

Conduct and Oppositional Defiant Disorders

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Problems related to delinquency and youth violence in our nation are entwined in a complex web of public concern, community fear and outrage, media attention, concerted research efforts, and multifaceted prevention and intervention programs. Even though official rates of antisocial behavior (ASB) among children, adolescents, and adults in the United States showed evidence of a leveling off or slight decline during the 1990s, following decades of steady increases (Snyder & Sickmund, 1995; Zimring, 1998; Fingerhut & Kleinman, 1990), few would contend that aggression and ASB have receded as salient, impairing, disturbing, and even (in some instances) lethal problems. Indeed, notorious instances of youth violence in middle-class, suburban settings in recent years have propelled national interest in the alarmingly high rates of aggression, acting out, and even murder among young people—rates that have long been salient in impoverished, urban neighborhoods. Furthermore, levels of violence in the United States continue to surpass those in other industrialized nations (Loeber & Hay, 1997; Rutter, Giller, & Hagell, 1998). Among youths in general, the highest rates of referral for mental health services involve aggressive, acting-out, and disruptive behavior patterns, which have shown a detectable increase over the period of time from the 1960s through the 1990s (Achenbach & Howell, 1993). In addition, the threat—or reality—of violence continues to create climates of fear, intimidation, and deprivation

in many communities (Richters & Martinez, 1993). Overall, despite the ever-increasing amounts of research on this topic, the need for sound scientific efforts directed toward understanding the roots, classification, underlying mechanisms, and treatment of ASB has never been greater.

Although we base much of the organizational scheme of this chapter on the contents of the parallel chapter in the first edition of this volume (Hinshaw & Anderson, 1996), we not only update the huge literature in the field but also pursue several expanded directions. First, we pay even greater attention to the multiple causal pathways that may portend clinically significant oppositionality and aggression among children and adolescents, incorporating the constructs of equifinality (the presence of divergent etiological roots that lead to phenotypically similar behavior patterns) and multifinality (the developmental diversity of outcomes from similar initial states) (Cicchetti & Rogosch, 1996). It is clear that the behavior patterns under consideration are the products of influences at multiple levels (e.g., genetic, temperamental, family systemic, socioeconomic, school-related, community-wide), which interact and transact in complex ways. Second, given the maturation of several important prospective, longitudinal samples into adulthood, we present additional information on the extended developmental outcomes of children with both early-onset and adolescent-onset manifestations of aggression and ASB. Third, we more

explicitly feature what is known (and unknown) about female manifestations of such behavioral patterns. Accordingly, we note the increasing recognition given to a less overt and less violent form of antisocial activities—indirect or relational aggression—which appear to be particularly salient among girls. Fourth, we present a preliminary conceptual model regarding the development and maintenance of ASB patterns, recognizing that any overarching theories must recognize the considerable heterogeneity among (1) types of externalizing behavior, (2) subtypes of youths at risk for such behavior, and (3) developmental pathways or trajectories that characterize youngsters with such tendencies.

At the outset, we make clear that our chapter does not focus on species-wide influences on aggression (Coie & Dodge, 1998). Rather, we deal with influences on individual differences in aggression and ASB, through a strongly developmental perspective. In addition, given the huge literature on this topic, we direct the reader to key review articles, chapters, and books that have appeared since the first edition of this volume was published. Such works include the masterful historical, conceptual, and developmental review of Coie and Dodge (1998); key reviews of developmental issues by Loeber and Hay (1997), Loeber and Stouthamer-Loeber (1998), Maughan and Rutter (1998), and Tremblay (2000); the comprehensive edited volumes of Hill and Maughan (2001), Loeber and Farrington (1998, 2001), Quay and Hogan (1999), and Stoff, Breiling, and Maser (1997), each of which contains a large number of seminal chapters; the lucid and comprehensive book-length account of Rutter et al. (1998), and the data-rich volume on ASB plus other mental health problems by Loeber, Farrington, Stouthamer-Loeber, and Van Kammen (1998); the recent work on female manifestations of ASB by Moffitt, Caspi, Rutter, and Silva (2001); the review of the diagnostic categories of oppositional defiant disorder (ODD) and conduct disorder (CD) by Loeber, Burke, Lahey, Winters, and Zera (2000); the syntheses of young children's risk for ASB by Keenan and Shaw (1997), Campbell, Shaw, and Gilliom (2000), and Loeber and Farrington (2000); and the integrative causal model of Lahey, Waldman, and McBurnett (1999). Note that this list is far from exhaustive; our entire reference section is, of necessity, limited to selected citations.

As highlighted in the first edition (Hinshaw & Anderson, 1996), considerable theoretical con-

troversy still surrounds the field. At the most general level, there is dispute regarding the proper disciplines that should investigate antisocial activity and the optimal perspectives from which to view such behavior patterns. Indeed, because of the differing definitions of normative behavior across cultures, perhaps anthropological or sociological perspectives on antisocial functioning should receive primacy (e.g., Hirschi, 1969). Although our focus herein is directed more toward individual, familial, and social-contextual influences than toward the role of culture per se, the ascription of ASB exclusively to intraindividual causes is a real danger. Throughout this work, we develop the argument that only a subset of individuals displaying ASB patterns fall under the umbrella of mental disorder or impairment (Richters & Cicchetti, 1993), given the age- and sex-normative nature of a wide range of aggression and ASB during adolescence. On the other hand, just because a large proportion of antisocial youths appear to be those with adolescent onset, without long histories of multiple childhood impairments, does not imply that such youths are not in need of intervention (Moffitt, Caspi, Harrington, & Milne, 2002). In other words, the diagnoses of ODD and CD do not “cover the map” with respect to the personal and societal impact of aggression and violence.

Along this line, a salient theme throughout the chapter is that antisocial patterns, whether considered as dimensions of behavior or as distinct categorical entities, are heterogeneous with respect to constituent behaviors, causation, developmental mechanisms, and long-term course. Phrased alternatively, antisocial actions that appear similar at a given point in time may betray fundamentally disparate subtypes when viewed longitudinally (Loeber, 1988; Moffitt, 1993; Rutter et al., 1998). Any theories of such actions must actively consider the divergent underlying patterns and differing developmental trajectories relevant for distinct subgroups of youngsters.

Our main goals are to present current perspectives on the extensive literature surrounding patterns of aggression and ASB in childhood and adolescence (including brief coverage of the adult construct of psychopathy), and to illuminate current thinking about definitions, conceptualization, prevalence, comorbidity, and models of risk and etiology. Although we focus on the psychiatric disorders of ODD and CD, we go well beyond these categorical conceptions to consider dimensional features of aggression and ASB in child-

hood and adolescence, and alternative means of categorizing such behavioral manifestations. We emphasize throughout that ASB develops in relation to multiple influences, including biological and psychobiological risk variables, parent–child interactions, familial traits, school settings, neighborhood characteristics, peer networks, social service agencies and mental health services, and sub-cultural and societal norms; we also highlight that patterns of interaction and transaction across such influences is the rule rather than the exception in terms of the development of significant aggression and violence (Campbell, in press; Rutter et al., 1998). That is, underlying predispositions are translated into antisocial and violent behavior only through complex patterns of active engagement with the environment (Lahey, Waldman, & McBurnett, 1999). We hope that readers will come to appreciate the complexity of the issues surrounding this salient and troublesome type of behavioral disturbance, as well as the necessity of considering developmental perspectives on their etiology, maintenance, and outcome.

We begin by defining several key terms in the field and by discussing a number of core conceptual issues regarding aggression and ASB (see parallel consideration by Maughan & Rutter, 1998). We next provide a brief historical account of conceptions of ASB, covering current diagnostic criteria and related issues. After a discussion of prevalence and developmental progressions, we highlight the themes of comorbidity as well as risk and etiological factors, with emphasis on integrated, transactional models related to the development of ASB. We then provide an expanded section on sex differences, and conclude with an attempt at an integrated theoretical model of the development of aggression and ASB. Page limitations necessitate our neglecting almost entirely the topics of assessment and of prevention/intervention (for recent perspectives on these topics, see Hinshaw & Zupan, 1997; Hinshaw & Nigg, 1999; relevant chapters in Quay & Hogan, 1999; Rutter et al., 1998, Chs. 11 and 12; and McMahon & Wells, 1998, among many other sources).

TERMINOLOGICAL AND CONCEPTUAL ISSUES

Defining the Domain

Judges and juvenile justice workers, research investigators, clinicians, and societal commenta-

tors have utilized a host of terms to describe ASB in children, adolescents, and adults, yielding a sometimes chaotic level of imprecision and confusion in the field at large. Even basic definitions of ASB and aggression are problematic (see the lucid discussion in Coie & Dodge, 1998). For example, must harmful intent be present for an act to be considered aggressive? If so, key problems in defining intentionality come into play. In addition, can ASB patterns be considered in any way universal, or are judgments of such actions always constrained by cultural norms? Expanded consideration of such definitional and philosophical issues can be found in Coie and Dodge (1998), Parke and Slaby (1983), and Rutter et al. (1998).

First, from a legal perspective, child and adolescent manifestations of ASB are termed “delinquent,” and adult manifestations are called “criminal.” Indeed, with rates of imprisonment at unprecedented levels in the United States, legal definitions of antisocial activity are salient. These types of definitions have limitations for psychological analysis, however, including the usual necessity of apprehension in allowing their usage; this means that relevant investigations may index the correlates of “being caught” or of police targeting (such as ethnic discrimination or selective reporting), rather than of ASB per se. In addition, most studies of delinquency neglect of the aggressive or antisocial activities of young children, whose early, “predelinquent” behavioral patterns may be the most likely routes for investigations of risk and causal factors and of preventive intervention. Note also that delinquency may be defined by a single act rather than a pattern of related behaviors, contributing to disparate estimates of its prevalence. We point out that many investigators distinguish between “official” delinquency and “self-reported” delinquency, with the latter indexed by children’s or adolescents’ self-report disclosures of various illegal activities. Readers are cautioned to make note of the particular measurement strategy in use in any particular investigation of delinquency.

Second, empirical psychological investigations often distinguish so-called “externalizing” behavior patterns—those marked by impulsive, overactive, aggressive, and antisocial actions—from “internalizing” (e.g., anxious, dysphoric, withdrawn, thought-disordered, somaticizing) features (Achenbach, 1991). Indeed, a long tradition of research posits fundamental distinctions between these two domains with respect to underlying behavioral components, risk and etiological

factors, and long-term course (e.g., Quay, 1986). Clearly, the subject matter of interest for this chapter lies in the externalizing domain. On the other hand, we highlight at the outset that overlap between externalizing and internalizing patterns is strong. We also point out that externalizing and internalizing behavior patterns are clearly dimensional in nature, ranging from normative levels to the extreme ends of their respective continua. Whether dimensional or categorical conceptualizations of ASB better fit the underlying nature of the constituent problems is a thorny and long-standing problem, as we take up subsequently.

It is essential to recognize that within the externalizing domain—also termed “acting-out,” “disruptive,” or undercontrolled¹—a fundamental distinction exists between aggression and ASB on the one hand, and the spectrum of inattentive/impulsive/overactive symptoms, which are the constituent behavior patterns of attention-deficit/hyperactivity disorder (ADHD), on the other (Fergusson, Horwood, & Lloyd, 1991; Loney, 1987). Whereas these types of behavior frequently co-occur—as witnessed by their loading together on higher-order externalizing dimensions (Achenbach, 1991), and by the diagnostic overlap of categories reflecting oppositional or aggressive actions with disorders of attention and impulse control (Biederman, Newcorn, & Sprich, 1991)—the distinction has been validated in many investigations (see Hinshaw, 1987; Jensen, Martin, & Cantwell, 1997).² Because the overlap or comorbidity between aggressive/antisocial actions and the constituent behaviors of ADHD is quite important for the development of long-term antisocial patterns (e.g., Loeber et al., 1998; Moffitt, 1990; Rutter et al., 1998), we scrutinize this association later in the chapter.

Third, in the psychiatric tradition of forming diagnostic categories, CD and ODD are the constituent disorders of the “disruptive behavior disorders” category in the *Diagnostic and Statistical Manual of Mental Disorders*, Fourth edition (DSM-IV; American Psychiatric Association, 1994). ODD is denoted by the age-inappropriate and persistent display of angry, defiant, irritable, and oppositional behaviors; CD includes a far more severe list of aggressive and antisocial actions that involve the infliction of pain (e.g., initiating fights, fire setting), denial of the rights of others (e.g., stealing, breaking and entering), as well as status offenses such as running away from home (American Psychiatric Association, 1994).

The intention behind these diagnostic categories is to include youngsters whose patterns of defiance or ASB are persistent and clearly impairing. Thus, whereas most youngsters diagnosed with CD will by definition display delinquent behavior patterns, only a minority of delinquent adolescents would qualify for a diagnosis of CD, given the transitory and relatively nonimpaired nature of much delinquency during adolescence (Hinshaw, Lahey, & Hart, 1993; Moffitt, 1993). Elaboration of the diagnostic criteria for ODD and CD, and appraisal of their validity and viability, are central topics of this chapter.

Fourth, the diagnostic category for adults with the persistent display of ASB is antisocial personality disorder (ASPD), found on Axis II of the DSM-IV nosology (American Psychiatric Association, 1994). As we highlight later, conceptions of ASPD in recent decades have emphasized the repetitive display of multiple illegal behaviors, with the necessity of a history of CD before adulthood. Such a behaviorally based definition can be differentiated from an alternative conception of “psychopathy,” which emphasizes a callous, manipulative, impulsive, and remorseless psychological and interpersonal profile (Cleckley, 1976; Hart & Hare, 1997; Sutker, 1994), over and above antisocial and socially deviant actions per se. Importantly, careful empirical research supports the viability of psychopathy as a separable taxon, whereas repetitive criminality is best conceived as dimensional in nature (Harris, Rice, & Quinsey, 1994).

Fifth, in order to bring in a needed developmental perspective on the roots of psychopathic behavior and functioning, investigators have begun to identify traits among children termed “callous/unemotional” (Barry, Frick, DeShazo, & McCoy, 2000), which may be downward extensions of the affective/interpersonal factor of psychopathy noted in the preceding paragraph. The objective is to identify those psychological features (rather than oppositional or aggressive behaviors per se) that could identify those youths at the highest risk for displaying subsequent psychopathy. Recent investigations have been promising in this regard, although ultimate validation awaits prospective research into adulthood.

In sum, the various terms for depicting the domain under consideration often hamper clear communication in the field. Although our primary focus herein is on the diagnostic categories of ODD and CD in childhood and adolescence, the heated debate in the field as to the utility of

such categorical conceptions, the considerable research on dimensional approaches to aggression and ASB, and the voluminous literature on delinquency all necessitate our explicitly considering alternate frameworks in the sections that follow. Note also that our clear focus is on childhood and adolescence; we consider adult ASPD and psychopathy as potential outcomes of earlier ASB, but we do not have room to take up the extensive literature on adult psychopathic behavior and crime.

Subtypes of Aggression and ASB

Key reviews of the development of aggression (Feshbach, 1970; Parke & Slaby, 1983; Coie & Dodge, 1998) emphasize the importance of subdividing this class of behaviors into theoretically and empirically distinguishable subcategories. Brief descriptions of several dichotomized distinctions may help to convey such diversity. Although a contrasting view is that there is an overarching, underlying “antisocial trait” or propensity that subsumes most if not all of the distinctions below (e.g., Jessor & Jessor, 1977), the subtypes of aggression and ASB we discuss have received considerable external validation. In short, investigators, clinicians, and all interested parties must pay close attention to precise definitions of the behavior patterns they are studying and treating.

1. Interpersonal aggression may be *verbal* (taunting, threatening, name calling, swearing) versus *physical* (bullying, fighting, assaulting). This distinction is evidenced not only descriptively but developmentally: Physical aggression emerges rather early in development, with peak levels during the preschool years, whereas verbal aggression shows a later onset (Parke & Slaby, 1983). Thus the persistence of high levels of physical aggression into middle childhood may signal the need for clinical attention, as may the early onset of noteworthy verbal aggression during the preschool years. In addition, as development progresses, physical aggression may become *violent*, marked by assaultive behavior, injury, and (frequently) the use of weapons. Such violence is, of course, of extreme interest to scientists, clinicians, and society at large.

2. Aggression can be categorized as *instrumental* (goal-directed) versus *hostile* (Feshbach, 1970); for the latter type, the infliction of pain is characterized as the intent of the behavior. Some

levels of instrumental aggression are clearly normative for toddlers, whose cries of “mine” as they grab toys may signal a consolidating sense of self. On the other hand, extreme levels of hostile aggression demand further assessment at any age

3. Relatedly (but not identically), aggressive behavior may be *proactive* (bullying, threatening) versus *reactive* (retaliatory). In a systematic program of research, Dodge and colleagues (e.g., Dodge, 1991; Dodge, Lochman, Harnisch, & Bates, 1997) have shown that these two subtypes of aggression are marked by different kinds of social-cognitive information-processing deficits and distortions. That is, whereas children with a propensity for reactive aggression underutilize cues in reaching interpersonal decisions and show a propensity to attribute hostile intent to others in ambiguous social situations, those with a tendency toward proactive aggression tend to hold strong expectations that aggressive actions will help them obtain desired ends. Thus, in terms of the multistage social-cognitive information-processing model of Crick and Dodge (1994, 1996), reactive aggression involves “early” problems in encoding and interpretation of cues, whereas proactive aggression is associated with “late” expectancies regarding the value of aggressive behavior. Overall, despite the moderate to strong empirical associations between these forms of aggression, this distinction appears to have important theoretical and empirical underpinnings.

4. Another important distinction pertains to aggression that is *direct* (see the verbal and physical manifestations noted above) versus *indirect* or *relational* (“getting even” by having a third party retaliate; degrading another’s reputation by spreading rumors; excluding a peer from activities). Such indirect aggression may pertain to girls more than to boys (Bjorkqvist, Lagerspitz, & Kaukianinen, 1992); its consideration may illuminate (and mitigate) the often-cited sex differences in rates of aggression and ASB (see Goodman & Kohlsdorf, 1994). Indeed, a growing literature on relational aggression in girls (e.g., Crick & Grotpeter, 1995; Crick & Bigbee, 1998) highlights the impairing nature of such means of excluding or harming the reputations of others. We elaborate on relevant research subsequently, when discussing sex differences in aggression. Note that the terms “indirect” and “relational” are not interchangeable, as certain forms of socially and relationally excluding agemates may be quite direct (delivered to a peer’s face), whereas

others may be surreptitious and performed via third parties (see Coie & Dodge, 1998).

5. At a broader level of categorization, ASB can be defined as *overt* (exemplified by most of the types of physically aggressive actions noted in the preceding paragraphs) versus *covert*, clandestine, or nonaggressive, with the latter subcategory characterized by such actions as lying, stealing, destroying property, abusing substances, being truant, and firesetting. On the widely used and well-validated Child Behavior Checklist (CBCL; Achenbach, 1991), the overt-covert distinction is evidenced by separate narrow-band scales of Aggressive Behavior versus Delinquent Behavior. Although many severely antisocial youths display both types of antisocial activity, the overt-covert distinction is empirically robust (Loeber & Schmalzing, 1985), with growing evidence for divergent external correlates and causal factors. For instance, the Aggression Behavior scale of the CBCL displays substantial heritability, but the Delinquent Behavior scale (covering covert behaviors) yields lower heritability estimates (Edelbrock, Rende, Plomin, & Thompson, 1995). These two domains are also marked by somewhat different familial child-rearing styles (Patterson & Stouthamer-Loeber, 1984) and disparate developmental trajectories (Loeber, Wung, et al., 1993; Loeber & Hay, 1997). Importantly, because the DSM-IV diagnosis of CD includes an admixture of overt and covert behavioral criteria—for example, assault and forced sexual activity as well as lying, shoplifting, and truancy—the CD diagnosis incorporates, by definition, disparate subtypes of antisocial youths (Achenbach, 1993). We return to this point in our subsequent discussion of diagnostic criteria.

Overall, even at the level of description of the constituent actions, the realm of aggression/ASB is complex and variegated. Much of the literature on aggression, antisocial activity, and delinquency confounds multiple subcategories of this domain, leading to difficulty in comparing investigations from different laboratories or from different time periods and inconsistencies in reports of the correlates of or risk factors for such externalizing behavior. Given that precision in terminology is of critical importance for the field, we again highlight the careful attention that must be paid to the definitions of ASB and to the subtypes of antisocial youths in research and clinical endeavors.

Dimensions or Categories?

A key issue for the field of psychopathology in general and ASB in particular pertains to the conception of deviance as dimensional or continuous on the one hand, versus discrete or categorical on the other (see Eysenck, 1986, for a seminal discussion). For our purposes, the question may be posed as follows: Is ASB in children and adolescents best conceived as lying on a continuum, with quantitative (but not qualitative) differences between youngsters in the levels of their constituent behaviors? Or do actual categories, diagnostic entities, or taxa (e.g., ODD or CD) exist—constructs that are qualitatively distinct from other forms of psychopathology? In other words, are there cutoff points for the underlying behavioral features (or for correlates of the symptom patterns) that reflect true discontinuities in the population? This deceptively simple dichotomy between dimensional and categorical perspectives is quite pertinent to any discussion of ASB.

In the first place, current nosologies (e.g., DSM-IV) are presented largely in a Kraepelinian framework, in which distinct disorders—defined by inclusionary and exclusionary criteria—are held to be present versus absent and to be distinct from other diagnoses (see Achenbach, 1993). As argued elsewhere (Hinshaw et al., 1993), however, categorical approaches must reflect actual discontinuities in the underlying distributions of the constituent behavior patterns if they are to be viable. If such discontinuities are not found, the chief advantages of categorical approaches would be convenience or the maintenance of tradition. Although few data explicitly address this issue regarding ASB, the empirical report of Robins and McEvoy (1990) is heuristic. Here the question was whether the overt and covert symptoms of CD in childhood, assessed retrospectively by American adults who participated in the landmark Epidemiologic Catchment Area study (Robins & Regier, 1991), could predict adolescent and adult patterns of substance abuse. Specifically, would the prediction be linear, with each successive number of aggressive/antisocial symptoms incrementing the predictive power in stepwise fashion, or would it increase precipitously when a certain diagnostic threshold was reached? The prediction function was in fact entirely linear: Each successively higher number of childhood CD symptoms incremented the prediction to later substance abuse, with no evi-

dence for a “jump” in predictive power above any given cutoff. Overall, despite the limitations of this example, a conception of CD as continuous or dimensional appeared optimal.

