). Childhood aggression and social adjustment as antecedents of delinquency. *Journal of Abnormal Child Psychology, 12,* 111–126.

Rosenbaum, J. L. (1989). Family dysfunction and female delinquency. *Crime and Delinquency, 35,* 31–44.

Rutter, M. (1990). Changing patterns of psychiatric disorders during adolescence. In J. Bancroft & J. M. Reinsch (Eds.), *Adolescence and puberty* (pp. 124–145). New York: Oxford University Press.

Sadker, M., & Sadker, D. (1994). *Failing at fairness: How our schools cheat girls.* New York: Touchstone.

Sanson, A., Prior, M., Smart, D., & Oberklaid, F. (1993). Gender differences in aggression in childhood: Implications for a peaceful world. *Australian Psychologist, 28,* 86–92.

Seidman, L. J., Biederman, J., Farone, S. V., Weber, W., Menin, D., & Jones, J. (1997). A pilot study of neuropsychological function in girls with ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry, 36,* 366–373.

Serbin, L. A., Powlishta, & Gulko, J. (1993). The development of sex typing in middle childhood. *Monographs for the Society for Research in Child Development, 58* (Serial no. 232).

Shaffer, D., Fisher, P., Dulcan, M., Davies, M., Piacentini, J., Schwab–Stone, M. E., Lahey, B. B., Bourdon, K., Jensen, P. S., Bird, H. R., Rubio–Stipec, M., & Rae, D. S. (1995). The NIMH Diagnostic Interview Schedule for Children Version 2.3: Description, acceptability, prevalence rates, and performance in the MECA study. *Journal of the American Academy of Child and Adolescent Psychiatry, 35,* 865–877.

Silverthorn, P., Frick, P. J., Kuper, K., & Ott, J. (1996). Attention deficit hyperactivity disorder and sex: A test of two etiological models to explain the male predominance. *Journal of Clinical Child Psychology, 25,* 52–59.

Silverthorn, P., Green, S., & Loeber, R. (1998). *Onset of conduct disorder and timing of puberty in girls: Is puberty a necessary development precursor?* Manuscript submitted for publication.

Silverthorn, P., Hannahan, B. S., & Frick, P. J. (1995). Sex differences for psychopathy in a clinic and community sample. In P. J. Frick (Chair), *Applying the concept of psychopathy to understanding children with conduct problems.* Symposium presented at the annual meeting of the Association for Advancement of Behavior Therapy, Washington, DC.

Simmons, R. G., & Blyth, D. A. (1987). *Moving into adolescence.* New York: Aldine de Gruyter.

Snell, T. L. (1994). *Women in prison: Survey of state prison inmates, 1991* (BJS Publication No. NCJ-145321). Annapolis Junction, MD: Bureau of Justice Statistics.

Snyder, H. N., & Sickmund, M. (1995). *Juvenile offenders and victims: A national report.* Washington, DC: Office for Juvenile Justice and Delinquency Prevention.

Sprengelmeyer, P. G., & Borduin, C. M. (1995). *The ecology of serious offending in female adolescents.* Paper presented at the annual convention of the American Psychological Association, New York.

Stattin, H., & Magnusson, D. (1984). *The role of early aggressive behavior for the frequency, the seriousness, and the types of later criminal offenses.* Stockholm: Department of Psychology, University of Stockholm.

Taylor, D. C., & Ounsted, C. (1972). The nature of gender differences explored through ontogenetic analyses of sex ratios in disease. In C. Ounsted & D. Taylor (Eds.), *Gender differences: Their ontogeny and significance* (pp. 215–240). London: Churchill Livingstone.

Tremblay, R. E., Masse, B., Perron, D., Leblanc, M., Schwartzman, A. E., & Ledingham, J. E. (1992). Early disruptive behavior, poor school environment, delinquent behavior, and delinquent personality: Longitudinal analyses. *Journal of Consulting and Clinical Psychology, 60,* 64–72.

Viale–Val, G., & Sylvester, C. (1993). Female delinquency. In M. Sugar (Ed.), *Female adolescent development.* New York: Brunner–Mazel.

Warren, M. Q. (1986). The female offender. In H. Toch (Ed.), *Psychology of crime and criminal justice.* Prospects Heights, IL: Waveland.

Warren, M. Q., & Rosenbaum, J. L. (1986). Criminal careers of female offenders. *Criminal Justice and Behavior, 13,* 393–418.

Webster–Stratton, C. (1996). Early-onset conduct problems: Does gender make a difference? *Journal of Consulting and Clinical Psychology, 64,* 540–551.

Werner, E. E. (1987). Vulnerability and resiliency in children at risk for delinquency: A longitudinal study from birth to young adulthood. In J. D. Burchard & S. N. Burchard (Vol. Eds.), *Prevention of delinquent behavior: Vol. 10. Primary prevention of psychopathology* (pp. 16–43). Newbury Park, NJ: Sage.

White, J. L., Moffitt, T. E., Earls, F., Robins, L., & Silva, P. A. (1990). How early can we tell? Predictors of childhood conduct disorder and adolescent delinquency. *Criminology, 28,* 507–533.

Widom, C. S. (1989). Does violence beget violence? A critical examination of the literature. *Psychological Bulletin, 106,* 3–28.

Wootton, J. M., Frick, P. J., Shelton, K. K., & Silverthorn, P. (1997). Ineffective parenting and childhood conduct problems: The moderating role of callous-unemotional traits. *Journal of Consulting and Clinical Psychology, 65,* 301–308.

Zahn–Waxler, C. (1993). Warriors and worriers: Gender and psychopathology. *Development and Psychopathology, 5,* 79–89.

Zoccolillo, M. (1993). Gender and the development of conduct disorder. *Development and Psychopathology, 5,* 65–78.

Zoccolillo, M., & Rogers, K. (1991). Characteristics and outcomes of hospitalized adolescent girls with conduct disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 30,* 973–981.

https://doi.org/10.1017/S0954579499001972 Published online by Cambridge University Press