In addition, it may well be the case that criteria other than levels of the constituent behaviors may define distinct categories. In fact, as we discuss in considerable detail, early age of onset plus the presence of ADHD symptoms, neuropsychological dysfunction, family discord, peer rejection, and academic failure appear to be joint markers of fundamentally divergent taxa of ASB: (1) an early-onset type, with youngsters manifesting clear psychopathology and showing strong evidence for considerable persistence of ASB across the life span; and (2) an adolescent-onset type, with youngsters failing to show most indicators of psychological disturbance, and demonstrating instead an age-expected tendency to violate social norms during the extended “gap” between physical and social maturity in Western societies (Moffitt, 1993). Importantly, both subgroups display similar rates of offending during adolescence (except for the higher rates of physical violence in the group with early-onset/persistent ASB), highlighting the need to transcend symptoms in forming this typology or categorization.

Dimensional and categorical approaches to psychopathology can be complementary and supplementary, rather than mutually exclusive (Achenbach, 1993; Pickles & Angold, *in press*; Rutter et al., 1998). For instance, subgroups of individuals with discrete psychopathology may emerge when cluster-analytic approaches are applied to dimensional measures of behavioral disturbance (e.g., Nagin & Tremblay, 1999). In other words, empirically derived typologies may emerge from quantitative, dimensional data, but the crucial criteria for validating such taxa must emanate from measures (e.g., risk factors, biological or environmental correlates, long-term course, treatment response) that are external to the defining symptoms themselves. Along this line, investigators who examine the predictive relations between dimensional measures of ASB on the one hand, and external correlates on the other, should carefully examine whether the associations that are found hold up at all points along the ASB continuum, especially the extreme scores that could potentially define a distinctive subgroup. Alternatively, those who study existing categories of ASB (e.g., ODD or CD) should examine whether group differences in mean levels of the dependent measures of interest might be

better predicted from the dimensionalized symptom scores than from the diagnostic groups themselves (see Fergusson & Horwood, 1995). We hasten to point out, however, that categorical definitions may yield great practical advantages. For example, clinical or placement decisions (e.g., should a child receive special education services or be placed outside the home?) are far more easily made via yes–no designations, even if those involve dichotomizing an underlying dimension. Furthermore, the often-cited statistical dictum that dimensional scores always yield more statistical power than categorical indicators may not always be the case, as ably discussed by Farrington and Loeber (2000).

A related point bears mention. Particularly within the realm of ASB, for which multiple socioeconomic, familial, peer-related, neighborhood, and societal influences are salient, it may be that classification of youths into discrete categories—especially those with a psychiatric, intra-individual orientation—may render relevant parties (e.g., clinicians, families, policy makers) insensitive to the very real social influences on these troublesome behavior patterns. That is, a child may be seen as the sole locus of the “disorder.” Indeed, because treatment decisions are quite likely to follow from conceptions regarding the source of the problem, classification of a child as psychopathological or mentally disordered could well steer clinicians or practitioners toward individual rather than systemic prevention or treatment strategies. (Note, in this regard, that recent high-quality prevention programs blend efforts directed toward individual, school, family, and wider community levels; see Conduct Problems Prevention Research Group, 2002). In sum, despite the scientific and practical benefits that may accrue to accurate classification, real dangers exist when labeling unjustly or unthinkingly ascribes the underlying problem to psychopathology or to a mental disorder (Richters & Cicchetti, 1993). On the other hand, if viable categories are found to exist, and if certain youngsters with ASB are found to evidence clear psychopathology, the resultant precision could aid in the mounting of therapeutic efforts (Moffitt, 1993; Rutter et al., 1998).

DSM-IV DEFINITIONAL CRITERIA

We begin this section with a short history of categorical psychiatric classification of externalizing

or disruptive behavior disorders, and then proceed to a review of the DSM-IV definitional criteria for ODD, CD, and ASPD. We also raise the issue of how to decide whether patterns of aberrant emotion and behavior lie properly in the domain of psychopathology or mental disorder, with particular emphasis on juvenile aggressive and antisocial patterns, which present difficult problems with respect to such decisions.

Brief Historical Overview

Children and Adolescents

Some of the earliest applications of multivariate statistical analysis to child psychopathology helped to establish the psychometric viability of aggressive and ASB patterns in children and adolescents as key dimensions (e.g., Hewitt & Jenkins, 1946). This work also began the tradition of subtyping this domain, as youngsters' social bonds and types of antisocial activities formed the basis of two discrete dimensions: (1) "undersocialized," marked by assaultive, aggressive behaviors that were typically committed alone; and (2) "socialized" or "group-delinquent," characterized by the presence of social connections and by covert as well as overt antisocial activity. This empirical distinction has continued to receive internal and external validation. Indeed, so-called "socialized" delinquency—which may be evidenced by gang membership—is typically marked by fewer indicators of psychopathological functioning and a better long-term course than the "undersocialized" variant (Quay, 1987). Accordingly, much of the psychological and psychiatric literature on aggression in childhood and adolescence has focused on youngsters who display an undersocialized pattern of ASB. Indeed, as we discuss later, the undersocialized group is similar in most respects to subgroups defined by early age of onset.

In the landmark 1980 publication of DSM-III (American Psychiatric Association, 1980), which revolutionized psychiatric nosology in the United States through its neo-Kraepelinian orientation, CD received "operational" criteria for the first time, which incorporated a number of severe overt and covert manifestations of ASB. Because only one constituent action, displayed over long time periods, was necessary for a diagnosis, inflated prevalence rates were a potential problem. DSM-III also listed four subcategories of CD, corresponding to the cells of a 2 × 2 matrix of (1)

socialized versus undersocialized and (2) aggressive versus nonaggressive dimensions. The reliability of classification into these subtypes was poor, however, largely because of the confounding of the two components (e.g., few undersocialized–nonaggressive youngsters were found).

In DSM-III-R (American Psychiatric Association, 1987), the number of symptoms required for a diagnosis of CD was increased to 3 (from a list of 13), with each needing to be displayed for at least 6 months. This raising of the threshold reflected the established finding that the diversity (rather than any particular form) of antisocial activity—committed at early ages—best predicts chronic antisocial functioning and recidivism in adolescence and adulthood (e.g., Robins, 1966; Stattin & Magnusson, 1989). Furthermore, the subtyping scheme was simplified to the following: (1) a group (or socialized) type; (2) a solitary, aggressive subcategory; and (3) an undifferentiated type with mixed features.

With regard to milder forms of ASB, DSM-III included, for the first time, a variant of CD termed "oppositional disorder." The intention behind this category was to capture early manifestations of aggression/ASB that are exhibited in early to middle childhood. The constituent symptoms were irritable, stubborn, and defiant behavioral features, displayed at rates considered deviant developmentally. Because of the ubiquity of such behavioral features in young children, however, along with marginal reliabilities in empirical investigations, considerable doubt was raised as to the viability of this category (Rey et al., 1988). The revision in DSM-III-R—with the name changed to ODD—included nine behavioral symptoms, five of which were necessary for diagnosis. In our consideration of developmental trajectories related to ASB, we consider whether ODD constitutes a valid diagnostic category (e.g., Achenbach, 1993; Loeber, Lahey, & Thomas, 1991).

Adults

Regarding adult manifestations of chronic ASB, a sizable literature has appeared over the years with respect to so-called "psychopathy" or "sociopathy," signified by a manipulative, exploitive, predatory lifestyle (see Cleckley, 1976; Hart & Hare, 1997). Psychopathy has been the subject of considerable research regarding its psychodynamic, familial, and psychobiological underpinnings, with early socialization practices as well

as the potential for impaired avoidance conditioning and diminished response to punishment implicated as key mechanisms for this disorder (see the review by Sutker, 1994).

In DSM-III (American Psychiatric Association, 1980), Axis II personality disorders were presented for the first time, with the goal of operationalizing chronic, maladaptive traits that yield substantial impairment. Reflecting the DSM's adherence to non-etiology-oriented operational criteria, the new category of ASPD borrowed heavily from the formulations of Robins (1966, 1978), who eschewed inferences of internal psychological processes and instead advocated a set of behavioral indicators of chronic antisocial functioning in adulthood. Many of the psychological and interpersonal hallmarks of psychopathy per se (e.g., callousness, manipulateness, charm, deceitfulness, superficiality) were ignored in favor of multiple indicators of a persistent antisocial lifestyle (e.g., inconsistent work behavior, lack of monogamous relationships, aggression, multiple offenses). In addition, the diagnosis of ASPD in DSM-III and DSM-III-R has mandated the presence of CD in childhood or adolescence. Thus, by definition, the display of antisocial patterns beginning early in development is considered a necessary precondition for ASPD. This requirement has continued in DSM-IV, as discussed below.

The exclusively behavioral focus of the ASPD criteria was criticized by Hare, Hart, and Harpur (1991), who contend that psychopathy comprises key psychological and interpersonal features (e.g., callousness and manipulation as well as shallow, nonempathic affect). Indeed, their position is that ASPD definitions run the risk of labeling repetitive criminality as a form of personality disorder, with pertinent psychological and interpersonal features ignored in the diagnostic criteria. In defense of their position, psychopathy per se has been found to constitute a discrete taxon, as noted above, whereas repetitive criminality in adulthood appears to fit a dimensional characterization (Harris et al., 1994). Investigation of the developmental roots of adult antisocial functioning, however characterized, is crucial.

Current Definitions of ODD and CD

The DSM-IV definitional criteria for ODD are presented in Table 3.1, and the criteria for CD are listed in Table 3.2. As can be seen, ODD requires four of eight indicators of hostile, defiant,

TABLE 3.1. DSM-IV Diagnostic Criteria for Oppositional Defiant Disorder (ODD)

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- A. A pattern of negativistic, hostile, and defiant behavior lasting at least 6 months, during which four (or more) of the following are present:
- (1) often loses temper
 - (2) often argues with adults
 - (3) often actively defies or refuses to comply with adults' requests or rules
 - (4) often deliberately annoys people
 - (5) often blames others for his or her mistakes or misbehavior
 - (6) is often touchy or easily annoyed by others
 - (7) is often angry or resentful
 - (8) is often spiteful or vindictive
- Note.** Consider a criterion met only if the behavior occurs more frequently than is typically observed in individuals of comparable age and developmental level.
- B. The disturbance in behavior causes significant impairment in social, academic, or occupational functioning.
- C. The behaviors do not occur exclusively during the course of a Psychotic or Mood Disorder.
- D. Criteria are not met for Conduct Disorder and, if the individual is age 18 years or older, criteria are not met for Antisocial Personality Disorder.
-

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negativistic, and irritable behaviors for a duration of at least 6 months, which must be present at levels considered developmentally extreme and impairing. A diagnosis of CD mandates 3 of 15 examples of more serious overt and covert antisocial behaviors, with personal and social impairment required. Thus, compared to DSM-III-R, the symptom list was decreased by one for ODD and increased by two for CD. Several themes and issues regarding the definitions of these taxa bear discussion.

Developmental Norms

As reviewed by Coie and Dodge (1998), oppositional and defiant symptoms are relatively common during the preschool years; this means that it would take an extremely high level (and severity) of such patterns, in comparison with age and sex norms, to warrant diagnosis. The typical developmental course, in fact, is for such difficul-

TABLE 3.2. DSM-IV Criteria for Conduct Disorder (CD)

A. A repetitive and persistent pattern of behavior in which the basic rights of others or major age-appropriate societal norms or rules are violated, as manifested by the presence of three (or more) of the following criteria in the past 12 months, with at least one criterion present in the past 6 months:

Aggression to people and animals

- (1) often bullies, threatens, or intimidates others
- (2) often initiates physical fights
- (3) has used a weapon that can cause serious physical harm to others (e.g., a bat, brick, broken bottle, knife, gun)
- (4) has been physically cruel to people
- (5) has been physically cruel to animals
- (6) has stolen while confronting a victim (e.g., mugging, purse snatching, extortion, armed robbery)
- (7) has forced someone into sexual activity

Destruction of property

- (8) has deliberately engaged in fire setting with the intention of causing serious damage
- (9) has deliberately destroyed others' property (other than by fire setting)

Deceitfulness or theft

- (10) has broken into someone else's house, building, or car
- (11) often lies to obtain goods or favors or to avoid obligations (i.e., "cons" others)
- (12) has stolen items of nontrivial value without confronting a victim (e.g., shoplifting, but without breaking and entering; forgery)

Serious violations of rules

- (13) often stays out at night despite parental prohibitions, beginning before age 13 years
- (14) has run away from home overnight at least twice while living in parental or parental surrogate home (or once without returning for a lengthy period)
- (15) often truant from school, beginning before age 13 years

B. The disturbance in behavior causes clinically significant impairment in social, academic, or occupational functioning.

C. If the individual is age 18 years or older, criteria are not met for Antisocial Personality Disorder.

Specify type based on age at onset:

Childhood-Onset Type: onset of at least one criterion characteristic of Conduct Disorder prior to age 10 years

Adolescent-Onset Type: absence of any criteria characteristic of Conduct Disorder prior to age 10 years

Specify severity:

Mild: few if any conduct problems in excess of those required to make the diagnosis and conduct problems cause only minor harm to others

Moderate: number of conduct problems and effect on others intermediate between "mild" and "severe"

Severe: many conduct problems in excess of those required to make the diagnosis or conduct problems cause considerable harm to others

Note. From American Psychiatric Association (1994, pp. 90–91). Copyright 1994 by the American Psychiatric Association. Reprinted by permission.

ties with stubbornness, tantrums, defiance, and the like to attenuate by middle childhood. For those children who do not display the usual age-related declines, and particularly for the subgroup who "diversify" into more frankly aggressive behavior as well, the ODD diagnostic category appears warranted.

As for CD, the types of seriously aggressive and antisocial actions in the symptom list are not normative during childhood. Preadolescents who begin to display such actions are therefore a group for whom clinical concern is deserved. Yet sharp increases in the prevalence of multiple forms of delinquent activity can be observed in early to

middle adolescence (see review in Coie & Dodge, 1998), with a particularly steep rise for girls. Furthermore, the covert actions from the CD symptom list show clear increases, even in normative samples, through adolescence (Loeber & Hay, 1997).

Viability of the Categories

A key means of appraising the validity of ODD and CD is to appraise their distinctiveness from other behavioral syndromes. As discussed earlier, ODD and CD display such divergent validity from ADHD, whether the domains are considered dimensionally or categorically (e.g., Fergusson et al., 1991; Hinshaw, 1987; Jensen et al., 1997). In brief, ADHD is frequently associated with “individual” risk factors (difficult temperament, cognitive deficits), whereas aggressive-spectrum disorders are embedded in environmental/contextual risks such as discordant family interactions, harsh or inconsistent discipline, and impoverished neighborhoods (e.g., Hinshaw, 1992; Patterson, Reid, & Dishion, 1992). Yet these behavioral patterns frequently overlap, as do some of their risk factors (Hinshaw, 1987; Jensen et al., 1997; Waschbusch, 2002), and the theoretical and empirical importance of their co-occurrence bears much closer scrutiny in the subsequent section on comorbidity. With respect to predictive validity, it is clear that diverse aggressive and antisocial activities with an early onset strongly predict persistent ASB as well as myriad adjustment difficulties (e.g., Robins, 1966; Stattin & Magnusson, 1989; Zoccolillo, Pickles, Quinton, & Rutter, 1992; Rutter et al., 1998).

Another means of validation is to examine whether clear impairment accrues to the behavioral symptomatology. That is, we may ask (1) whether the constituent behavioral features of ODD and CD yield clear evidence of dysfunction in school, at home, and in interpersonal relationships; and (2) whether any evidence exists for discontinuous levels of such impairment above the diagnostic thresholds. In regard to point 1, the disruption, pain, and even tragedy resulting from CD-like behavior patterns are clear, as violence and property destruction may take a considerable toll on individuals, families, and communities at large. Furthermore, nonaggressive aspects of CD (e.g., theft, truancy) can yield considerable harm to the self and to others as well. Indeed, youths with CD are at substantial risk for peer rejection, academic failure, and a persistent course, attest-

ing to the virulence of the syndrome (Rutter et al., 1998; Patterson et al., 1992). As for point 2, however, whereas the field trials for DSM-IV confirmed that three or more constituent symptoms of CD are associated with marked impairment, it is not clear that a true discontinuity with respect to external criteria exists at or above any given threshold of the defining behaviors (e.g., Robins & McEvoy, 1990). In short, the viability of categorical notions of CD that are based on the number of constituent symptoms is not at all assured, whereas the subdivision of this category on the basis of age of onset has more potential to yield a qualitative distinction (see subsequent discussion).

To an even greater extent, the validity of ODD as a diagnostic entity is an unresolved issue (see Loeber et al., 2000). Unlike most of the actions subsumed under the CD criteria, which involve severe manifestations of overt and covert behaviors, the constituent symptoms of ODD are clearly in the realm of normal developmental actions, particularly for children of preschool ages and again during adolescence (Coie & Dodge, 1998; Loeber et al., 2000). Furthermore, as we take pains to elaborate in the subsequent section on developmental progressions, the majority of youngsters reliably diagnosed with ODD in childhood will *not* progress to the more serious manifestations of CD. Yet, if a child’s initial diagnosis must await display of the severe list of CD symptoms, intervention efforts may be unduly delayed (Loeber et al., 1991), particularly given evidence that the pathways to serious ASB often involve high levels of opposition and defiance earlier in development (Loeber, Wung, et al., 1993). As a result, debate regarding the appropriateness of this category in formal nosologies has continued for well over a decade (Achenbach, 1993; Loeber, Keenan, Lahey, Green, & Thomas, 1993; Lahey, Loeber, Quay, Frick, & Grimm, 1997). Lahey et al. (1997) concluded that ODD might well be considered a developmental precursor to CD—in other words, that ODD is basically a less severe variant of CD—but ODD does not inevitably portend CD, and some cases of CD do not originate with ODD patterns, constraining such a classification. We note, as well, that popular-press critiques of diagnostic systems like DSM-IV have featured ODD as a prime example of the overmedicalization of normal-range behavior (e.g., Kirk & Hutchins, 1994). Resolution of this issue will not come easily.

In sum, the coherence and distinctiveness of ASB patterns have been recognized for decades in the field of child psychopathology. The divergent validity of these patterns from ADHD has clearly been established, despite considerable overlap or comorbidity in actual samples; and diverse forms of ASB emerging early in development are highly predictive of a persistent course. It is still unclear, however, whether current symptom cutoff scores yield truly discontinuous categories, and the viability of ODD in particular is hotly debated. We turn now to additional issues raised by the categories of ODD and CD.

Admixture of Overt and Covert Symptomatology

Examination of Table 3.2 reveals clearly that a combination of overt and covert features is included in the diagnostic criteria for CD. Indeed, the subheadings for the criterion list highlight the disparate symptomatology among the 15 behavioral indicators of this category. As noted by Achenbach (1993), as well as other investigators, this diverse listing—combined with the requirement of only 3 of the 15 symptoms for diagnosis—means that some youngsters with CD will have exclusively covert problems, some others will show only overt aggression, and still others will have mixed symptomatology. This state of affairs guarantees the heterogeneity of the diagnosis; indeed, samples of youngsters diagnosed with CD may well contain fundamentally disparate subgroups in terms of symptom presentation. Because of the separability and discriminant validity of overt and covert dimensions, we reiterate a point made in the first edition of this chapter (Hinshaw & Anderson, 1996) that the field would be well served by investigations that identify the explicit types of behavioral symptoms characterizing CD samples.

Social/Environmental Context

In psychiatric nosologies, the locus of deviant behavior is by definition intraindividual. As a result, the clear roles of poverty, traumatic stress, and violent communities in fostering ASB may be greatly underappreciated. To counter the overascription of all aggressive/antisocial activity to individual psychopathology, DSM-IV has incorporated the following wording regarding the diagnosis of CD (American Psychiatric Association, 1994, p. 88):

Concerns have been raised that the Conduct Disorder diagnosis may at times be misapplied to individuals in settings where patterns of undesirable behavior are sometimes viewed as protective (e.g., threatening, impoverished, high-crime) . . . the Conduct Disorder diagnosis should be applied only when the behavior in question is symptomatic of an underlying dysfunction within the individual and not simply a reaction to the immediate social context. Moreover, immigrant youth from war-ravaged countries who have a history of aggressive behaviors that may have been necessary for their survival in that context would not necessarily warrant a diagnosis of Conduct Disorder. It may be helpful for the clinician to consider the social and economic context in which the undesirable behaviors have occurred.

Although these words convey a crucial point, we hasten to add that the clinical realities of severe ASB are complex, without clear demarcations of contextual versus intraindividual locus of the behavior patterns. Who can determine, for example, that an aggressive or antisocial lifestyle fostered by exposure to violence-prone environments is purely a “reaction” to such settings, as opposed to an internalized, pervasive way of life that now threatens others? Or that some children exposed to violent neighborhoods have not also suffered from a host of individual and family risk factors as well? Or that genetic mediation could explain some (but certainly not all) of the prediction to later ASB among children who live with abusive parents? A pointed example of the ambiguity inherent in the “environmental reaction” versus “intraindividual pathology” perspectives is found in the provocative portrayal of mob boss John Gotti by Richters and Cicchetti (1993). Although Gotti developed and acted in a subculture that clearly sanctioned extremes of aggression and violence, he appeared to display severe psychopathy as well, with a long history of brutality that transcended even his prescribed and chosen environment. Deviant behavior is multidetermined and transactional, with no clear separation of cultural, environmental, or intraindividual causal factors at the level of the individual case.

Mental Dysfunction or Disorder?

Along this line, what are the criteria that should be invoked to decide that a certain individual suffers from psychopathology or a mental disorder? This issue has received close scrutiny by nosolo-

gists and critics alike. The perspective of Wakefield (1992, 1999) has been heuristic for the field. Not satisfied with the often-utilized criteria of social deviance, personal distress, psychological handicap, and the like for defining mental disorder—standards that are too prone to cultural variation and that may not reflect actual psychopathology—Wakefield has invoked the dual-criterion set of “harmful dysfunction” to characterize mental disorder *per se*. First, the deviant behavioral or emotional pattern must yield actual harm, in the form of meaningful suffering or impairment. Clearly, this criterion is admittedly context-dependent, as “harm” may be variously defined across cultures or subcultures. Second, however, the pattern must also be dysfunctional, in the sense of exemplifying aberrations in the abilities of mental mechanisms to perform natural functions, with the latter defined as having been selected by evolution for the good of the species. Through this latter criterion, Wakefield (1992, 1999) is attempting to transcend arbitrary, cultural definitions of deviance and posit underlying dysfunction in evolutionary terminology.

Few would doubt the clear harm yielded by conduct-disordered behavior patterns (and by severe manifestations of oppositional defiant patterns), at least as defined by most cultures. As noted earlier, physical and sexual assault, property destruction, fire setting, and stealing are inherently harmful, engendering understandable fear. Yet what of the dysfunction criterion? The evidence, in fact, suggests rather strongly that the majority of ASB is committed by individuals during adolescence, who lack childhood histories of cognitive dysfunction, ADHD symptoms, or serious family discord that would indicate intra-individual dysfunction (e.g., Moffitt, 1993). Only a relatively rare type of antisocial youngster—marked by early aggression and, nearly always, by severely impulsive and hyperactive behavior patterns and neuropsychological deficits early in development—may actually display the kinds of dysfunctions of mental mechanisms that would yield evidence for mental disorder. But even here, can it be unequivocally asserted that such early childhood problems (e.g., somewhat sub-average cognitive abilities, mild neuropsychological deficits, patterns of insecure attachments) are actually dysfunctional in the sense of reflecting aberrations in mental processes selected by evolutionary forces (Lilienfeld & Marino, 1999)? Or are such patterns perhaps reflective of a poor fit with current environmental contingencies, which

differ substantially from those in the environment of evolutionary adaptation? In other words, the use of naturally selected mechanisms as the key criterion for defining mental disorder leads to difficult and probably untestable scientific problems, and the lack of consensus regarding these issues casts doubt on the viability of Wakefield’s authoritative guide for deciding what is and what is not mental disorder (e.g., Richters & Hinshaw, 1999).

Despite the thorny philosophical, scientific, and evolutionary issues involved in deciding on the boundaries of mental disorder, consensus has emerged that defining subtypes of CD on the basis of age of onset (and persistence of symptomatology) may reveal fundamentally disparate types of youngsters. The importance of the age-of-onset variable mandates specific discussion of this means of subdividing the diagnosis. Indeed, the only officially recognized subcategorization of CD in DSM-IV is made on the basis of the timing of the onset of core symptomatology.

Subtypes of CD Defined by Age of Onset

The DSM-IV criteria include childhood-onset and adolescent-onset subtypes of CD, with the difference relating to the presence of at least one constituent symptom prior to the age of 10 years. The rationale for this bifurcation can be traced in large measure to the work of Moffitt (1993), Loeber (1988), and Patterson (1993; Patterson, DeBaryshe, & Ramsey, 1989), all of whom have formulated parallel conceptions of early-onset or “early-starter” models of ASB and CD.

To present a capsule perspective, we cite Moffitt’s (1993) description of the puzzling nature of the literature on ASB and delinquency—in particular, the troubling inconsistency in findings related to causal factors, correlates, underlying mechanisms, and response to intervention. Her key contention is that such confusion stems largely from the confounding of two subgroups in most cross-sectional investigations of adolescent functioning: (1) a relatively small subgroup of youngsters (predominantly boys) with onset of aggressive behavior in childhood, who are at high risk for display of a persistent course of antisocial activity that unfolds and expands with development; and (2) a far larger category of youths (including a far higher proportion of girls) for whom forays into antisocial activity begin in adolescence and are relatively time-limited. Importantly, the former group is characterized by several features

that suggest chronic psychopathology: high levels of ADHD symptoms, neuropsychological deficits, problems with academic underachievement, family members within the antisocial spectrum, discordant family interaction patterns (including histories of insecure attachment as well as overly punitive parenting practices), and a high likelihood of escalation into physically aggressive and violent actions. Because this group with early-onset ASB accounts for a disproportionate percentage of illegal antisocial acts and is quite likely to persist in ASB across the life span, Moffitt's term for this subgroup is "life-course-persistent." Note, however, that in the research of Moffitt and colleagues, this subgroup is pre-defined as having not only early onset but also persistence of ASB throughout childhood, and in some reports into adolescence (e.g., Moffitt & Caspi, 2001; Moffitt et al., 2002; Moffitt, Caspi, Dickson, Silva, & Stanton, 1996). In practice, of course, a clinician or investigator needs to make a diagnosis immediately, without the luxury of waiting for longitudinal follow-up. A key question, then, is whether all early-onset antisocial activity (plus the additional intra-individual and familial risk factors that presumably go along with such early onset) will escalate into violence and continuing ASB.

Youngsters with adolescent-onset ASB, on the other hand, do not evidence the signs of psychopathology characteristic of their peers with the early-onset type (Moffitt & Caspi, 2001). Crucially, although they display significant rates of antisocial activity during adolescence, their behavioral profiles are not nearly so likely to include violent offending. Moffitt (1993) invokes the concept of social mimicry to explain the onset of ASB in such otherwise "normal" youths. That is, because of the ever-increasing gap between biological maturity and the opportunity for full psychological and educational independence in modern society, with puberty emerging earlier but the need for higher education becoming more important than previously, many adolescents mimic the antisocial actions of early-onset youngsters in an attempt to gain prestige and desired commodities (e.g., sexual partners, money, status).

The upshot is that unless investigators and policy makers differentiate these subgroups, little progress will be made in efforts to understand, predict, and treat juvenile ASB, because youngsters with two fundamentally different types of ASB will be lumped together. Several other points are salient:

1. The specific age-of-onset criterion in DSM-IV (i.e., one CD symptom prior to the age of 10 years) is arbitrary, often difficult to discern from retrospective accounts, and of unknown validity for females with aggressive behavior patterns (see the later section on sex differences)

2. To the extent that the age-of-onset variable is valid, it may well be a more parsimonious subtyping scheme than the socialized–undersocialized and aggressive–nonaggressive designations from DSM-III, in that the clear majority of early-onset CD is both undersocialized and aggressive (Hinshaw et al., 1993). Thus the current DSM-IV subtyping algorithm may be a viable, and simpler, replacement for the prior subcategorizations.

3. Empirical data from the past few years provide a rather complex picture of the validity of these two key subgroups. Indeed, investigations suggest strongly that (a) early onset of ASB does not always portend life course persistence (i.e., a relatively high percentage of those with early-onset ASB may desist later; see Nagin & Tremblay, 1999; Rutter et al., 1998); (b) youths with adolescent-onset ASB may still display noteworthy difficulties in young adulthood (see Moffitt et al., 2002); and (c) a small subgroup of individuals do not display noteworthy ASB until young adulthood. Furthermore, the viability of the age-of-onset distinction may depend on the clinic-referred versus community nature of the samples under investigation (Lahey et al., 1998). In our subsequent discussion of developmental progressions, we take up such evidence in detail.

We note, in passing, that the 10th revision of the *International Classification of Diseases* (ICD-10; World Health Organization, 1992) has attempted reconciliation of its CD diagnosis with the DSM classification system; it includes a childhood- versus adolescent-onset subcategorization, similar to that of DSM-IV. Several additional subtypes are also listed: CD confined to the family context, unsocialized CD, socialized CD, and ODD.

Current Definition of ASPD

Although our focus herein is on child and adolescent ASB patterns, we mention briefly the DSM-IV (American Psychiatric Association, 1994) definition of ASPD. In contrast to the preceding editions of the American nosology (DSM-III and DSM-III-R; American Psychiatric Association, 1980), the DSM-IV (American Psychiatric Association, 1994) definition of ASPD is based on a

ciation, 1980, 1987), which defined ASPD almost exclusively in terms of ASB patterns (see earlier discussion), the DSM-IV definition began haltingly to integrate conceptions of psychopathy (Cleckley, 1976; Sutker, 1994) and psychopathic personality disorder (see Hare et al., 1991) with the behavioral indicators of antisocial activity (see Table 3.3). As can be seen, several indicators of psychological and interpersonal features are now displayed in the symptom list (e.g., deceitfulness, lack of remorse, impulsivity), supplementing the patterns of ASB, nonconformity, and aggression that predominated in the DSM-III and DSM-III-R conceptions of ASPD. DSM-IV marks a step toward integration of the personological/interpersonal and the more behavioral definitions that have competed in recent decades, although without the explicit separation of these two subdomains, as is done in the two-factor model of Hare and colleagues. Accordingly, this category

guarantees that there will be confounding of interpersonal and affective symptoms with those of a more behavioral/antisocial nature in the DSM-IV nosology.

PREVALENCE

Consideration of prevalence estimates for ODD and CD must immediately be qualified by several important considerations. First, definitions of these disorders have changed at a fast rate over the past two decades. Indeed, as discussed earlier, oppositional disorder was first introduced as a diagnostic category in 1980 (DSM-III), with its name changed to ODD and other changes made in 1987 (DSM-III-R), and the diagnosis further modified in 1994 (DSM-IV); the definition of CD was made significantly more stringent in DSM-III-R, with additional modifications in DSM-IV. Not only are American epidemiological data with respect to the current definitional criteria relatively sparse, given that a national-level investigation of the incidence and prevalence of child mental disorders has not been undertaken, but estimates of prevalence are highly dependent on the particular definitional criteria and particular samples that are utilized (Lahey, Miller, Gordon, & Riley, 1999). Second, given developmental progressions with and between ODD and CD (see the next section), the rates of adolescents meeting diagnostic criteria in any single cross-sectional evaluation may be misleading. Along this line, it is crucial to specify the ages of onset in samples or populations of aggressive or conduct-disordered youths. Yet, in the absence of prospective investigations of representative samples that begin at early ages, reports that rely on retrospective recall of the age of onset are bound to be suspect. Third, as highlighted in our earlier discussion, categorical definitions of aggressive and ASB patterns may reflect rather arbitrary numbers of constituent symptoms. Thus, unless considerable efforts are made to index impairment that accrues to the disruptive behavior patterns, prevalence estimates may be misguided.

Overall, estimates of the prevalence of ODD have ranged widely—from under 1% to more than 20%, with a median prevalence estimate of about 3% (Lahey, Miller, et al., 1999). Prevalence estimates of CD among children and adolescents also range widely, from less than 1% to slightly over 10% (see, e.g., Lahey, Miller, et al., 1999; Zoccolillo, 1993). The DSM-IV cites rates of

TABLE 3.3. DSM-IV Diagnostic Criteria for Antisocial Personality Disorder (ASPD)

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- A. There is a pervasive pattern of disregard for and violation of the rights of others occurring since age 15 years, as indicated by three (or more) of the following:
- (1) failure to conform to social norms with respect to lawful behaviors as indicated by repeatedly performing acts that are grounds for arrest
 - (2) deceitfulness, as indicated by repeated lying, use of aliases, or conning others for personal profit or pleasure
 - (3) impulsivity or failure to plan ahead
 - (4) irritability and aggressiveness, as indicated by repeated physical fights and assaults
 - (5) reckless disregard for safety of self or others
 - (6) consistent irresponsibility, as indicated by repeated failure to sustain consistent work behavior or honor financial obligations
 - (7) lack of remorse, as indicated by being indifferent to or rationalizing having hurt, mistreated, or stolen from another
- B. The individual is at least age 18 years.
- C. There is evidence of Conduct Disorder with onset before age 15 years.
- D. The occurrence of antisocial behavior is not exclusively during the course of Schizophrenia or a Manic Episode.
-

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6–16% for males and 2–9% for females (American Psychiatric Association, 1994), although developmental differences in prevalence are not emphasized. Indeed, varying definitional criteria and sampling methods heavily influence results. The well-executed investigation of Offord and colleagues in Canada uncovered an overall rate among children and adolescents aged 4–16 of 5.5% (8.1% for boys and 2.8% for girls), with DSM-III criteria defined by CBCL items serving as the operationalization of CD (Offord, Alder, & Boyle, 1986). Importantly, a majority of the youths diagnosed with CD had at least one additional psychiatric diagnosis, highlighting the widespread nature and the importance of comorbidity for this disorder. Substantial impairment characterizes most youngsters meeting criteria for CD, in peer-related, academic, family, and personal/psychological domains.

Key intraindividual and systemic factors appear to influence prevalence of CD. For instance, most reports find substantially lower rates in females than in males, particularly for children; yet by adolescence, the gender disparity abates markedly (Zoccolillo, 1993). Furthermore, inner-city life and its attendant insults to families and children (e.g., impoverishment) clearly increase the risk for CD (e.g., Rutter et al., 1974, 1998). In all, epidemiologists would do well to heed the advice of Costello and Angold (1993) regarding the importance of developmental perspectives on the epidemiology of disruptive behavior disorders. Clearly, CD is not a static clinical entity, as the next section details; the field must begin to incorporate notions of flux and of developmental pathways into future nosological efforts.

DEVELOPMENTAL PROGRESSIONS

Heterotypic Continuity

Students of the development of aggression and ASB must come to terms with two competing facts: (1) measures of these behaviors show considerable stability across the life span, with correlations across lengthy intervals approaching those for IQ (e.g., Olweus, 1979; Loeber, 1982; Farrington, 1992; Frick & Loney, 1999); but (2) the composition of antisocial activity changes markedly over the years (see, e.g., Cairns, Cairns, Neckerman, Ferguson, & Garipey, 1989; Coie & Dodge, 1998). How can these seemingly disparate findings be reconciled?

Increasingly, developmental psychopathologists recognize that predictability and congruence across development are not necessarily synonymous with simple consistency or similarity. That is, developmental precursors may be related in systematic and meaningful ways to subsequent outcomes, even though the topographic patterns of behavior shift markedly with development. ASB or antisocial traits may therefore show moderate to strong stability over the course of development, but the surface manifestations of the underlying propensity will shift with growth, typically in terms of the accretion of new and more virulent forms of the behavior patterns across time. Patterson (1993) uses the term “chimera” to describe this phenomenon in relation to ASB, analogizing to the mythical creatures that grow new appendages on the core underlying frame. In short, the constituent behavior patterns change with development, but appear to do so in predictable and lawful ways (Patterson, Forgatch, Yoerger, & Stoolmiller, 1998). Such so-called “heterotypic continuity” is an important concept for the topic at hand.

More specifically, in individuals with strong antisocial tendencies, the argumentative and defiant behaviors of preschool and early childhood predate physical aggression and stealing in middle and late childhood and sexual assault, substance abuse, and/or concentrated property destruction in adolescence. Extending the developmental span, infant and toddler behavioral patterns of irritability, overactivity, and fussiness may be part of the same continuum, as may the chronic criminality and interpersonal callousness (as well as patterns of spousal or partner abuse) of antisocial adults. At a statistical level, the field can explore predictability: What is the magnitude of such relationships, in terms of correlating earlier patterns with later ones, if we assume that all are manifestations of an underlying antisocial propensity? Note that correlation coefficients, the usual means of portraying stability, describe the preservation of rank order of ASB across time; they index interindividual continuity (see Cairns, 1979, for a masterful analysis of the various definitions of the construct of continuity). But such correlations do not begin to tell the whole story. First, the rank order may be preserved, when at the same time the mean levels of ASB are decreasing with development (as is usually the case with overt aggression) or, in contrast, increasing (as may be the case with covert ASB or violence through adolescence). Second, most correlation

coefficients are not corrected for measurement error in either the early or later markers of ASB; when they are, estimates of overall stability rise (see commentary in Moffitt & Caspi, 2001). Third, and crucially, there may well be individual differences in stability. In fact, the most stably aggressive persons tend to be those with the highest or the lowest baseline levels of aggression (Loeber & Hay, 1997). Omnibus correlations may mask these individual differences, which are of crucial importance for deciding which individual profiles of youthful ASB are likely to escalate or diminish (for additional information on person-centered approaches to psychopathology, see Bergman & Magnusson, 1997).

In addition, other concepts may help to illuminate developmental progressions. “Pathways”—defined as within-individual changes in the patterns of ASB—were initially explored by Loeber (1988). The first developmental pathway defined by Loeber was an Exclusive Substance Use path, involving progression from more accessible to “harder” substances, but not including aggression or covert activities other than drug use. The onset of substance use for youths following this trajectory is typically rather late, beyond childhood. Another path was a Nonaggressive (or covert) trajectory; a third was a pernicious Aggressive/Versatile pathway, involving early onset and linkages with ADHD, as well as escalation into increasing violence. It is noteworthy that all three paths were found to contribute to adolescent substance abuse, exemplifying “equifinality”—the presence of similar outcomes from disparate paths (Cicchetti & Rogosch, 1996).

Loeber, Wung, et al. (1993) have since proposed expanded versions of such pathways, with origins earlier in development. First, the Authority Conflict trajectory typically pertains to defiant, oppositional patterns that progress to more serious conflict with adults; the Overt path progresses from early fighting and overt aggression to assault; and the Covert pathway focuses on links between shoplifting and property defacement at earlier stages and more serious property crime later in development. Most pertinent to the ongoing discussion is that many antisocial youngsters do not stay in any one path, but tend to “expand” into multiple trajectories over time (see also Patterson, 1993). Furthermore, the validity and viability of pathway conceptualizations may well depend on subclassifications of youths with ASB. That is, the three-pathway model appears to be more valid for children and adolescents defined as persistent in

their ASB than for those with more transient forays into such behavior patterns (Loeber, Keenan, & Zhang, 1997). In our subsequent discussion of etiological factors, we return to the complex ways in which developmental predictability, continuity, and discontinuity may be shaped by interactive and transactional processes (for discussion, see Campbell et al., 2000; Lahey, Waldman, & McBurnett, 1999; Maughan & Rutter, 1998; Rutter et al., 1998).

At this point, we first discuss the linkage between ODD in early to middle childhood and CD in late childhood and early adolescence. Evidence regarding such predictability is crucial for validating both the diagnostic category of ODD and the concept of a childhood-onset variant of CD. Second, we note briefly extant evidence for linkages between syndromes of ASB in childhood/adolescence and adult ASPD. We highlight, in addition, evidence for the predictability of ASB patterns from extremely early precursors in infancy or toddlerhood. In light of the rather descriptive focus of this discussion, we highlight that the material presented later on (1) comorbidity with other childhood emotional and behavioral disturbance and (2) risk factors and etiological formulations is necessary for a full understanding of developmental processes.

Developmental Trajectories: Progression from ODD to CD

We have noted earlier the ongoing debate regarding the viability of ODD as a diagnostic category. In the first place, important meta-analytic findings provide some corroboration of the ODD symptom complex (Frick et al., 1993; see also Loeber et al., 1991). As shown in Figure 3.1, the overt-covert continuum, described earlier in the section on definitions of ASB, is supplemented by an orthogonal destructive-nondestructive dimension. When these two dimensions of ASB are crossed, four quadrants of constituent behaviors emerge; the region defined by overt, nondestructive behaviors corresponds quite closely to the ODD symptom pattern in DSM-IV (e.g., argumentative, stubborn, defiant, angry). In terms of separability from other forms of ASB, then, and at least from a cross-situational perspective, the ODD behavioral complex has coherence.

Next, with regard to developmental timing, the behaviors characteristic of ODD emerge 2–3 years earlier than do CD symptoms (Loeber, Green, Lahey, Christ, & Frick, 1992; Lahey et al., 1997;

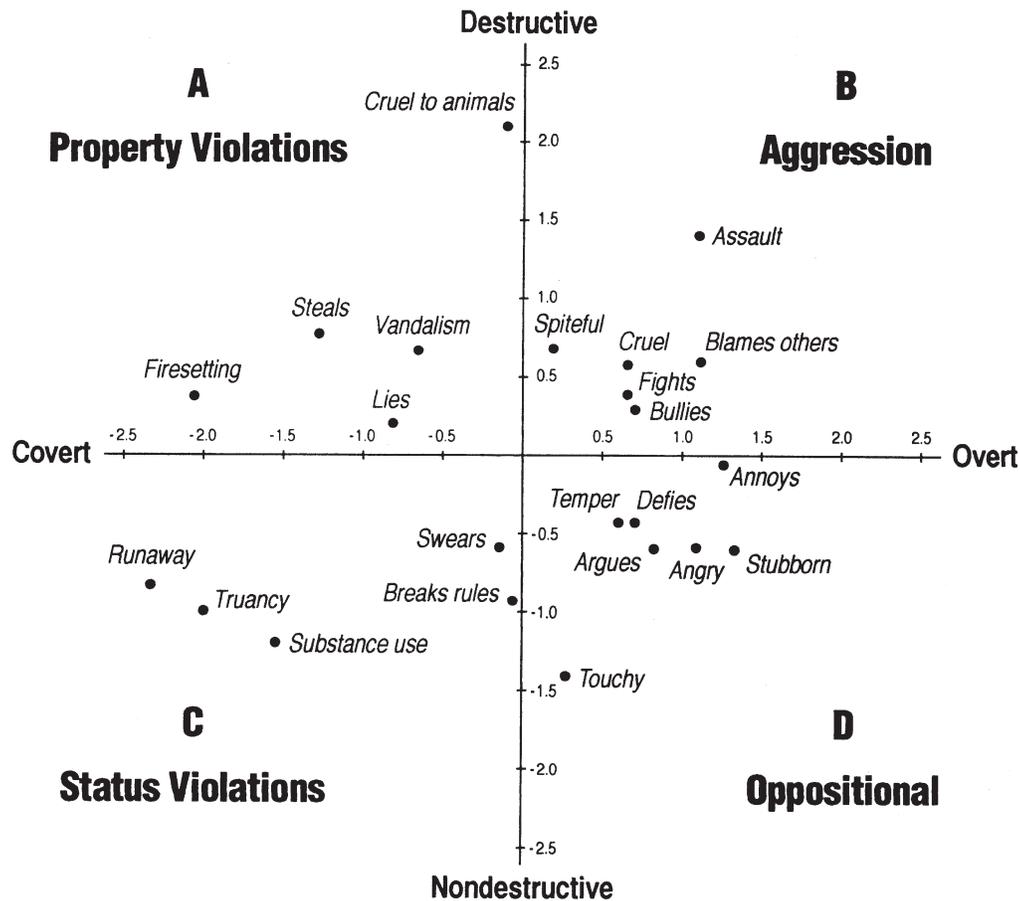


FIGURE 3.1. Results of meta-analysis of factor analyses of disruptive child behavior (see Frick et al., 1993). Copyright by Benjamin B. Lahey. Reprinted by permission.

Loeber & Farrington, 2000): The average age of onset for the former is approximately 6 years, compared to 9 years for CD behaviors. This finding provides circumstantial evidence that the ODD pattern could serve as a developmental precursor to CD (Loeber et al., 1991). Also, key risk factors (e.g., poverty, family history of antisocial activity) appear salient for the development of both ODD and CD, but the magnitude of association between such factors and these behavioral repertoires is stronger for CD than for ODD (Lahey et al., 1997; Loeber et al., 2000). The suggestion, once again, is that ODD serves as a milder (and presumably earlier) variant of CD.

All of this evidence, however, does not pertain directly to the predictive relationship between early ODD and subsequent CD. The Developmental Trends Study of Loeber, Lahey, and col-

leagues provides an important data base regarding this issue (e.g., Loeber, Keenan, et al., 1993). In this two-site sample of approximately 175 clinic-referred boys initially aged 7–12 years—recruited to reflect large proportions of disruptive and attention deficit disorders—newly developing cases of CD over the initial 3-year longitudinal interval were almost always preceded by ODD patterns earlier in development (Lahey, Loeber, Quay, Frick, & Grimm, 1992). In addition, children with the severe behavioral profile constituting CD typically “retained” many of the features of ODD that had emerged earlier in development. (Note that in DSM-IV, a diagnosis of CD supersedes a diagnosis of ODD in the diagnostic algorithm.) It therefore appears that a developmental sequence exists linking ODD in middle childhood with CD in late childhood or

early adolescence. Yet the sensitivity of a measure or construct (the proportion of cases with the prior marker) is not equivalent to “positive predictive power” (PPP; the proportion of individuals with the predictor or marker who later become cases). Indeed, despite the strong sensitivity of the prediction to CD from ODD—that is, over 90% of youths with CD have previously met (and still retain) ODD criteria—the majority of youngsters with ODD do *not* appear to progress to the more severe constellation of ASB characterizing CD. That is, false-positive predictions predominate, lowering the PPP. In the Developmental Trends Study, for example, only about a quarter of the boys with ODD had developed CD by the initial 3-year follow-up interval, whereas approximately one-half maintained this diagnosis over the 3-year period but without progressing to CD, and a final quarter desisted from diagnostic criteria for ODD (see Hinshaw et al., 1993). Overall, the clear developmental progression from ODD to CD exists only for a minority of children with ODD—and this rate may actually be smaller in community than in clinical samples. Still unresolved, however, is the question of what other forms of diagnosis or impairment may pertain to those youngsters with early ODD who do not progress to CD.

Different scientists have formed divergent conclusions regarding such findings. Loeber et al. (1991) contend that such predictive validity signifies the importance of the ODD taxon; without this category, they claim, early manifestations of later CD would be missed, depriving the child, family, and community of important early intervention strategies. Waiting for a child to display the severe symptom constellation characterizing CD may well overlook, in early childhood, the oppositional defiant patterns that serve as a sensitive predictor. Yet, with a PPP of well under 50%, it is apparent that most youngsters with ODD will not develop CD; which indicates that early identification and/or treatment may be misguided, particularly if it leads to labeling of a child as inevitably “delinquent” or “criminal.” This cautionary note would apply even more to designations of young children based on ODD symptoms.

In all, whereas CD is nearly universally preceded by ODD, in only a minority of cases does the latter symptom pattern predict the former. For a subgroup of youngsters, then, ODD appears to have heterotypic continuity with subsequent antisocial activity—signifying clear harm and possible dysfunction, in terms of the criterion

set of Wakefield (1992, 1999). In most instances, however, ODD may signify an extreme of normal developmental variation, linked with important triggering factors (e.g., family discord, extremes of temperament), but transitory in nature and not portending escalation to a “toxic” course. Also salient is the age-normative rise in prevalence of ODD in adolescence, a period often marked by authority conflict and testing of limits. Highly needed is precise specification of those risk factors that propel certain cases of ODD toward a continuing antisocial trajectory (see Loeber, Green, Keenan, & Lahey, 1995; see also Caspi & Moffitt, 1995), as well as of the protective factors that aid in desistance (for theoretical accounts of risk and resilience, see Luthar, Cicchetti, & Becker, 2000). Finally, we note in passing that nearly all of the evidence cited in this section pertains to males. For females, it is possible that different predictive relationships hold across development (see the subsequent section on sex differences).

Progression to Adult ASPD

A parallel set of findings to those presented immediately above for relationships between ODD and CD appears to hold regarding the linkage between CD in childhood or adolescence and ASPD in adulthood. That is, (1) adults with ASPD have almost always met criteria for CD earlier in their development, signifying the extremely high sensitivity of the link between CD and ASPD; but (2) only a minority of youths with CD go on to develop the chronic ASB patterns characteristic of ASPD, highlighting the rather low PPP of the predictive relationship (see the reviews by Hinshaw, 1994, and Robins, 1978). The PPP ranges from about 25% to 40% (Robins, 1966; Zoccolillo et al., 1992). Importantly, if the adult outcome is broadened to include exclusive substance use, the predictive power increases significantly (Robins, 1991).

Several additional findings supplement these descriptive statistics. First, several variables increment the predictability of adult ASPD from childhood manifestations, including early onset of diverse aggressive and antisocial behaviors and persistence of symptomatology in childhood (Robins, 1978, 1991). A host of additional factors (academic underachievement, family variables, poverty, association with deviant peers) will receive additional attention subsequently. Second, the predictive relationship with the adult disorder

appears to differ in females as opposed to males, as CD in girls is a strong predictor of later *internalizing* disorders and features as well as antisocial tendencies (Robins, 1986). Third, despite the rather low PPP statistics for predicting ASPD from CD, the clear majority of youths displaying CD will show substantial social and personal impairment in adulthood, even if full diagnostic criteria for ASPD are not met (Rutter et al., 1988; Zoccolillo et al., 1992). In other words, the lack of perfect prediction to adult ASPD should not lead to the conclusion that CD has a benign outcome in most cases. Fourth, the predictive relationships to adult antisocial personality dysfunction have been obtained almost exclusively with respect to the more behavioral conceptions of ASPD rather than to the psychological/interpersonal features of psychopathy, meaning that far less is known about the developmental roots of psychopathy per se. A growing literature has taken on the identification of the “fledgling psychopath” (Lynam, 1998), but no prospective data into adulthood are yet available on the predictive power of such childhood characteristics.

In sum, nearly all individuals meeting criteria for adult ASPD will have begun their antisocial activity earlier in development. Indeed, a requirement for the DSM-IV ASPD diagnosis is that the person must have met criteria for CD before the age of 15. Yet the predictive validity—like that for ODD-CD links—is far lower, on the order of one-third. Spanning the years from early childhood to adulthood, then, only a tenth or fewer of youngsters (calculated by multiplying the approximate rate of the ODD-CD linkage by the CD-ASPD progression rate) with ODD will progress to persistent adult manifestations of ASPD; those who do will almost inevitably have displayed the symptomatology of CD “en route.” Also, there is a dearth of literature prospectively linking CD in childhood with ASPD or psychopathy in adulthood; prospective data are critically needed to help elucidate mechanisms underlying developmental links. Finally, although Robins (1978) has contended that the emergence of serious ASB de novo in adulthood is extremely rare (and is linked when it happens with the emergence of psychosis or other extremely severe psychopathology), there may be a small but important subgroup of adults who engage in antisocial activities without noteworthy childhood precursors (see Kratzer & Hodgins, 1999). Our childhood focus in this chapter precludes discussion of this important contention.

Viability of the Childhood-Onset and Adolescent-Onset Subtypes in Light of Recent Evidence

We have discussed earlier the crucial addition, in DSM-IV, of age of onset as a subtyping variable for the CD diagnosis, and the linkages of this subcategorization to the by now substantial empirical work of Moffitt (1993; Moffitt & Caspi, 2001), Loeber (1988; Loeber, Wung, et al., 1993), and Patterson (1993; Patterson et al., 1992). At this point, sufficient longitudinal data have been accumulated to provide initial evidence regarding the viability of this classification, with particular focus on two key questions: (1) Is the childhood-onset subtype truly psychopathological and/or “life-course persistent,” and (2) is the adolescent-onset subtype truly “adolescence-limited”?

We have space for only a brief review. First, in terms of the Dunedin data base that provided the impetus for Moffitt’s (1993) formulation, Moffitt and Caspi (2001) have demonstrated that the backgrounds of the subgroup whose ASB has been defined as childhood-onset and persistent through adolescence are substantially different from those of a subgroup defined in terms of adolescent onset of ASB. Specifically, neuropsychological, neurocognitive, familial, and temperamental variables are clearly more extreme in the former than in the latter, which typically does not show deviance compared with population norms. Crucially, such findings have been shown to be robust across several different cultures and nations (see review in Moffitt & Caspi, 2001). Thus, in terms of multiple indicators of serious psychopathology, the subgroup whose ASB has been defined as both early-onset and persistent with respect to aggression (the so-called “life-course-persistent” group) appears clearly impaired, whereas the subgroup with adolescent-onset ASB is not.

Second, and crucially, there should be no automatic assumption that children (especially boys, who are overrepresented in such groups) with early onset of aggression will automatically “progress” to a life-course-persistent path. Note that the life-course-persistent subtype of Moffitt and colleagues is defined on the basis of both early *and* persisting aggression and ASB; when ASB is defined solely on the basis of early onset, a substantial proportion of children desist by adolescence (Rutter et al., 1998). Indeed, even in the Dunedin sample, a group of “desisters” (tentatively called “recoveries”) were identified by Moffitt et al. (1996), although further analyses

revealed that the recovery in these cases was far from complete (see Moffitt et al., 2002). Furthermore, the careful, typological work of Nagin and Tremblay (1999) revealed that the *majority* of boys displaying high rates of physical aggression at age 6 “desisted” across the next decade of life. Although this group’s rates of aggression at age 6 were high, the relatively small group of “persisters” (under 5% of the total population screened during kindergarten) had demonstrated even more severe levels of physical aggression during the initial assessment. It is almost certainly the case as well that looking beyond aggression *per se* in early childhood—to the constellation of neurobiological/neurocognitive, familial, and socioeconomic factors posited by Moffitt (1993; Moffitt & Caspi, 2001) to be linked specifically to a propensity toward life course persistence—will help to enhance the predictability from childhood through adolescence or adulthood. Indeed, Gorman-Smith, Tolan, Loeber, and Henry (1998) distinguished serious and chronic offenders from other subgroups of youths with ASB on the basis of multiple family problems and extreme deviance of family values and attitudes.

In addition, within several important population cohorts, a small subgroup of “low-level chronic” offenders has been noted. These individuals are defined on the basis of persistent, but low-base-rate, ASB from adolescence to adulthood (Nagin, Farrington, & Moffitt, 1995) or even from childhood onward (Fergusson, Horwood, & Nagin, 2000). The initial levels of ASB are low enough, however, that the children were not “flagged” as having early-onset ASB on the basis of severity alone.

Thus, given the presence of at least three subtypes of children (nearly exclusively boys) with early signs of ASB—“persisters,” “desisters,” and “low-level chronics,” the typology of children displaying impairing aggression at an early age appears variegated. As we discuss later in the section on sex differences, extremely few girls with early-onset ASB have emerged in existing research. In addition, recent large-scale population data suggest that there may well be more “adult-starter” individuals with ASB and criminality (Kratzer & Hodgins, 1999) than was suggested by the widely cited formulations of Robins (1978).

As for the group with adolescent-onset ASB, the recent investigation by Moffitt et al. (2002), reporting on the progress of the Dunedin male population investigation up to age 26, is essential

reading. Those participants defined as having adolescent-onset ASB (who were, it should be noted, matched with the early-onset/life-course-persistent group in terms of rates of offending during adolescence) were predicted to desist from ASB in early adulthood (see Moffitt, 1993), in keeping with their designation as adolescence-limited. However, in their mid-20s, this subgroup showed elevations (compared to the norm) on impulsivity, substance abuse and dependence, property crime, and mental health variables in addition to financial difficulties. Such findings challenge the use of the term “adolescence-limited” as applied to this subgroup. We hasten to reiterate that the subgroup with life-course-persistent ASB, defined as both early-onset and persistent through adolescence, had a far more virulent pattern of poor education, partner and child abuse, fighting and violence, psychopathic tendencies, and general life difficulties than did those with adolescent-onset ASB. Yet the current findings challenge the assumption that the life course of those with adolescent-onset ASB will be benign, given their continuing criminality (chiefly property crimes), mental health problems, poor employment histories, and substance use problems. As stated by Moffitt et al. (2002, p. 199), “Despite all this promise [referring to their nonpathological childhood histories], the [adolescence-limited] men at 26 were still in trouble” (see also Aguilar, Sroufe, Egeland, & Carlson, 2000; Kratzer & Hodgins, 1999; Rutter et al., 1998). It may be that offending in adolescence leads to “snares” and losses of opportunity that accumulate to yield serious consequences (see Caspi & Moffitt, 1995, and Moffitt, 1993, for elaboration of the sequelae of aggressive behavior that may propel a continuing course, even in the absence of early indicators of psychopathology). It is also possible, as articulated by Moffitt et al. (2002) that the poorer-than-expected outcomes of this subgroup at age 26 can be traced to the extended “maturity gap” present in modern Western societies (e.g., delays in ages of becoming parents), particularly in a nation like New Zealand with high unemployment. Only further follow-up will tell whether the participants with onset of ASB in adolescence finally desist in earnest by their early to mid-30s. In all, even without a virulent childhood history of psychopathology and early ASB, youths initiating delinquent actions in adolescence may have difficulty in suddenly desisting at the close of the teenage years.

(For additional reading on the timing of adolescent-onset delinquency in urban youths, see Tolan, Gorman-Smith, & Loeber, 2000.)

Additional Issues

Although we have been discussing the predictability of persistent ASB from childhood manifestations, it is also important to ask whether ASB can be predicted with any confidence from even earlier behavioral manifestations or risk tendencies. Although indices of difficult temperament in infancy appear to be only weakly correlated with later behavioral manifestations, and although sex differences in early temperament are not noteworthy (see the subsequent section on sex differences), Campbell and Ewing (1990) and White, Moffitt, Earls, Robins, and Silva (1990) have shown that extremes of early childhood problems can predict later hyperactive and antisocial tendencies at rates far above chance levels. Furthermore, Caspi, Henry, McGee, Moffitt, and Silva (1995) showed that temperamental features at age 3—particularly a dimension called “lack of control”—were a significant predictor of later antisocial tendencies (see also Henry, Caspi, Moffitt, & Silva, 1996; for a lucid summation of more general research on temperamental links to adult outcome, see Caspi, 2000). Although some might contend that temperament measured at age 3 is confounded with behavioral styles that are similar to the outcomes of interest, or in other words that the boundaries between “temperament” and “behavior” are fluid, the point is that predictability is an established fact (see Tremblay, Pihl, Vitaro, & Dobkin, 1994). At the level of individual cases, however, the predictability from early temperamental patterns is far from certain. As we highlight in the upcoming section on risk factors and etiological formulations, such developmental pathways typically involve multiple risks, including socioeconomic disadvantage, family adversity, victimization by abuse, achievement problems, neuropsychological deficits, and (later in development) a peer network that supports antisocial activity. Thus pathways to extremes of ASB are quite likely to include a multiplicity of interacting and transacting variables (Campbell, in press; Moffitt, 1993; Capaldi & Patterson, 1994), and there is no inevitability of high risk for ASB from early temperamental patterns.

Along this line, we encourage readers to examine three important research programs: (1) the

studies by Shaw and colleagues (e.g., Shaw, Bell, & Gilliom, 2000; Shaw, Owens, Giovannelli, & Winslow, 2001), which pertain to very young children’s risk for developing ASB; (2) the research of Campbell (e.g., Campbell, in press), which involves the investigation of 3- and 4-year-olds with severe ADHD and oppositionality; and (3) the studies by Speltz, DeKlyen, and Greenberg (e.g., Speltz, DeKlyen, & Greenberg, 1999; Speltz, Greenberg, & DeKlyen, 1990; DeKlyen & Speltz, 2001), which pertain to preschoolers with oppositional patterns. All three of these programs have examined the influences of multiple, interacting factors in relation to oppositional, aggressive, and hyperactive behavior patterns across development. With findings too numerous to recount herein, these research programs suggest strongly that the presence and interaction of intraindividual risk factors, including difficult temperament; family disharmony, incorporating insecure attachment and discordant parent–child interactions; and sociocultural risk, involving poverty and low social support, are most likely to propel the continuation, intensification, and chronicity of early aggressive behavior patterns into later childhood and adolescence.

Finally, what about the predictability of psychopathic traits and features from childhood indicators? The work of Lynam (e.g., 1998) suggests that the “fledgling psychopath” is likely to be a child (a boy, in most instances) with the constellation of ADHD symptomatology and early aggressive tendencies. Frick and colleagues, however (see Barry et al., 2000; Frick, Bodin, & Barry, 2000), contend that only the subset of such children who also display the constellation of emotional, interpersonal, and personality-related features termed “callous/unemotional” are at elevated risk for subsequent psychopathy. Wootton, Frick, Shelton, and Silverthorn (1997), for example, found that whereas most aggressive boys showed the expected associations between negative parenting practices and the severity of their ASB, the subgroup high on callous/unemotional traits actually showed *no* association between parenting practices and severity; these findings suggest that this subset of boys is relatively impervious to conditioning from parents. As noted earlier, however, only prospective follow-up of youths with such configurations of predictor variables in childhood into adulthood can confirm the predictability of psychopathic traits and functioning.

Conclusions

Although serious ASB is almost always preceded by earlier manifestations of aggression or oppositionality, only a minority of oppositional, defiant, and aggressive youths progress to diagnosable CD in adolescence. Similarly, despite the continuing interpersonal, academic, and personal adjustment problems of adolescents with CD, only a minority of youths with this disorder develop the adult manifestations of ASPD. Such information highlights the importance of identifying a subgroup of youngsters with early-onset ASB (who will also tend to display a constellation of individual, family, and neighborhood variables indicative of psychopathology), but prediction across development from such a group is plagued by overprediction to subsequent ASB. Nonetheless, adolescents and young adults with the most violent and virulent forms of ASB will have had histories from childhood; this relatively small subgroup (5% or fewer of the population) commits a highly disproportionate level of criminal acts. Despite the far larger numbers of youths with adolescent-onset ASB, and their typical absence of childhood indicators of psychopathology, recent longitudinal evidence suggests strongly that their difficulties may well extend beyond the adolescent period. Current perspectives point to the interaction of multiple risk factors in predicting extremes of antisocial activity later in life; a subgroup of children with (1) indicators of early ADHD and aggression plus (2) callous/unemotional traits may be at particular risk for psychopathic outcomes later in life. Finally, nearly all of the information presented in this section regarding developmental trajectories is available for male samples only; of crucial importance is examination of females with extremes of aggression and ASB (see the subsequent discussion of sex differences). Indeed, females with CD and delinquent tendencies appear to be at risk for a broad spectrum of internalizing as well as externalizing problems in adulthood.

COMORBIDITY

Although we have highlighted that the group of children at highest risk for persistent ASB appears to display both aggressive behavior and the symptoms of ADHD early in development, it would be wise to frame such associations in terms of the more general topic of “comorbidity.” This term

refers to a greater-than-chance rate of overlap between two or more independent disorders. Such overlap between conditions (or, dimensionally, this association between behavioral syndromes or dimensions) is receiving considerable attention in the field—in part because of evidence for widespread comorbidity across multiple childhood behavioral/emotional disorders, and in part because of the theoretical importance of such cross-domain linkages (Angold, Costello, & Erkanli, 1999; Caron & Rutter, 1991; Hinshaw et al., 1993; Jensen et al., 1997). Whereas a great deal of so-called comorbidity in child psychopathology may relate to (1) poor or ambiguous definitions of mental disorders³ or (2) conflation of what are actually developmental progressions into the overlap of two independent conditions (Caron & Rutter, 1991; Lahey et al., 1997; Rutter et al., 1998), true comorbidity challenges univariate conceptions of the genesis of disorders, and investigation of comorbidity may help to uncover relevant developmental mechanisms of psychopathology. In the interests of space, we restrict our discussion of comorbidity to ADHD, academic underachievement/learning disabilities, and key internalizing disorders. Unfortunately, space limitations dictate that we bypass the important domain of comorbidity between conduct problems/CD and substance abuse/dependence, a key concern during adolescence. We highlight that recent reviews demonstrate reciprocal effects regarding this comorbidity, with aggressive behavior fueling substance abuse and dependence, as well as the converse (e.g., White, Loeber, Stouthamer-Loeber, & Farrington, 1999). Please refer also to Chassin, Ritter, Trim, and King, Chapter 4, this volume.

Before discussing substantive issues regarding comorbidity, we note briefly that any discussions of this topic are tied in with the clinic-referred versus representative nature of the samples involved. That is, with clinical samples, rates of comorbidity are spuriously inflated (Angold et al., 1999; Berkson, 1946; Caron & Rutter, 1991). The clear implication is that community samples are a prerequisite for accurate estimates of true comorbidity.

Attention-Deficit/Hyperactivity Disorder

When ADHD is considered dimensionally, scales of its constituent symptoms correlate significantly and at least moderately with counterpart dimensions of overt and covert antisocial actions

(Hinshaw, 1987; Quay, 1986). In particular, the hyperactivity-impulsivity subdimension of ADHD is more strongly associated with aggression and ASB than is the inattention dimension. When ADHD is viewed categorically, substantial overlap between it and either ODD or CD is apparent. Such association and degrees of overlap are not complete, however, and as noted earlier, there is clear evidence for a differential pattern of external correlates of these two domains (Hinshaw, 1987; Waschbush, 2002). Thus the preponderance of evidence supports the contention that ADHD and conduct problems/aggression are partially independent aspects of child and adolescent psychopathology.

Yet the association between these two areas of externalizing behavior is quite important for consideration of developmental patterns. First, the overlapping subgroup with conduct problems and ADHD displays a far more pernicious form of psychopathology than does either single-diagnosis category. Indeed, youngsters with both CD and ADHD display greater amounts of physical aggression, a greater range and greater persistence of antisocial activity, more severe academic underachievement, and higher rates of peer rejection (see Hinshaw, 1999). In addition, they tend to have parents with not only ADHD-related symptomatology but also high rates of maternal depression, paternal ASB, and substance abuse and dependence. Not surprisingly, the parent-child interactions in such family configurations are marked by coercion and discord (Patterson, DeGarmo, & Knutson, 2000). All of these factors have been shown to be strong predictors of negative outcomes in later life. Importantly, as demonstrated by Walker, Lahey, Hynd, and Frame (1987), such greater impairment accrues specifically to the comorbidity of CD with ADHD, and not to the overlap of CD with other symptom patterns.

Second, the conjoint presence of ADHD serves to propel an earlier onset of CD symptomatology (Hinshaw et al., 1993; Loeber et al., 1995; Rutter et al., 1998). In terms of mechanisms, one key possibility is that the strongly heritable, temperamentally difficult emotional and behavior patterns associated with ADHD (e.g., irritability, impulsivity, high activity level, sensation seeking) elicit negative reactions from the environment, with aggressive behavior highly likely to result from the resultant coercion and stress (Lahey et al., 1999; Patterson et al., 2000). Whatever the mechanism or mechanisms, the

early onset of serious aggression continues to be the strongest predictor of the subsequent development of antisocial patterns in adolescence and adulthood, as emphasized repeatedly above. Indeed, Robins (1991) has shown that age at onset of CD symptomatology remains an independent predictor of APD in adulthood, even when the number and diversity of CD symptoms in childhood are controlled for statistically. Thus comorbidity of early aggression with ADHD is strongly associated with early onset of conduct problems, setting in motion a chain of interactions likely to lead to escalation and persistence.

Third, a crucial question is whether ADHD symptomatology is an independent predictor of subsequent ASB, or whether its apparent predictability is tied in chiefly with its high likelihood of association with aggression and conduct problems during childhood. The literature on this point is voluminous as well as contentious. Some of the first systematic follow-up investigations of "hyperactivity" (e.g., Satterfield, Hoppe, & Schell, 1982) contained evidence that this syndrome was a strong predictor of adolescent delinquency. Such reports did not, however, account for the potential overlap or comorbidity between ADHD-related symptomatology and aggression during childhood. With this confound accounted for, careful reviews concluded that childhood aggression and conduct problems were stronger predictors of later antisocial tendencies than were ADHD symptoms per se (Lilienfeld & Waldman, 1990). On the other hand, two key European investigations (Farrington, Loeber, & Van Kammen, 1990; Magnusson, 1987) demonstrated that dimensions of hyperactivity, impulsivity, and inattentiveness in childhood independently predicted antisocial outcomes in adulthood. In addition, with the Dunedin sample, Moffitt (1990) found that ADHD-related behavior patterns contributed independent variance to the prediction of adolescent delinquency, with early aggression partialled out. Furthermore, the American longitudinal investigation of Mannuzza et al. (1991) concluded that ADHD in childhood, in the absence of CD, still yielded a strong risk for substance abuse and antisocial disorders in young adulthood. It is conceivable, however, that this latter sample could have displayed ODD (or other forerunners of CD) in preadolescence and that the oppositional behavior patterns, rather than the attention deficits, presaged the later ASB. In fact, in a further follow-up of the Satterfield sample, it was found that that whereas child-

hood ADHD symptoms somewhat increased the later risk for delinquency, the childhood combination of ADHD and aggression yielded the strongest risk (Satterfield, Swanson, Schell, & Lee, 1994). A key recent review contends that ADHD is a risk factor largely, if not exclusively, through its association with early ODD in boys, but that ADHD could be an independent risk marker in girls (Lahey, McBurnett, & Loeber, 2000).

Whether ADHD in and of itself is a predictor, early ADHD in conjunction with early aggression is clearly a risk factor for a persistent, problem-laden course of ASB. Indeed, the bulk of recent research has pointed clearly to the conclusion that early ADHD features constitute a risk factor for such a negative course largely through their fueling of an early onset of conduct problems (see, e.g., Nagin & Tremblay, 1999; Loeber et al., 2000). Conceptually, it is unclear which aspect or aspects of ADHD symptomatology (inattention vs. overactivity, restlessness, impulsivity) constitute the risk mechanism, although impulse control problems and hyperactivity appear to be stronger candidates than inattention (Coie & Dodge, 1998). Regardless, the genetic, familial, peer-related, academic, cognitive, neuropsychological, and socioeconomic backgrounds of this comorbid subgroup set the stage for complex, interactional, and transactional models related to their high risk for later psychopathology. In terms of assessment, we note that because the externalizing behaviors are relatively difficult to disentangle during the preschool years, clinicians and investigators must use instruments that can separate ADHD from oppositional and aggressive symptoms early in development (see Hinshaw & Nigg, 1999; Hinshaw & Zupan, 1997).

Academic Underachievement/Learning Disabilities

Another important comorbid condition or associated dimension pertains to the domain of academic failure. For years, linkages between aggressive/delinquent behavior patterns and underachievement have been noted, but only in the past decade did key developmental manifestations of this important comorbidity become clarified. At the outset, we must note that many forms of academic failure and underachievement exist. Indeed, because such variables as grade retention, placement in special education, and suspension or expulsion follow rather directly from acting-out behavior patterns, we

focus our attention on academic underachievement per se.

Hinshaw (1992) has presented an integrated account of the association between ASB and academic underachievement; in the interests of space, we highlight only the key conclusions herein (see also the thorough consideration of Maguin & Loeber, 1996). First, developmental shifts in this relationship are salient. In early to middle childhood, the specific association pertains to underachievement and ADHD, as opposed to underachievement and ODD or CD. Indeed, the apparent link between aggressive-spectrum disorders and learning failure before adolescence relates to the comorbidity of such disorders and ADHD (see Frick et al., 1991, for a clear empirical demonstration). By the teenage years, however, underachievement is clearly and specifically associated with delinquency and ASB patterns, signaling a developmental shift in the causal linkages. Second, unilateral, “main effect” models that aspire to account for the relationship between externalizing behavior problems and underachievement are usually oversimplifications. Whereas in some individuals early underachievement may predict later ASB (via demoralization, frustration, and the like), and whereas in others early aggression and defiance may precipitate learning failure (via poor classroom behavior, oppositional attitudes, etc.), the association between inattentive and aggressive behavior patterns on the one hand, and risk for school failure on the other, often appears quite early in development, prior to the start of formal schooling. This state of affairs strongly suggests that underlying “third variables”—for example, language deficits, socioeconomic disadvantage, or neurodevelopmental delay—require further exploration as mechanisms underlying the subsequent comorbidity (Hinshaw, 1992; Maguin & Loeber, 1996). In other words, the association between academic failure and ASB, is marked by a complex developmental trajectory, with an array of intraindividual and familial variables pre-dating the formal comorbidity, and with ADHD-related symptomatology playing a key role in middle childhood.

Thus the effects of underachievement and ASB are likely to be reciprocally deterministic, “snowballing” across development. A child with subtle language deficits may have difficulty with the phonological processes necessary for mastery of reading; he or she may also have trouble comprehending parental requests, fueling the develop-

ment of contentious relationships with caregivers and peers. In addition, ADHD symptomatology will interact negatively with both academic readiness and behavioral regulation. Over time, the early adolescent with poor academic preparation is increasingly likely to lose motivation for schooling and to form bonds with deviant, antisocial peers, intensifying his or her own aggression and ASB patterns. Recall, however, that differential trajectories appear salient for different subgroups: For some underachieving children, ASB may follow from learning failure without childhood signs of aggression (Maughan, Gray, & Rutter, 1985), whereas in most youths the comorbid pattern is evident early in development. Overall, in adolescence, virulent ASB is all too often associated with school failure and early termination from formal education, yielding another “snare” that may compromise ultimate adjustment. Appraising the academic aptitudes and skills of youths on the trajectory toward ASB is important clinically as well as conceptually.

Internalizing Disorders

Although internalizing conditions like anxiety disorders and depression may appear at first glance to be diametric opposites of such prototypically externalizing difficulties as ODD and CD, dimensional and categorical investigations reveal substantially above-chance rates of overlap for these two domains (see reviews of Rutter et al., 1998; Loeber & Keenan, 1994; and Zoccolillo, 1992). In the interests of space, we summarize recent directions.

First, with respect to anxiety disorders, contradictory findings have been evident regarding their linkages with ASB. As reviewed in the first edition of this chapter (Hinshaw & Anderson, 1996), data from the Developmental Trends Study revealed a puzzling finding—namely, that whereas the comorbidity of conduct problems with anxiety disorders appeared to predict a less intense and assaultive type of CD during initial assessments, over time the comorbid subgroup appeared to become more aggressive than youths with CD but without anxiety disorders (see also Hinshaw et al., 1993). Other investigations have been puzzling as well: In some, anxiety disorders appeared to serve as a protective factor with respect to outcomes for children with externalizing disorders, whereas in others, anxiety-related problems appeared to heighten the risk (see review in Rutter et al., 1998). Some resolution may be found in the im-

portant distinction within anxiety-related symptomatology between inhibition and fear on the one hand, and social withdrawal on the other. The former appears to be a protective factor with regard to both the presence of assaultive behavior and the intensification of ASB, whereas the presence of social isolation and withdrawal predicts severity of aggression as well as a worse course. This area of research promises to be an important one, given the linkages between anxiety and such crucial (and heterogeneous) constructs as behavioral inhibition (Nigg, 2000).

Second, the other major type of internalizing problem encompasses depressive symptoms and syndromes. Importantly, CD and depressive syndromes display comorbidity at significant levels (see Kovacs, Paulauskas, Gatsonis, & Richards, 1988; Loeber & Keenan, 1994; for a meta-analytic review, see Angold et al., 1999). At present, it is indeterminate whether (1) major depression precipitates acting-out behavior; (2) CD and its associated impairment lead to demoralization and dysphoria; or (3) similar “third variables” or underlying causal factors—psychological, familial, or psychobiological—trigger the joint display of such symptomatology (for empirical data related to this issue, see Patterson et al., 1992). It is likely that each of these causal scenarios applies to certain subgroups. The comorbidity between these domains is important theoretically. Depression is believed to emanate from loss events, with aggressive impulses introjected and displayed against the self. Thus the dynamic boundary between self-directed and other-directed aggression may be a narrow one. This relationship is also demonstrated by the increased likelihood of suicidal behavior among youths with features of both CD and depression (e.g., Shaffi, Carrigan, Whittinghill, & Derrick, 1985). Indeed, adolescent aggression alone is a significant risk factor for suicidal behavior (Cairns, Peterson, & Neckerman, 1988; Loeber & Farrington, 2000). Psychobiologically, decreased serotonergic activity is associated with both (1) dimensions of impulsivity and aggression and (2) suicidal behavior (Brown & van Praag, 1991; see review in Lahey, Hart, Pliszka, Applegate, & McBurnett, 1993). In all, research strategies that focus on groups with CD, depression, and both disorders will be necessary to uncover important relationships between these domains (for exemplary research, see Capaldi, 1991).

Finally, in terms of a broader construct of internalizing symptoms (depression, shyness/withdrawal, and anxiety), Loeber, Stouthamer-Loeber,

and White (1999) showed that pathways to persistent substance use and delinquency during adolescence were predicted by oppositionality during early childhood, followed by persistent internalizing problems in middle to late childhood. Such prospective investigations reveal fascinating linkages between externalizing and internalizing symptoms and syndromes, and indicate the complex ways in which comorbidity with internalizing problems may influence the serious problems of conjoint delinquency and substance abuse in adolescence.

Summary

It is an exception for child disorders to occur in isolation; indeed, comorbidity is the typical state of affairs for clinical samples and even for representative samples. Although it is essential to differentiate artifactual from “true” comorbidity, the latter clearly exists in child psychopathology. For youth with ODD or CD, overlap with ADHD occurs in approximately 50% of cases. Such comorbidity is clearly associated with an early onset of aggression and with substantial impairment in personal, interpersonal, and family domains. In particular, the impulse control problems pertinent to youngsters with joint attention deficits and aggression appear to propel a negative course. Next, academic underachievement often appears in youngsters with early-onset conduct problems and in delinquent adolescents; in childhood, learning failure is linked specifically with ADHD, but over time ASB and underachievement become more clearly associated, with reciprocally deterministic modes of interplay likely. Understanding the unfolding transactions between and among cognitive, academic, and behavioral factors in aggression and CD will be important clinically and conceptually. Finally, internalizing disorders (anxiety disorders, depression) also appear alongside aggression and ASB at above-chance rates. Subtypes of anxiety-related features may show different types of association with aggressive behavior patterns, with fearful inhibition serving as a protective factor but social withdrawal acting as an intensifier. Importantly, understanding of comorbidity with depression may also help to uncover causal pathways to suicidality as well as to certain forms of violence. In sum, all relevant research must include information on the phenomenon of comorbidity; without it, mistaken attributions regarding the etiology of ODD and CD are likely to be made.

RISK FACTORS AND ETIOLOGICAL FORMULATIONS

Any comprehensive attempt to account for the etiology of disruptive behavior disorders and ASB would easily encompass book length (for an exemplary work, see Rutter et al., 1998; see also the edited volumes of Quay & Hogan, 1999, and Stoff et al., 1997). We cannot do justice to the huge literature on risk and causal factors herein but instead attempt to provide a heuristic (though far from complete) guide to intraindividual, familial, cognitive, peer-related, and wider community influences. Four key points bear mention at the outset. First, as before, we do not feature discussion of influences that shape species-wide aggressive responding, such as crowding, perception of threat, and the like (for a lucid account of such factors, see Coie & Dodge, 1998). Rather, our focus is on those individual, social, and community factors that relate to individual differences in the display and development of clinically significant aggression and ASB. Second, as perceptively discussed by Kraemer et al. (1997), risk factors—those variables associated with a higher-than-expected rate of an outcome of interest—range from variables that show statistical correlations with such outcomes (whether or not they are independent of confounds or third variables) to those that display independent contribution and those that may in fact be implicated in the causal chain. Given the near-impossibility of performing experimental research on most risk or causal influences regarding aggressive behavior disorders, the attribution of causal or etiological influence to a variable (or set of variables) known to yield risk for ASB is an effort made at the peril of the investigator, clinician, or consumer. Nonetheless, although we begin this section with a table of empirically established risk and etiological influences on aggression and ASB, we wish to move this section beyond a mere listing of the many known risk factors to an account of possible causal influences.

Third, we point out that the act of subdividing this section into subsections on different classes of risk and causal factors belies the actual state of affairs in the field—namely, that causal mechanisms are multifaceted and transactional. Indeed, as emphasized throughout this chapter, combinations of risk factors, interacting and transacting in chain-like fashion, are crucial for the development of persistent aggression and ASB (Caspi & Moffitt, 1995; Patterson et al., 1998;

Rutter et al., 1998). Given the ascendancy of models that integrate psychobiological, personological, familial, neighborhood, and socioeconomic causal factors, and that attempt explanation in terms of moderation and mediation (e.g., Campbell, in press; Capaldi & Patterson, 1994; Hinshaw & Park, 1999; Rutter et al., 1998), our subdivision should not be read as an attempt to compartmentalize risk and etiological influences into neatly demarcated domains or to posit univariate, “main effect” models. Although it should go without saying at this point in the history of relevant research endeavors, we feel compelled to say it again: Biology influences behavior at the same time that behavior influences biological mechanisms; persons both shape and are shaped by the environment (e.g., Coie & Dodge, 1998; Lahey et al., 1993; Richters & Cicchetti, 1993; Rutter et al., 1998).

Fourth, the organization of this section into separate subsections related to various classes of causal factors betrays a “variable-centered” approach to the problem at hand. That is, we examine whether certain variables of interest, singly or in combination, predict dimensions of ASB or differentiate diagnostic categories of interest (e.g., CD vs. other disorders). From a different perspective (see Bergman & Magnusson, 1997), we could be distinguishing homogeneous subgroups of children or adolescents who display similar background characteristics or trajectories. Such “person-centered” research has the potential for painting a clearer picture, as variable-centered models typically assume that a risk or causal factor operates homogeneously across subgroups of the population (see, e.g., Greenberg, Speltz, DeKlyen, & Jones, 2001). In fact, however, recent research efforts in the field are taking important steps toward an integration of variable- and person-centered approaches (see, e.g., the subtype models of Moffitt, 1993, and Loeber, Wung, et al., 1993; see also Nagin & Tremblay, 2001, who utilize a person-centered approach to defining different trajectories of antisocial actions across development and then attempt to discern intraindividual and family factors that can distinguish these subgroups). As summarized by Hinshaw and Park (1999), the field will be best served at present by research strategies that bridge the strengths and delimit the shortcomings of each approach.

We open this section with a reproduction of a table of empirically validated risk factors for childhood aggression and delinquency from Loeber

and Farrington (2000); see Table 3.4. Even though this table is far from exhaustive, note the sheer numbers of risk factors involved, as well as the multiple levels at which they occur—from intraindividual to family, school, peer, and neighborhood. No such listing, however, can give any kind of explanatory story regarding how such factors may fit together in a coherent fashion, or can describe which variable may be relevant for which particular types of aggressive behavior or which subcategories of children with ASB. In addition, such a listing does not distinguish those factors that may directly influence aggression and violence from those that are indirect risks, mediated by other factors or combinations of factors. Following our truncated accounting of several classes of risk and etiological factors, we attempt an integrated model (see subsequent section).

Intraindividual Factors

Genetic influences

Are aggression and ASB heritable? Although “crime genes” do not, of course, exist (Raine, 1993; Rutter et al., 1998), any discussion of genetic influences on this domain of behavior is laden with ethical and policy implications. We believe that further knowledge of genetic effects can be used to empower and enhance treatment rather than discriminate and stigmatize, but only if the relationship between genes and behavior becomes more thoroughly known (e.g., Plomin & Crabbe, 2000). For instance, it must be remembered that “heritability” refers to genetic influences on individual differences across a population of interest and that even for conditions or traits that are strongly heritable, environmental influences are quite salient at the level of the individual (Hinshaw, 1999). Furthermore, estimates of genetic (and environmental) influences on any behavior patterns will be influenced significantly by the types of twin or adoptive samples under investigation. As such, extant investigations in the field, particularly those of adoptees, are highly likely to underestimate environmental influences because of the restricted range of adoptive families (for a lucid discussion, see Rutter et al., 1998).

In the first place, genetic effects differ for different classes of ASB. Across the externalizing spectrum in childhood, heritability is strongest (and of considerable magnitude) for ADHD symptomatology, of moderate strength for overt

TABLE 3.4. Childhood Risk Factors for Child Delinquency and Later Serious and Violent Juvenile Offending

<u>Child factors</u>	Single parenthood
Difficult temperament	Large family
Hyperactivity (but only when co-occurring with conduct disorder)	High turnover of caretakers
Impulsivity	Low SES of family
Substance use	Unemployed parent
Aggression	Poorly educated mother
Early-onset disruptive behavior	Family members' carelessness in allowing children access to weapons, especially guns
Withdrawn behavior	
Low intelligence	<u>School factors</u>
Lead toxicity	Poor academic performance
	Old for grade
<u>Family factors</u>	Weak bonding to school
Parental antisocial or delinquent behavior	Low educational aspirations
Parental substance abuse	Low school motivation
	Poorly organized and functioning schools
<u>Parents' poor child-rearing practices</u>	<u>Peer factors</u>
Poor supervision	Associations with deviant/delinquent siblings/peers
Physical punishment	Rejection by peers
Poor communication	
Poor parent-child relations	<u>Neighborhood factors</u>
Parental neglect	Neighborhood disadvantage and poverty
Maternal depression	Disorganized neighborhoods
Mother's smoking during pregnancy	Availability of weapons
Teenage motherhood	Media portrayal of violence
Parents disagree on child discipline	

Note. From Loeber and Farrington (2000, p. 749). Copyright 2000 by Cambridge University Press. Reprinted by permission.

ASB, but relatively small for covert forms of ASB (e.g., Edelbrock et al., 1995). Indeed, for the latter, shared environmental influences are considerable. It is noteworthy as well that the comorbidity of ASB with ADHD symptomatology is itself quite heritable (Silberg et al., 1996), although recent evidence points to large contributions from shared environmental factors to covariation between ADHD, ODD, and CD (Burt, Truger, McGue & Iacono, 2001). In addition, and provocatively, recent evidence suggests stronger heritability for childhood-onset than for adolescent-onset forms of aggression and ASB (Taylor, Iacono, & McGue, 2000), again bespeaking the need to subtype accurately the domains under consideration. Also, stronger heritabilities have emerged by adulthood for property crimes than for violent crimes (Rutter et al., 1998).

Second, the heritability of ASB appears to increase with development (Jacobson, Prescott, & Kendler, 2002). That is, genetic contributions to children's aggression are relatively small, but these influences appear to increase with age, demonstrating the dynamic influences of genes on behavior and belying the notion that genetic

effects are static and immutable (Jacobson et al., 2002).

Third, and crucially, the heritability of violence per se is not strong (Rutter et al., 1998). Thus society-wide cultural and legal norms (e.g., access to guns in the United States vs. other nations) and exclusively psychosocial socialization processes (e.g., Athens, 1997; Rhodes, 1999) appear to exert considerable influence on rates of violence. Along this line, it must be remembered that that Moffitt's (1993) and Patterson's (Patterson et al., 1992) models of life-course-persistent ASB, although starting with (presumably heritable) temperamental, neuropsychological, and neurophysiological influences early in development, are transactional in nature; they posit that the child's early biologically based difficulties will both evoke and be influenced by aberrant family socialization, peer rejection, and academic failure. Indeed, gene-environment interactions and correlations are undoubtedly the rule with regard to the development of severe ASB (Lahey, Waldman, & McBurnett, 1999; Rutter et al., 1998). Genes appear to exert their influence on temperamental irritability, lack of inhibition (i.e., high impulsivity), or sensation seeking; these ten-

dencies in turn (1) are likely to be met by similar tendencies in biological parents, and (2) serve to elicit maladaptive parent–child interaction patterns. Furthermore, (3) children with biological parents with severe psychopathology may be more “susceptible” to punitive or inconsistent relations. In all, genetic influences are therefore likely to be indirect in relation to the development of ASB (Lahey, Waldman, & McBurnett, 1999); it is impossible to consider one category of causal influence (e.g., genes) without invoking others (e.g., parenting). For essential, current thinking on the need to transcend simple genetic determinism with respect to behavior, see Gottlieb (1995, 1998) and Maccoby (2000).

Psychobiological Influences

The first edition of this chapter highlighted the review of Lahey et al. (1993) regarding psychobiological influences on aggression and ASB. We cannot recapitulate such evidence herein, and we point out that interest in this area has continued (e.g., Lahey, McBurnett, Loeber, & Hart, 1995; Raine, 1993; Raine & Liu, 1998; see in particular the section on “Biology of Antisocial Behavior” in Stoff et al., 1997, containing nine excellent chapters). The area is far too complex for a reasonable synthesis herein. We feature brief highlights, but we point out that nearly all psychobiologically oriented investigators of ASB have commented on the need to incorporate biosocial or integrated environmental–biological accounts (see Coie & Dodge, 1998; Raine, 1997). Indeed, we note the contention of Lahey et al. (1993) that investigation of biological variables—whether they be neurotransmitter systems, skin conductance, event-related potentials, or hormonal influences—does not rule out important roles for psychosocial factors in the genesis or maintenance of antisocial behavior: Lahey et al. state “that a socioenvironmental event (e.g., abnormal infant experience) could be one of the *causes* of aggression, but that the effect of this experience on aggression is *mediated* by alterations in neurotransmitter activity” (1993, p. 142; emphasis in original). “Either–or” characterizations of biology versus environment, attachment versus temperament, and the like, will not facilitate progress in the field.

A key theoretical milestone in the field was the integration and synthesis by Quay (1993) of the complex neurobiological and neuroanatomical work of Jeffrey Gray, who posited a behavioral

activation (or reward) system, a behavioral inhibition system, and a generalized arousal (fight–flight) system, with each comprising distinct neuroanatomical regions and neurotransmitter pathways. Several lines of research have indicated that youths diagnosed with CD who display (1) early onset, (2) aggressive features, and (3) “undersocialized” symptom patterns demonstrate low psychophysiological or cortical arousal and low autonomic reactivity on a variety of relevant indices. Such findings are strongly reminiscent of findings with adult samples composed of psychopathic individuals (see Fowles & Missel, 1994). Note that the opposite pattern is found in youths with nonaggressive, socialized, and/or late-onset CD, who often display arousal and reactivity responses that are elevated above those not only of the aforementioned CD subgroups, but of normal comparison samples as well (see McBurnett & Lahey, 1994; Lahey et al., 1995). The assumption is that the low arousal and low reactivity associated with early-onset CD reduce the potential for avoidance conditioning to socialization stimuli, fueling poor response to punishment. Related findings have been interpreted in terms of an imbalance favoring the behavioral activation/reward system over the behavioral inhibition system in youths with undersocialized, aggressive CD (Quay, 1993; see also Kruesi et al., 1990).

Research on hormonal influences is less conclusive, with inconsistent findings pertinent to testosterone levels (see Coie & Dodge, 1998), but with recent evidence that low cortisol levels may characterize boys at risk for persistent ASB (McBurnett, Lahey, Rathouz, & Loeber, 1999). This latter finding is based on a prospective investigation—a crucial methodological issue when the goal is to ascribe causal status to biological variables with respect to the development or maintenance of ASB. As for other biological influences, several reports have demonstrated a linkage between perinatal factors and later propensity for ASB (e.g., Brennan, Mednick, & Raine, 1997). Of crucial importance for these variables are patterns of interaction with early parental rejection (see the subsection on interactional and transactional processes). As for prenatal exposure to teratogenic substances, several reports have linked maternal smoking during pregnancy with the offspring’s subsequent risk for ASB (e.g., Brennan, Grekin, & Mednick, 1999; Wakschlag et al., 1997). Such effects appear to hold up when related variables (e.g., low maternal age, socioeconomic status [SES]) are con-

trolled for. Furthermore, Arseneault, Tremblay, Boulerice, Seguin, and Saucier (2000) recently documented that the total number of minor physical anomalies (particularly those around the mouth area, appraised in adolescence) was associated with violent delinquency, even when childhood rates of physical aggression and an index of family adversity were partialled out. Whether this finding reflects neurological injury, the likelihood of early feeding problems, and/or a risk for difficult socialization requires prospective investigation from infancy onward. On the other hand, Hodgins, Kratzer, and McNeil (2001) found that inadequate parenting, rather than early obstetrical complications, was the stronger predictor of later criminal behavior.

Overall, the range of potential psychobiological influences on ASB is wide, and replication of predictive findings is crucial. Research on female samples is urgently needed in the field. Interactions of neurophysiological and neuropsychological risk with pathogenic environmental circumstances are strongly implicated in the genesis and maintenance of ASB patterns (see Raine, Brennan, & Mednick, 1997; see also the subsection on interactive and transactional effects, below).

Familial Factors

Parental Psychopathology

At the outset, we alert the reader to the obvious but often overlooked point that in biological families, familial influences on child development may be psychological in nature, may be genetically mediated, or may result from correlated (or interacting) joint influences of genes and environment. Given our prior discussion of the heritability of ASB—and the arbitrary designation of our placement of genetic influences in the subsection on intraindividual as opposed to familial influences, highlighting the artificiality of the classes of risk and causal factors herein and elsewhere—we briefly take up the kinds of family configurations in which individuals with ASB are raised and then discuss more explicitly family socialization and interaction, noting once again the strong potential for gene–environment correlation and interaction.

Intergenerational linkages with respect to criminal behavior have been demonstrated for some time, with mounting evidence that certain types of parental psychopathology are associated with child aggression and ASB. Parental ASPD

is strongly and specifically related to child CD (Faraone, Biederman, Keenan, & Tsuang, 1991; Lahey, Piacentini, et al., 1988). This association is particularly clear for fathers (Frick et al., 1992), as are the links of (1) paternal substance use disorders and (2) maternal histrionic personality configurations with child ASB patterns (Lahey, Piacentini, et al., 1988). Intriguingly, children's aggression is also associated with their parents' childhood aggression when they were the same age as the children (Huesmann, Eron, Lefkowitz, & Walder, 1984). Maternal depression has also been implicated in linkages to child aggression, with an association found in some investigations but not in others. One explanation for such inconsistency is that maternal depression is a non-specific risk factor for child maladjustment, predicting a wide range of psychopathology (Downey & Coyne, 1990; Goodman & Gotlib, 1999).⁴

Family Structure

ASB in children is associated with single-parent status, with family dissolution (particularly parental divorce), with large family size (i.e., large number of children), and with young age of mothers. Each of these seemingly straightforward risk factors actually signals a complex story. First, all of these family structural features are associated with poverty, which is itself a risk factor for ASB (see the subsequent subsection on wider contextual influences). The effects of poverty, however, as well as those of these structural variables, appear to be indirect in terms of their influence on ASB—mediated largely if not exclusively by parenting practices and parent–child interactions, which we discuss in the next subsection (Capaldi & Patterson, 1994; Coie & Dodge, 1998; McLoyd, 1990; Rutter et al., 1998). Second, we take up each family structural variable in turn:

1. Single-parent status is associated with a host of strains and stresses on parents (the modal case applies to single mothers); these strains negatively affect a mother's ability to provide authoritative parenting.

2. Death of a parent is not typically a risk factor for ADHD, whereas family divorce is, particularly for boys (Rutter et al., 1998). Yet careful longitudinal work reveals that the marital conflict and the discordant parent–child interactions often both precede and postdate the divorce; moreover, pre-existing behavior patterns in the child, rather than the dissolution per se, are the risk factors for

the offspring's aggression and antisocial activities (Amato & Keith, 1991). Furthermore, Lahey, Hartdagen, et al. (1988) discovered that the effects of divorce on boys' conduct problems were reduced dramatically when diagnoses of ASPD in the parents were statistically controlled for.

3. Large family size is associated with poverty; it appears to exert its etiological influence on ASB through poor parental monitoring of the index child or adolescent and/or modeling of aggressive actions by older siblings.

4. Young age of motherhood, especially teenage parenthood, is clearly associated with ASB in the offspring (Rutter et al., 1998). Genetic mediation could play a role here—the same sorts of impulsive tendencies in the mother leading to early pregnancy could be passed on to the children—as could “assortative mating,” the tendency for antisocial girls/young women to procreate with similarly antisocial males. Furthermore, in an important investigation, Wakschlag et al. (2000) found that young maternal age was confounded with maternal history of problem behavior in predicting the ASB of the offspring. Thus the compromised parenting skills of teenage mothers are embedded in a wider historical net of influences (see Jaffe, Moffitt, Caspi, Belsky, & Silva, 2001).

In all, complex causal chains appear to be the rule in relation to the effects of family structure on ASB.

Family Functioning and Parent–Child Interaction

Several features of parent–child interaction display moderate to strong relationships with children's aggression and ASB (for an earlier synthesis, see Loeber & Stouthamer-Loeber, 1986): (1) low levels of parental involvement in children's activities, (2) poor supervision of offspring, and (3) harsh and inconsistent discipline practices. The most comprehensive model in the field is the coercion theory of Patterson (1982; Patterson et al., 1992), which is supported by microanalyses of in-home observations of family interaction.⁵ What emerges is a pattern of harsh, ultimately unsuccessful interchanges between parents and child, leading to the development and intensification of ASB. In brief, by backing down from requests and adhering to the child's escalating demands, parents negatively reinforce the child's increasingly defiant and aggressive behavior pat-

terns; similarly, harsh and abusive discipline practices, displayed when the child escalates to severe misbehavior, are rewarded by the child's temporary capitulation (see Patterson, 1982; see also the cogent summary in Coie & Dodge, 1998). Such mutual training in aversive responding fuels both aggressive child behavior and greater levels of harsh, nonresponsive parenting. (Note that in Wahler's alternative formulation, the child's misbehavior serves to reduce the uncertainty associated with inconsistent parental responses.) Aversive interchanges serve to intensify aggressive behavior outside the home and to precipitate a widening array of negative consequences for the child and family, including risk for academic underachievement and peer rejection by the child, depressed mood in family members, and a strong likelihood of persisting ASB (Patterson et al., 1992). Thus discordant parent–child interactions propel in motion a cascade of additional risk factors and impairments associated with ASB.

Research on family socialization related to aggression increasingly recognizes bidirectional influences, in which child behavior influences parent behavior as well as the converse (Lytton, 1990). It is conceivable, in fact, that negative parenting is largely a reaction to the difficult, oppositional, and aggressive behaviors displayed by the child with developing CD. Anderson, Lytton, and Romney (1986) performed an intriguing experimental study involving mothers of boys with CD and comparison boys, in which each mother interacted with (1) her own son, (2) an unrelated boy with a diagnosis of CD, and (3) an unrelated comparison boy. Mothers in both groups displayed more negativity toward and made more requests of the youngsters with CD, strongly supporting child-to-parent effects in eliciting coercive interchange. Importantly, however, mothers of the youngsters with CD responded with the most negativity to their own boys, suggesting that a history of negative interactions plays an important role. Indeed, research with clinical samples demonstrates that maternal negativity during parent–child interactions predicts the independently observed noncompliance and covert ASB of children with ADHD, over and above the effects of the children's negativity during the interaction and maternal indices of psychopathology (Anderson, Hinshaw, & Simmel, 1994). Understanding the “ultimate” cause (parent- vs. child-related) of the escalating behavior patterns is probably futile; reciprocal determinism is likely to paint the most accurate picture. It is also clear

that the negative interchanges in at-risk families begin quite early in children's development (Campbell, in press; Shaw et al., 2001), leading Tremblay (2000) to conclude that subsequent investigations of risk for ASB must begin with recruitment of families during pregnancy! Finally, we reiterate that interactional and transactional models and those that consider subtypes of aggressive behavior are the rule rather than the exception with respect to parenting practices and parent-child interactions (e.g., Coon, Carey, Corley, & Fulker, 1992; Coie & Dodge, 1998). For example, O'Connor, Deater-Deckard, Fulker, Rutter, and Plomin (1998) uncovered evidence for gene-environment correlations regarding linkages between coercive parenting and ASB. In addition, as noted earlier, Gorman-Smith et al. (1998) found that multiple family problems, including severely deviant parental attitudes and patterns of interchange that could be considered neglectful, characterized only a subgroup of their inner-city sample defined as serious, chronic offenders. Once again, transactional models and specificity of effects are essential to consider.

The most conclusive evidence for the causal role of parenting practices in promoting ASB would emanate from experimental investigations with interventions designed to reduce coercive interchange. In fact, Dishion, Patterson, and Kavanagh (1992) demonstrated that in families randomly assigned to receive intensive behavioral intervention, the risk for child ASB was markedly reduced, with indices of parenting skill following treatment serving as predictors of teacher-reported ASB patterns. Similarly, in a large sample of children with ADHD, many of whom displayed comorbid ODD or CD, Hinshaw et al. (2000) discovered that reduction of negative and ineffective discipline practices mediated the effects of combined medication plus behavioral intervention on children's social skill and disruptive behavior at school. Thus, despite the potential for genetic mediation or for bidirectional effects, ineffective parenting practices appear to play a causal role in the genesis of ASB.

Are there variables that moderate the relation between parenting practices and ASB? First, as noted above, Wootton et al. (1997) found that aggressive boys scoring high on callous/unemotional behavioral traits did not show the expected association between negative parenting and rates of aggressive/externalizing behavior, suggesting that males with this "prepsychopathic" personality configuration were less responsive to paren-

tal socialization influences (and presumably that their proclivity toward ASB was more biologically mediated). Second, Deater-Deckard and Dodge (1997) discovered that whereas European American children assessed during the preschool years showed the expected correlation between harsh, authoritarian parental practices and risk for aggression and conduct problems several years later, African American children did not show such predictability at all. (Importantly, however, children from both ethnic backgrounds showed strong associations between actual abusive parenting and later aggression.) This provocative finding of an ethnic moderator effect requires explanation: Is there a different cultural meaning related to authoritarian parenting in different cultural subgroups? What are the implications for parenting interventions across ethnic and other subcultural groups? Clearly, the search for moderator variables is necessary to qualify any generic theories of the development of aggression and ASB.

Abuse

With respect to effects of abuse and family violence, physical abuse is a strong and consistently replicated risk factor (and, quite probably, etiological factor) for later aggression and violence in the child (see Coie & Dodge, 1998). Dodge, Bates, and Pettit (1990) discovered that early physical abuse was a clear risk factor for later aggressive behavior reported in school settings, even with statistical control of family ecological variables and child temperament. Indeed, intergenerational effects of abuse are empirically validated (Widom, 1989, 1997), strongly supporting the need for prevention and early intervention efforts in this area. Such effects could of course be genetically mediated, but evidence for psychosocial mechanisms in transmission is compelling (Coie & Dodge, 1998). Intriguingly, the effects of familial abuse on children's antisocial tendencies appear to be mediated in part by social-cognitive information-processing variables that emanate from the abuse experience and that appear related to reactive, retaliatory aggression (Dodge, 1991; Dodge, Pettit, Bates, & Valente, 1995; see also subsequent section). Furthermore, for girls sexual abuse may be a salient risk factor (Chesney-Lind & Shelden, 1992), albeit one that has diffuse and nonspecific effects on a host of behavioral and psychological facets of later functioning.

Along this line, we highlight that, given the low heritabilities of violent behavior per se (see earlier section), abusive psychosocial influences (beyond those pertinent to child abuse per se) may be strongly implicated in the causal pathways to violent interactions. A provocative perspective on this issue is provided by the sociologist Athens (1997; see description in Rhodes, 1999), who posits an exclusively psychosocial pathway termed “violentization.” In brief, this formulation claims that a necessary and sufficient explanation of extreme violence encompasses a process of brutalization (often from abusive parents, but potentially from other sources), which includes violent subjugation, personal horrification, and “coaching” in violence—all of which lead to dramatic alterations in self-perception and interpersonal judgment, and which then proceed through stages toward belligerence, violent acts, and in some cases virulence. It is tempting to posit that such experiences are more likely to occur in families and individuals with biological proclivities toward impulse control problems and aggression, but Athens insists that the process can be entirely psychosocial. Furthermore, it can pertain to females as well as males (though the latter are more likely to receive training in violentization). Readers are encouraged to discover this fascinating, alternative perspective on the socialization of violence.

Attachment and Multiple Family Risk Factors

A different approach to the development of conduct problems has been taken by theorists and investigators within the attachment tradition, with primary focus on the development of problem behavior early in life. Attachment theory focuses on the quality of parent–child relationships (not only in infancy but across the life span) to explain the development of psychopathology; behavior problems in children are often seen as strategies for receiving attention or gaining proximity to caregivers who may not respond to other approach signals (see the seminal formulation of Greenberg & Speltz, 1988).

Empirical studies of attachment security have found that some of the behaviors differentiating securely from insecurely attached children are identical to symptoms of early disruptive behavior disorders (Greenberg, Speltz, & DeKlyen, 1993). Furthermore, investigations linking infant attachment status with behavior problems in the

preschool years have yielded provocative (but inconsistent) findings: The avoidant pattern of insecure attachment is prospectively linked to oppositional defiant problems in the preschool years, and the disorganized/disoriented classification at 18 months predicts subsequent behavior problems of a hostile nature (Lyons-Ruth, Alpern, & Repacholi, 1993). Negative findings have also been reported, however (see Coie & Dodge, 1998).

Current formulations (DeKlyen & Speltz, 2001; Greenberg, Speltz, & DeKlyen, 1993) synthesize extant results by concluding that main effects from insecure attachment to child ASB have not been found but rather that attachment relationships interact with the child’s sex, with biological/temperamental aspects of the child, family ecological variables, and parent management practices to precipitate ASB. Indeed, the most supportive evidence comes from high-risk samples, which by definition include additional risk factors. Thus, as noted at the outset of the section on etiology, multivariate, transactional causal pathways are gaining ascendancy in the field. In addition, the meaning of and predictability from different attachment classifications may differ across cultures.

Cognitive and Social-Cognitive Variables

IQ and Neuropsychological Functioning

With regard to neuropsychological variables, we highlight the important synthesis of Moffitt and Lynam (1994), as well as the more recent integrative model of Lynam and Henry (2001). Their initial contention was that the often-cited IQ deficit (approaching half a standard deviation) in antisocial and delinquent samples is actually far greater (over a full standard deviation) in youth with early-onset CD and is not explicable on the basis of such factors as official detection of delinquency, motivation, racial status, SES, or school failure (Lynam, Moffitt, & Stouthamer-Loeber, 1993; Moffitt & Silva, 1988). Moving to more specific types of neuropsychological dysfunction, Moffitt and Lynam (1994) posited that deficits in (1) verbal reasoning and (2) “executive” functioning characterize the profiles of youngsters with early-onset, aggressive ASB and comorbid ADHD. Such deficits, which appear at quite early ages, yield cumulative effects on ASB over the course of development, by promoting impulsive responding, facilitating disruption of early care-

taker–child relationships, precipitating harsh or inconsistent parenting, and presaging academic underachievement. This framework thus holds that even subtle neuropsychological deficits will interact with a host of other variables—including parental socialization influences—to produce indirect and distal effects on the development and intensification of ASB. Indeed, one contention is that neuropsychological difficulties may increase vulnerability to pathological environmental circumstances (Moffitt & Lynam, 1994; see also Lahey, Waldman, & McBurnett, 1999).

We point out several additional considerations. First, recent research has highlighted that executive deficits as typically measured in neuropsychological batteries are specific to ADHD and not to ODD or CD (Nigg, Hinshaw, Carte, & Treuting, 1998; Hinshaw, Carte, Sami, Treuting, & Zupan, 2002). Still, the early comorbidity of aggression and ADHD is a clear risk factor for persistent conduct problems, as noted earlier; the executive dysfunction related to this comorbidity may well be implicated in the causal chain. In addition, for severe and violent criminality, Raine and Liu (1998) clearly implicate the role of frontal lobe/executive dysfunction. Furthermore, a long history of research implicates verbal deficits in the causal pathway to early-onset ASB and delinquency (Lynam & Henry, 2001). Second, Aguilar et al. (2000) have challenged the contention that early neuropsychological and biological indicators are the key components of early-onset ASB, proposing instead that family relational factors are the key. For a lively debate on the primacy of psychobiological versus environmental variables, see the rejoinders of Moffitt and Caspi (2001) and Moffitt et al. (2002). Third, neuropsychological effects are typically of small magnitude in relation to the risk for persistent ASB. Yet, as noted, their key influence may be in interaction and transaction with environmental factors. Fourth, despite the strong attention paid to deficits in verbal skills as related to CD and ASB, recent evidence suggests that early in development, spatial and perceptual forms of cognitive processing may set the stage for the development of aggression and ASB (Raine, Yaralian, Reynolds, Venables, & Mednick, 2002). More research with a developmental focus is sorely needed in this area.

Social-Cognitive Information Processing

One mechanism by which both psychobiological and familial factors may exert effects on ASB patterns is through a child's means of perceiving, con-

struing, and evaluating the social world. Because this area has received extensive attention in the cogent reviews of Crick and Dodge (1994) and Coie and Dodge (1998), we present only headlines herein. Spanning developmental, cognitive, and clinical child psychology, this work has proven heuristic for the study of aggressive behavior.

In the most detailed formulation of this model (Crick & Dodge, 1994), a dynamic, transactional network of cognitive processes is held to mediate children's interpersonal responses and ultimate social adjustment. These processes include, at early stages of information processing, the encoding and interpretation of social cues and the clarification of social goals; at intermediate stages, response access/construction and response decision; and finally, behavioral enactment, with consequent evaluation and response. Interrelationships among these stages are believed to be fluid and nonlinear, with continual interplay among biological predispositions, environmental cues, information-processing variables per se, and feedback from the interpersonal behavior and peer response.

A programmatic series of investigations has revealed that aggressive youngsters display deficits and distortions at various levels of this information-processing model. At an overview level, such children and adolescents (in comparison with nonaggressive youths) underutilize pertinent social cues, misattribute hostile intent to ambiguous peer provocations, generate fewer assertive solutions to social problems, and expect that aggressive responses will lead to reward (e.g., Dodge & Frame, 1982; Dodge, Price, Bachorowski, & Newman, 1990; see review in Crick & Dodge, 1994). Importantly, such effects are found in both community and clinical samples of aggressive youths, including severely violent offenders (Lochman & Dodge, 1994). More specific examination of subgroups, however, reveals that such "early-stage" deficits as cue underutilization and attributional distortions pertain specifically to the subgroup of aggressive youngsters with comorbid ADHD (Milich & Dodge, 1984) and/or to the earlier-noted subtype displaying reactive aggression (see Dodge, 1991). Presumably, the impulsive cognitive style displayed by these children limits a full scanning of pertinent social cues before behavioral decisions are made, and ambiguous interpersonal situations are (mis)construed as threats to the self. In contrast, proactively aggressive children, whose aggression subserves instrumental goals, may show their primary information-processing differences at later stages of the

model that incorporate the expectation of positive outcomes from aggressive acts (Dodge, 1991). In short, the model has allowed for specificity with respect to subcategories of aggressive youths.

As highlighted throughout the chapter, interplay among causal factors and underlying mechanisms is increasingly recognized as critical for accurate formulation of aggressive behavior patterns. It is certainly conceivable, for example, that certain temperamental styles, including those characterized by suboptimal attention, may relate to impulsive cognitive processing. Furthermore, as discussed earlier regarding familial influences, punitive and abusive parenting practices appear to influence aggressive behavior through their instigation of early-stage information-processing deficits and distortions (Dodge, Bates, & Pettit, 1990; Dodge et al., 1995). In other words, a child exposed to a harsh, abusive upbringing may begin to attribute malevolent intent to others, fueling negative and aggressive interchanges that reinforce the biased attribution. In passing, we must point out that despite the elegance of the social-cognitive information-processing model, large effect sizes are the exception rather than the rule (Coie & Dodge, 1998); social-cognitive factors are not sufficient in providing a full explanation of persistent ASB. Thus, once again, it is necessary to invoke multivariate models that can predict and explain, with greater precision, the complex interrelationships among causal and risk factors.

Although space does not permit a separate heading, we wish to highlight that the variables of lack of inhibition (i.e., impulsivity), social-cognitive information-processing deficits, and compromised verbal abilities all point to the potential for youths at risk for ASB (particularly reactive forms of aggression and persistent ASB) to suffer from emotion dysregulation. Even defining this construct is laden with pitfalls, but theoretical and empirical accounts of the role of excesses in emotional reactivity and deficits in emotion regulation regarding the development of child psychopathology are beginning to appear (e.g., Keenan, 2000). Interested readers are advised to keep abreast of developments in this potentially fruitful area of investigation.

Peer Influences

In our truncated review, we make a key distinction—that between (1) peer rejection in childhood and (2) association with deviant (i.e., anti-

social) peers in preadolescence and adolescence. Each is related to the development of ASB and delinquency, yet perhaps in different ways, and apparently for different subgroups of youths with aggression and conduct problems.

First, peer rejection in childhood is strongly related to early onset of both aggressive behavior and ADHD-related symptomatology, and particularly to their combination (Hinshaw & Melnick, 1995). Indeed, whereas ADHD is clearly associated with peer rejection, aggression in the absence of ADHD (particularly, proactive aggression) may be related to “controversial” sociometric status (e.g., Milich & Landau, 1988). Yet children with comorbid ADHD and aggressive behavior patterns receive extremes of peer rejection (Hinshaw & Melnick, 1995). Importantly, considerable evidence (especially from the programmatic research of Coie and colleagues) demonstrates that peer rejection in childhood is a significant, incremental predictor of ASB and delinquent behavior during adolescence, even when baseline levels of aggression are controlled for (Coie, Terry, Lenox, Lochman, & Hyman, 1995; see review in Coie & Dodge, 1998). Thus, whereas peer rejection may be a marker during childhood of externalizing, intrusive, and insensitive behavior patterns, it also appears to be a causal factor in and of itself for the persistence and escalation of antisocial patterns. Mechanisms responsible for this predictive relationship could include a child’s exclusion from opportunities for peer socialization, modeling of aggressive behavior by other rejected children, or demoralization in response to the self-perception of peer rejection (see the discussion in Laird, Jordan, Dodge, Pettit, & Bates, 2001). Coie and Lenox (1994) provide a view from the microanalytic level as to the processes by which aggressive children who are also rejected by their peers display a qualitatively distinct pattern of peer interactions that promotes further escalation of aggressive behavior.

Second, even for children without a history of aggression and ASB during childhood, association with deviant, antisocial peers during early adolescence clearly appears to be a direct causal influence on the propensity for delinquent behavior (see Capaldi & Patterson, 1994). Two perspectives are important in this regard: One is “selection,” whereby youngsters with marginal social skills or subclinical aggressive tendencies select deviant peer networks; the other is “facilitation,” in which associations with antisocial peers propel and escalate a pattern of antisocial behaviors via conversational dynamics, modeling, and provision

of opportunity for delinquent involvement. The work of Dishion and his research group (e.g., Dishion, Andrews, & Crosby, 1995) provides a heuristic perspective on the types of peer processes that are salient in this regard.

In a recent longitudinal, multivariate model, Laird et al. (2001) showed that both processes may operate to pave the way for adolescent ASB, but that peer rejection may be more salient for early-onset ASB, whereas deviant peer association pertains selectively to those with later-onset ASB. (Laird et al. also found that the continuity of aggressive behavior mediated the relationship between early peer rejection and later association with deviant peers.) Indeed, the review of Capaldi and Patterson (1994) suggests strongly that involvement with antisocial peers is a direct influence on delinquent behavior patterns in adolescent-onset ASB. At the same time, both peer rejection and association with deviant peers do not occur in a vacuum; multiple levels of influence appear operative.

Wider Contextual Factors

For many years, investigators have noted a clear link between measures of psychosocial adversity—including impoverishment, high rates of crime in the neighborhood, family crowding, and related factors—and children's risk for ASB (see review in Coie & Dodge, 1998). Indeed, the risk for antisocial activity is far higher in crowded, poverty-stricken, inner-city areas than in rural settings (Rutter et al., 1974)—a factor of considerable influence for the large numbers of impoverished, urban youths (often of ethnic minority status). Whereas anything more than a cursory review of the long history of research regarding social/cultural influences on ASB and delinquency is beyond the scope of this chapter, a key issue is whether such socioeconomic and neighborhood factors contribute directly to ASB patterns or whether their effects are mediated by more specific variables, such as parent-child interactions or social-cognitive processes.

We again cite the masterful synthesis of Capaldi and Patterson (1994), who examined a wide array of contextual factors for their predictive relationships to ASB patterns for males, testing for direct versus indirect effects of such factors. The research program is provocative, in that Patterson and colleagues are conceptualizing a far broader network for the development of aggression and CD than microsocial parent-child interactions per

se. First, high levels of family adversity and several related contextual factors (multiple family transitions, unemployment, and low SES) were shown to relate specifically to early-onset (but not adolescent-onset) CD. This list of factors adds to those proposed by Moffitt (1993) for childhood-onset ASB, which include neuropsychological dysfunction and attention deficits as well as discordant family interchange. Early-onset, persistent ASB patterns are clearly overdetermined.

Second, as indicated above, evidence supported the direct (as opposed to mediated) effects on ASB of the contextual factor of exposure to a deviant peer group, particularly for boys *without* a childhood onset of ASB. High rates of such association strongly influence delinquency (Sampson & Groves, 1989). This finding once again underscores the importance of subtyping aggression and ASB; direct effects of deviant peer groups pertain chiefly to the adolescent-onset subtype. Third, the effects of several important contextual factors on ASB were reduced or rendered non-significant when parenting variables were added to the predictive equations of Capaldi and Patterson (1994). The direct effects of low SES in particular were erased when parent management variables were included (see also Dodge, Pettit, & Bates, 1994); the roles of family transitions, stress, and unemployment also appeared to be indirect. Fourth, community and other contextual variables related to antisocial outcomes in a “chain reaction” fashion (Capaldi & Patterson, 1994), whereby unemployment (for example) predicted greater levels of stress and greater numbers of family transitions, which in turn reduced family involvement and monitoring and predicted higher levels of coercive parenting.

We point out that neighborhood effects on child psychopathology have recently been found, in a genetically sensitive design, to be separable from genetic effects or genetic mediation and to be of substantive importance (Caspi, Taylor, Moffitt, & Plomin, 2000). Thus it is not just the case that neighborhood influences reflect “selection” (the tendencies of persons with antisocial histories to aggregate in disenfranchised locations); they also appear to exert causal influence on the risk for dysfunction and impairment. But again, interactive and protective factors are operative. For example, Richters and Martinez (1993) examined the role of children's exposure to community violence in predicting maladjustment. Whereas such exposure predicted youths' self-reported symptomatology, the effects were

mitigated when indices of family stability were controlled statistically. In this instance, family-level variables served as a protective factor against the risk incurred by high-frequency encounters with significant violence in the neighborhood and community.

School-based violence has been in the news considerably during the past several years. Mulvey and Cauffman (2001) provide a thoughtful perspective on (1) the difficulties involved in predicting extremely low-base-rate phenomena like school violence, and (2) the kinds of environmental changes that are most likely to be preventive. They note, as well, the contextual interrelatedness of school violence in neighborhood and family factors (see Laub & Lauritsen, 1998). Finally, we note that the lack of direct effects for many wider contextual variables does not reduce their importance in explaining ASB. Indeed, researchers and policy makers must be aware of the economic and community-level factors that predispose certain families to provide markedly poor socialization for their offspring. ASB patterns are not only intergenerational, but are intertwined with important economic, community, and family ecological factors.

Additional Data on Interaction and Transaction

We now present several additional examples of research findings regarding the development of ASB that exemplify interaction and transaction across risk and etiological factors. Our purpose here is to illustrate the kinds of results, and the kinds of models, that are most likely to portray how risk and etiological factors work in combination to yield the patterns of aggression and ASB likely to come to clinical attention.

First, as indicated in the first edition of this chapter (Hinshaw & Anderson, 1996), in a study that paved the way for her conceptualization of subtypes of ASB, Moffitt (1990) examined predictive relations between early (age 5) measures of aggressive and ADHD-related symptomatology and early adolescent indicators of delinquency. Whereas the strongest predictor of delinquent functioning incorporated early indicators of aggressive behavior, measures of ADHD behaviors at age 5 significantly incremented the prediction; that is, they accounted for significant variance, even when baseline aggression was controlled for. Crucially, however, the effects of early behavior patterns in predicting adolescent ASB were mod-

erated by (1) a composite measure of family adversity and (2) child IQ, such that the highest-risk youths were those displaying high rates of externalizing behavior patterns at an early age, but only if they also had either subaverage IQ scores or multiple indicators of family adversity. Hence intraindividual behavioral factors, intraindividual cognitive/neuropsychological factors, and several indices of family-level factors (e.g., parental distress, family discord) worked interactively to increase the risk for early adolescent ASB.

Next, Raine and colleagues (Raine, Brennan, & Mednick, 1994; Raine et al., 1997) have embarked on systematic research with respect to the “biosocial” interactive effects of (1) birth complications (defined as presence of any of the variables of forceps extraction, breech delivery, umbilical cord prolapse, pre-eclampsia at delivery, and/or long duration of the birth process) with (2) early maternal rejection of child (defined as public institutional care of infant, attempt to abort fetus, and/or unwanted pregnancy). Utilizing a large Danish birth cohort, they found that with respect to outcomes measured at ages 17–19 (Raine et al., 1994) and age 34 (Raine, Brennan, Mednick, & Mednick, 1996), interactions between these two factors attained significance with respect to the prediction of violent crime (as opposed to nonviolent offending) and to the prediction of early-onset (but not late-onset) ASB. Raine et al. (1996) discovered that the same interaction patterns held with respect to prediction of academic problems as well. For most outcomes, the interaction pattern was provocative, such that neither single-risk group displayed elevated rates of violence, whereas the “biosocial” (i.e., dual-risk) participants showed rates far above those of any other subgroup. Raine et al. (1997) found that the presence of maternal psychiatric history in the prediction equations did not mediate the core results, and that the key maternal rejection variables “carrying” the interactions were institutional placement and the attempt to abort the fetus. Although the viability of these findings has been challenged by Rutter et al. (1998)—who questioned, for example, the mechanism whereby birth complications would specifically influence risk for violence—the overall pattern strongly suggests that interactive effects of early biological and early environmental variables are influential.

Third, and briefly, Lynam et al. (2000) found a provocative interaction between neighborhood characteristics and an intraindividual child variable, impulsivity, in predicting risk for adolescent

offending. The pattern of findings was such that the expected predictive power from children's impulsivity was amplified when the children lived in more impoverished neighborhoods. Hence, in this report, both within-child and broad contextual factors were implicated in the highest risk for ASB.

Fourth, although it does not exemplify interactive effects per se, we highlight the recent research of Nagin and Tremblay (2001), who combined person-centered and variable-centered research strategies in an attempt to understand mechanisms responsible for persistence of ASB (in this case, physical aggression) from childhood through midadolescence. They first utilized their own typology (Nagin & Tremblay, 1999), which comprised four classifications of a Canadian, high-risk, kindergarten-defined, male sample: (1) chronic physical aggression (4% of the sample)—high aggression throughout the 9-year time span; (2) high-level declining trajectory (28%)—high aggression in kindergarten that subsequently declined; (3) moderate-level declining trajectory (52%)—modest rates in kindergarten that subsequently decreased to near zero; and (4) low trajectory (17%)—rare displays of physical aggression throughout development. (Recall our earlier discussion of Nagin & Tremblay's [1999] work, when we made the case that the majority of boys with early onset of ASB do not persist in it.) The goal of Nagin and Tremblay (2001) was to appraise which intraindividual and parental/family factors best distinguished the trajectory groups. In brief, child-level factors distinguished groups 1 and 2, those with high initial rates of physical aggression, from 3 and 4, those low on initial aggression. The specific factors were the presence of hyperactivity and oppositionality in kindergarten. On the other hand, family-level factors separated group 1 from group 2: Teenage status of mothers and their low educational attainment distinguished the small, but virulent, subgroup displaying physical aggression that persisted from age 6 through age 15 from the children showing high aggression in kindergarten that subsequently declined. Thus factors responsible for the onset of aggressive behavior patterns may differ from those predicting persistence.

Space does not permit additional examples (e.g., as noted above, O'Connor et al. [1998] present data on gene-environment correlations in relation to ASB). After discussing sex differences in aggression and ASB, we return to such interactive and person-centered models as we

attempt an integrated theoretical statement regarding the development of these behavior patterns.

SEX DIFFERENCES

Readers may have noticed that the vast majority of the literature reviewed herein pertains largely or exclusively to males. In fact, key reviews in the last decade have called for focused attention on the crucial topic of sex differences regarding ODD, CD, and ASB in general (e.g., Coie & Dodge, 1998; Keenan, Loeber, & Green, 1999; Rutter et al., 1998). We have deferred our discussion of this issue until now, so that the reader may be able to appraise information on sex differences in light of the prior evidence regarding definitional issues, background information, prevalence, developmental progressions, and etiological influences. For recent, essential reading on this domain, see Moffitt et al. (2001).

We note at the outset that among all the risk factors for conduct problems and ASB, male sex has been considered by some experts as the most important (see Robins, 1991). Yet increasing awareness of the growing problems of ASB among girls and women is clearly evident (Keenan et al., 1999), with recognition that female manifestations of disruptive behavior disorders and aggression are quite real and quite prevalent. Note, however, that investigations of sex differences in a particular form of psychopathology (or investigations of other kinds of group differences, including ethnic or socioeconomic) often begin and end with description of mean levels of the amounts of psychopathological functioning in the relevant subgroups (e.g., boys vs. girls). A key point in this regard is that similar mean levels in different subgroups may belie fundamentally different patterns of risk processes, just as divergent levels across subgroups may be undergirded by similar underlying causal processes. The essential goal is explanation, not just documentation of rates and sex differences in such rates.

Rates of Aggression, ASB, and Disruptive Behavior Disorders

Crucially, recent investigations of aggression among females, utilizing such objective data collection efforts as videotaped observations during laboratory assessments, yield remarkably consistent findings regarding baseline rates of external-

izing behavior in early development. That is, during the initial years of life, there are virtually no sex differences in activity level, noncompliance, other problem behaviors, and the temperament-related variables of “difficult” temperament or behavioral disinhibition (see reviews by Keenan & Shaw, 1997; Keenan et al., 1999). The exception here may relate to boys’ greater likelihood of angry expressions during infancy, though data are not clear in this regard. By the preschool years and certainly by the start of elementary school, however, sex differences are apparent and are robust until adolescence. That is, male predominance is evident across different forms of aggression, both physical and verbal, with samples spanning community, epidemiological, and clinic-referred ascertainment procedures (see review in Coie & Dodge, 1998). For a theoretically rich account of putative reasons why males begin to “outperform” females with respect to the display of aggressive behavior patterns during childhood, the synthetic review of Keenan and Shaw (1997) is essential reading. In brief, they note that girls’ earlier development of basic psychobiological, cognitive, and emotion-regulating capacities promote socialization patterns that funnel girls into internalizing, rather than externalizing, manifestations.

How strong are the sex differences in childhood regarding externalizing behavior patterns? With respect to categorical definitions, rates of ODD in early childhood appear similar between girls and boys, but by the late preschool and early elementary years, males predominate (Keenan et al., 1999). On the other hand, as do boys, girls display increases in rates of oppositionality and defiance in adolescence (McDermott, 1996; Rutter et al., 1998). With regard to CD, boys greatly outnumber girls in childhood and pre-adolescence, with ratios of 4:1 commonly reported (e.g., Zoccolillo, 1993). By adolescence, however, girls appear to show a precipitous rise in rates of disruptive behavior disorders and ASB, with the clear exception that rates of physical aggression, particularly violence, continue to be substantially elevated in males. Still, although males outnumber females in terms of CD diagnoses during adolescence, the sex ratio is closer to even. Thus CD constitutes a major mental health problem for girls during the teenage years.

We note, in passing, that research methods may be partly responsible for the overarching conclusion that males are more aggressive than females during childhood. For example, Webster-

Stratton (1996) utilized home observations by objective staffers and found no significant sex differences among a sample of boys and girls (ages 4–7) on scores of total externalizing behaviors, verbal deviance, noncompliance, and positive affect. On the whole, however, a plethora of research has found that beyond infancy and toddlerhood, male and female rates of aggressive behavior patterns begin to diverge (Coie & Dodge, 1998; Keenan & Shaw, 1997). One consequence of this general conclusion is that girls with early conduct problems are behaviorally more deviant relative to same-sex peers than are boys with conduct problems; as a result, girls suffer from more negative peer regard related to behavioral acting-out than do boys (e.g., Carlson, Tamm, & Gaub, 1997; Lancelotta & Vaughan, 1989). Furthermore, in terms of comorbidity, a gender paradox may be salient, whereby the sex (in this case, females) with *lower* base rates of the disorder in question tends to show *higher* rates of comorbidity with other disorders (see, e.g., Loeber & Keenan, 1994).

A notable exception to the male predominance in aggressive behavior patterns is the subdomain of indirect or relational aggression. Broadly defined (see also the earlier section on subtypes of aggressive behavior), “relational aggression” is an attempt to inflict harm upon another person by manipulating and damaging social relationships (Crick & Grotpeter, 1995). Relevant behaviors include efforts at ostracizing another student, encouraging retaliation by others, exclusionary play, and generating rumors. Among school-age children, girls show significantly higher rates of these acts than do boys; importantly, peer-nominated relational aggression predicts such negative outcomes as loneliness, social isolation, depression, and sociometric rejection (Crick & Grotpeter, 1995; Crick & Bigbee, 1998). Thus relational aggression appears to be an important variant of ASB in girls, with the potential for significant psychological distress. Most investigations appear to have underestimated the prevalence of aggression among girls, given the assumption that their behaviors would be identical to those exemplified by males.

Considerable controversy exists about the inclusion of other behavior patterns, which are not part of the current diagnostic classification systems, as relevant to disruptive behavior disorders. Substance use/abuse and sexual promiscuity are prime examples; although they lie outside the parameters of CD per se (American Psychiatric

Association, 1994), they may be important indicators of current or future psychopathology (and can certainly be impairing) for both sexes. Other investigators have suggested that somatization may be a constituent feature of the antisocial spectrum for girls (Lilienfeld, 1992), despite its lack of inclusion in formal diagnostic criteria. Discussion about potential changes in diagnostic thresholds (sex-specific vs. universal) has been an important debate in the field (see Zoccolillo, 1993; Zahn-Waxler, 1993). In brief, Zoccolillo (1993; see also Zoccolillo, Tremblay, & Vitaro, 1996) has contended that (1) addition of additional, pertinent behavioral features and (2) sex-specific norms would more fully capture the real range of ASB in females, whereas Zahn-Waxler (1993) has contended that “watering down” the criterion levels of behavioral deviance and including a range of nonviolent and nonharmful actions in the nosological systems would conflate nonharmful behavior patterns with diagnosable disorders in females. These and other issues underscore the points that current estimates of prevalence may reflect flawed assumptions about the manifestation of aggression and ASB in girls, and that classification and diagnostic systems must restrict diagnosis to individuals with significant impairment. Along this line, we once again call attention to the importance of recognizing the heterogeneity and subtypes of aggression and ASB, particularly when investigators are describing and discussing sex differences and positing developmental models for females.

Developmental Trajectories

A clear finding is that boys clearly outnumber girls in terms of early-onset variants of ASB and/or CD. Indeed, in the entire Dunedin sample (described earlier), only 6 girls out of over 500 qualified for the life-course-persistent subcategory, defined on the basis of early-onset and persistent aggression and ASB (Moffitt & Caspi, 2001). Note in this regard that boys also greatly outnumber girls with respect to key risk factors for and correlates of ASB, including ADHD, language delays, and neuropsychological deficits. Intriguingly, some evidence suggests that the construct of “difficult” temperament during toddlerhood may predict to later internalizing problems in girls as opposed to externalizing problems in boys (Fagot & Leve, 1998). In any event, by the late preschool years, boys outpace girls in terms of externalizing behavior problems.

Adolescence is a significant developmental transition that marks the onset of important changes with respect to rates of aggression, ASB, and CD. Whether measured dimensionally or categorically, the overall gender discrepancy appears to diminish beyond childhood. Findings from the Dunedin birth cohort in New Zealand reveal substantially increased rates of nonaggressive ASB in adolescent females (McGee, Feehan, Williams, & Anderson, 1992), and adolescent girls in other samples have shown an increase in their overall rates of CD (Offord, Boyle, & Racine, 1991), which collectively account for a significant portion of this narrowing gap. Thus girls show substantial increases in covert or status offenses, such as truancy, theft, substance use/abuse, and frequent lying, in the transition to adolescence. Overall, girls lag behind boys in the propensity to display physical aggression, especially violence; yet the peak age of offending among girls is during the period of early adolescence, whereas for boys the peak age is at the end of adolescence (Rutter et al., 1998). Thus girls—perhaps because their onset of puberty is earlier than that of boys—show particular risk for ASB during the early adolescent period (see below for potential mechanisms).

Whereas female rates of aggression and CD (at least the nonaggressive subtype) begin to approach those of males in adolescence, the underlying mechanisms and processes governing such relationships may be different. Despite the extensive impact of Moffitt’s (1993) typology, which features age of onset as a key subclassification variable, the applicability of these typologies to female aggression and ASB is still questionable. In fact, Silverthorn and Frick (1999) have hypothesized that a dual-pathway model may not be appropriate for severely antisocial girls. Specifically, they contend (1) that girls with significant levels of ASB show the same types of cognitive, neuropsychological, and familial risk factors as do boys with early-onset ASB, but (2) that such girls’ initiation of aggression and antisocial responding is “delayed” by several years into early adolescence. Furthermore, Kratzer and Hodgins (1999) found that a considerable amount of female criminal behavior in early adulthood was accounted for by adolescent-onset and even “adult-starter” subtypes, rather than the early-starter subtype, as was the case for boys. In all, according to this viewpoint, early age of onset per se may yield less robust predictions to persistent antisocial behavior for girls than it has for boys.

Yet at least some evidence exists that girls and boys with conduct problems have comparable ages at onset of problem behavior (see review in Keenan et al., 1999). Furthermore, recent data from the ongoing birth cohort study in Dunedin (Moffitt & Caspi, 2001) challenge the viability of the “delayed-onset” concept: Despite extremely low cell sizes for females on the life-course-persistent path, these girls demonstrated a pattern of early childhood risk factors (temperament, family adversity and ineffective parenting, and neurocognitive dysfunction) identical to that of the early-onset boys. In addition, the adolescent-onset boys and girls (n 's = 122 and 78 youths, respectively, showing a relative “catch-up” of girls with late-onset conduct problems) both displayed extremely high rates of contact with deviant peers, consistent with the “adolescent-limited” typology. Overall, examination of sex differences in pathways and mechanisms may also be facilitated by examination of other large samples (Aguilar et al., 2000; Fergusson et al., 2000; Kratzer & Hodgins, 1999). At present, the applicability of pathway notions developed for males to females is not assured.

Adult Outcomes: Evidence for Multi- and Equifinality

Although the stability of aggression and ASB is as stable over short time periods in female as it is in males, female stability appears lower than male stability over longer assessment intervals (Frick & Loney, 1999). In fact, a reliable conclusion from multiple investigations is that the adult outcomes of girls with severe externalizing behavior patterns reveal impairment across numerous psychological and functional domains (Robins, 1991; Woodward & Fergusson, 1999; Bardone, Moffitt, Caspi, Dickson, & Silva, 1996; Werner & Smith, 1992). Although such negative outcomes are frequently antisocial in nature—indeed, females may show the same rates of predictability of antisocial patterns in adulthood as do males (Keenan et al., 1999)—outcomes appear to be more highly dispersed in females than in males. Early pregnancy, suicide, physical partner violence, earlier marriage and earlier divorce, lower educational attainment, psychiatric distress (particularly internalizing conditions), difficult parent-child relationships, and higher rates of service utilization have all shown some association with childhood or adolescent aggression and ASB in girls (see reviews by Keenan et al., 1999, and

Pajer, 1998). These findings suggest that multifinality (the display of diverging outcomes from similar initial conditions) pertains more to girls than to boys with ASB. Such results should be viewed with some caution, however, as highly divergent methods, sample characteristics, and experimental designs make direct sex comparisons impossible. In addition, as we have emphasized throughout, a key priority for developmental psychopathologists is to elucidate the relevant mechanisms governing these relationships. For example, Woodward and Fergusson (1999) showed that predictions to adolescent pregnancy from early conduct problems were partially mediated by sociodemographic factors, family functioning, and “risk taking.”

On the basis of these preliminary results, it appears that females with externalizing disorders (many of whom have onsets of these disorders during adolescence) show more evidence than do males of multifinality, as evidenced by a wider range of outcomes (especially in the internalizing domain) that emanate from their early aggression. A requirement for future research efforts, however, is the inclusion of multiple clinical or psychiatric groups, allowing for comparisons of developmental trajectories across such groups, with the potential for finding equifinality between or among disorders. For example, in an important investigation, Bardone et al. (1996) showed that whereas CD versus depression in females showed several distinct outcomes at age 21, there were also similar outcomes, including comorbid anxiety disorders, multiple drug use, early school departure, and early childbearing. In addition, Kratzer and Hodgins (1997) discovered that the risk ratios related to the prediction of adult criminality and mental health problems from child conduct problems were *higher* for girls than for boys, once initial baseline differences in childhood conduct problems (higher in boys, as would be expected) were controlled for. Interestingly, for these girls, the adult criminal outcomes were limited nearly exclusively to substance use disorders, again suggesting that more specific predictions to aggression and violence occur in males.

Mechanisms of Differentiation

In the spirit of supplementing general models of development with work on specific, interactive mechanisms that may drive predictive outcomes, we review two domains that offer potential insight into the differentiation of sex-related ASB pat-

terns: the influence of social groups and pubertal development.

Maccoby's (1998) review examining sex differences of young children's play styles and play groups underscores several key lessons. First, she suggests that the characteristics of the groups in which children play are as salient for development as is temperament or personality. Among boys, play styles are generally more physical and active, involving greater risks. Thus developmentally extreme boys may miss important socialization from the peer group about "normative" levels and types of aggression. Given that levels of activity are generally lower among female groups, aggressive girls risk even more ostracism and loss of friendship (and consequently a key source of socialization). Put another way, the social sanctions against acting-out behaviors may contribute to lower base rates of aggression in girls than in boys; however, they may also explain the finding that girls who exhibit severe conduct problems despite such sanctions tend to show even greater impairment than boys with comparable behavior problems (Coie & Dodge, 1998).

Late childhood and early adolescence mark an important transition in the social groups of children, as individuals no longer participate in groups that are almost universally same-sex (Maccoby, 1998). As they enter adolescence, males and females begin to interact more consistently, perhaps with the effect of introducing females to certain ASB patterns that were previously the domain of boys. Such interactions are particularly salient among girls undergoing early puberty, to which we now direct attention.

Early menarche has been shown to be a reliable precursor to behavior problems among female adolescents (Caspi, Lynam, Moffitt, & Silva, 1993; Garber, Lewinsohn, Seeley, & Brooks, 1997; Ge, Conger, & Elder, 1996). However, such main effects disguise otherwise rich and complex relations, including the role of prior problem behavior in accentuating the effects of early puberty (Moffitt, Caspi, Belsky, & Silva, 1992); the moderating role of same-sex versus different-sex schools (Caspi et al., 1993); and the mediating roles of association with older and deviant male peers, as well as explicit sexual pressure (Ge et al., 1996). Thus early puberty appears to be a risk factor for ASB in girls only if the girls attend co-educational schools, where they experience boys with early-onset ASB as models, instigators, and provocateurs (Caspi et al., 1993). Furthermore, although hormonal influences may be associated

with mood and behavior problems in girls, such factors are likely to interact with other variables, such as the developmental stage of the endocrine system. Similarly, the direct impact of hormones is apt to influence related systems, such as excitability and emotionality, with indirect effects on psychopathology per se (Brooks-Gunn & Warren, 1989). Finally, the social context appears essential for expression of such propensities in terms of ASB. In summary, physiological development and maturation, particularly with an early onset, may represent a generative mechanism of behavior problems (or of accentuating existing distress) that transacts with the environment to elicit significant levels of ASB in females.

In closing this section, we note briefly the strong likelihood that conduct problems and CD predict risky sexual behavior and early pregnancy in girls (see Keenan et al., 1999). With this point in mind, recall that (1) a key risk factor for children's ASB (and particularly for persistent ASB) is being born to a teenage mother; and (2) the risk of teenage parenting in predicting offspring's conduct problems is accentuated by a history of acting-out behavior in the mother (see the subsection on family structure in "Risk Factors and Etiological Formulations"). It is likely, therefore, that conduct problems in the mother, if resulting in teenage pregnancy and birth, may precipitate an intergenerational cycle of conduct problems in the offspring, abetted by socioeconomic disadvantage and mediated via problematic parenting skills. If so, this would demonstrate reciprocal influences related to developmental trajectories span generations. The gravity and persistence of such multigenerational influences are sobering, in terms of how far the field needs to travel to make a significant difference in the trajectories pertaining to serious ASB.

THEORETICAL SYNTHESIS

In this final section, we attempt to amalgamate the extensive information reported above into a synthetic account of the development of ODD, CD, and persistent ASB. Of course, given the salience of such constructs as divergent developmental pathways, multifinality, and equifinality, no single unifying theory is adequate to the task. Rather, we incorporate a multipronged model. Critics will be able to detect many gaps in our brief synthesis, which is intended to be heuristic

rather than comprehensive, and which strains the page limits for our already long chapter.

Developmental Models

First, although our account reflects the considerable empirical data base supporting the notion that ASB has extensive intraindividual and familial risk factors, wide cultural factors are no doubt responsible for (1) the increases in aggression and violence across recent generations, and (2) the widely diverging rates across cultures and nations (Rutter et al., 1998). Indeed, variables and processes that promote and maintain individual differences in aggressive and antisocial tendencies need not overlap with those that promote cohort or area differences. For example, although the role of genetic vulnerability has now been shown to contribute substantially to the risk for early-onset ASB (Taylor et al., 2000), perhaps through its linkage with comorbid hyperactivity or impulsivity (Silberg et al., 1996; see also White et al., 1994), genetic factors have little if anything to do with the huge surplus of homicide in the United States (particularly among young people), which clearly relates more to the ready access to guns and other violent weaponry in our nation (Loeber, Delamatre, et al., 1999; Rutter et al., 1998). Furthermore, at the level of individuals, factors that promote initiation of aggressive and violent behavior are not necessarily the same as those that maintain such actions. Recall the relevant research of Nagin and Tremblay (2001): Child variables predicted early initiation of physical aggression, but family factors (teenage parenting, low parental educational attainment) predicted its persistence. (Note, however, that such parenting factors may themselves be subject to genetic mediation, raising yet again the interconnectedness of levels of causation.) Thus the strong evidence for multifactorial and interactive models of the development and maintenance of aggression and ASB makes it difficult to put forth an explanatory model in linear fashion.

We begin at the earliest stages of development, at which time (1) heritabilities for temperamental factors related to later aggression are not strong, and (2) sex differences in such emotional and behavioral patterns are minimal. By the preschool years, however, traits of impulsivity and sensation seeking become salient and more heritable, as are sex differences in aggressive interchanges, perhaps fueled by caregiver patterns of response to individual differences in difficult temperament

or to early neurocognitive and language deficits. Indeed, caregivers of young children with such intraindividual tendencies are likely to be young, poorly educated parents with problems of impulse control and emotion regulation themselves. Furthermore, surprisingly early in development (and particularly by the preschool years), boys' and girls' peer socialization patterns have become substantially separate, accentuating externalizing tendencies among boys and internalizing patterns among girls (Keenan & Shaw, 1997; Maccoby, 1998). Thus, even before the onset of formal schooling, a web of gene–environment correlations and interactions is being spun, such that youngsters with high ADHD-related symptomatology (particularly impulsivity) and low verbal abilities (and perhaps executive functions) tend to elicit chains of negative, coercive interaction from families and peers, (Snyder & Patterson, 1995; see also the model of Moffitt, 1993). Note in this regard that the cognitive and behavioral patterns characteristic of ADHD are strongly heritable; when they occur in combination with early oppositionality and aggression, they tend to fuel the onset of a pernicious pattern of escalating coercion at home (often preceded by insecure attachment during infancy), academic failure at school, and peer rejection from agemates, all of which predict continuation of externalizing behavior patterns (e.g., Campbell, in press; Hinshaw, 1992, 1999; Parker & Asher, 1987; Patterson et al., 1992). If physical abuse is added to the mix, the risk of ensuing aggression—mediated by social-cognitive information-processing biases and failures of empathic responding—is even stronger (Coie & Dodge, 1998).

Many of the risk factors identified in Table 3.4, in fact, pertain to such “early starters,” who are highly likely to be male and who are at far higher than average risk for continuation of aggression and ASB beyond childhood. Note that in the cases with the worst prognosis, individual and parenting risks are embedded in a matrix of family structural variables, neighborhood disenfranchisement, poverty, and unresponsive schooling. Such variables do not appear to have large direct effects on emerging ASB patterns, but rather appear to be mediated on the whole by discordant, harsh, and unresponsive parent–child interactions (Capaldi & Patterson, 1994).

We hasten to point out, however, three essential points. First, far from all boys with early signs of aggressive, hyperactive, and impulsive behavior will show an escalating, “life-course-

persistent” pathway; in fact, desistance is normative. Second, it may well take examination of factors present well before the preschool years to ascertain just which “early starters” show the highest rates of persistence and escalation. Indeed, those with actual risk may need to be tracked from infancy or even earlier (e.g., Tremblay, 2000). The flip side, of course, is that the earlier the time period of the prediction, the more likely it is that false-positive predictions will occur, given current knowledge; this state of affairs presents an empirical and ethical conundrum for the field. Third, those children most likely to show the greatest risk for intensification of ASB are those with combinations of etiological influences (Rutter et al., 1974; Greenberg et al., 1993). That is, risks from insecure attachment, difficult temperament, discordant parent–child interactions after infancy and toddlerhood, neuropsychological deficits, unfavorable family structural factors, and socio-economic adversity are far more pernicious in combination than when present singly or dually.

Thus, regarding the development of forms of ODD that are likely to escalate into CD, important patterns of transaction with the environment during the preschool years are essential contributing forces. In some cases, extremes of temperament; extremes of parental psychopathology/antisocial activities; extremes of heritable risk for ADHD; extremes of exposure to violent neighborhoods; and/or extremes of harsh, inconsistent, and unresponsive parenting may be sufficient in and of themselves to demarcate a trajectory heading toward aggression and delinquency. In most cases, however, the interaction and transaction of such risks are likely to yield higher probabilities of early initiation and persistence of ASB. Moreover, continued developmental influences via inconsistent and harsh families, unresponsive and chaotic schools, and deviant peer groups are undoubtedly necessary to maintain and fuel escalation to serious aggression and violence. Recall that early age at onset of diverse manifestations of antisocial activities is what best predicts persistent conduct problems. The developmental models of Lahey, Loeber, and colleagues are heuristic in this regard (see Hinshaw et al., 1993): When early ADHD symptoms and oppositionality are followed by physical fighting, stealing at home, and persistent lying by the start of elementary school, the pernicious problems of physical and sexual assault, serious burglary, initiation of substance abuse, and repetitive delinquency are likely by

adolescence. Furthermore, the constellation of callous/unemotional traits may betray a psychophysiological pattern of poor conditionability and poor response to threatened punishment, which sets in motion the precursors to adult psychopathy (Frick et al., 2000).

It must be recalled that it is normative for physical aggression to decrease throughout childhood and adolescence. Thus, from this perspective, perhaps the field should be examining not so much what propels increases in ASB across development as what factors attenuate the age-expected decrease in vulnerable individuals. In addition, we reiterate that heritabilities for violence are low, leaving open the possibility that psychosocial influences are strong determinants of the propensity for violent behavior patterns. Interested readers are again referred to the provocative work of Athens (1997) and Rhodes (1999) for an account of the psychosocial, social-cognitive, and developmental construct of violentization.

We have not adequately emphasized, throughout this chapter, the strong likelihood that aggressive offenders have a high rate of being victimized as well as of victimizing. They are also, as noted earlier, far more likely than the norm to attempt suicide (Cairns et al., 1988). Thus added risks of aggression and ASB include serious injury or death (Loeber & Farrington, 2000).

A different pathway to adolescent ASB and offending is seen in the “adolescent-limited” subtype of Moffitt (1993), comprising relatively (and, in some investigations, absolutely) large numbers of adolescents who engage in nonaggressive forms of conduct problems but without the complex psychopathological histories of those with early-onset ASB. Social and historical factors, especially the “maturity gap” in many Western societies, may contribute to the protracted adolescence of large numbers of youths, who seek power and status otherwise unavailable to them through antisocial actions. Association with delinquent and otherwise deviant peers is a direct socialization influence on such adolescents. Thus youths with early-onset ASB may provide negative models for a far larger subset of teens. Recent data suggest, as well, that the depiction of such individuals as rapidly desisting from ASB at the end of the teenage years may be overstated (Kratzer & Hodgins, 1999; Moffitt et al., 2002). Indeed, engagement in an antisocial lifestyle during adolescence may set in motion a host of roadblocks or snares to the types of educational,

vocational, and social experiences needed for optimal development.

Recent media attention to horrific acts of middle-class violence (e.g., the U.S. school massacres of the late 1990s) has suggested a different pathway to lethal violence—one marked by extreme peer victimization and scapegoating during childhood and adolescence, leading to vengeance when supported by (1) portrayals of violent models in the mass media (including the Internet) and (2) ready access to lethal weapons. Linkages between victimization, shame, and depression on the one hand, and uncontrolled rage on the other, require the serious attention of investigators and clinicians.

In all, our brief synthesis has emphasized the nature of interactive and transactional processes that begin early in life for a small subgroup of at-risk children, facilitating their development of threatening, aggressive, and antisocial patterns that constitute a major mental health and social problem for many years of their subsequent development. These youths are, in all likelihood, the “models” of antisocial responding for the far greater numbers of youth who begin to display delinquent behavior patterns in adolescence. Early intervention, and the search for factors that can promote desistance and resilience, are key goals for the field.

Closing Themes

We reiterate several central themes that have been the focus of our chapter. First, important subtypes and subcategories of the domain of ASB exist, and their recognition is essential for progress in the field. Second, these behavior patterns are multidetermined and multigenerational; breaking the cycles of aggression mediated by abuse, poverty, despair, and cultural acceptance of violence is a daunting goal. Third, causal pathways are complex and transactional: The interplay of psychobiological, psychological, familial, social-cognitive, socioeconomic, and sociocultural factors in shaping different types of ASB in different individuals is intriguing and challenging. Fourth, enhanced understanding of underlying mechanisms and of effective preventive intervention strategies is essential for individual and societal well-being.

For the future, at the level of developmental science, investigations are needed that span multiple levels of analysis (e.g., genes and behavior;

social-cognitive processes and peer/family socialization) and that span the entire life course (Tremblay, 2000). In addition, person-centered strategies should supplement variable-centered risk research paradigms, given the importance of identifying risk and protective mechanisms within validated subtypes. Intervention and prevention trials must be recognized not only for their clinical importance, but also for their ability to yield causal inferences about underlying psychopathological mechanisms (Hinshaw, in press). In all, ideological rancor must give way to informed, multidisciplinary efforts aimed at understanding, reducing, and channeling aggression and antisocial activity.

We note, in closing, that many historic issues pertinent to psychopathology tend to be cyclic in nature. For example, patterns of use and abuse of different substances have ebbed and flowed in recent years, as a function of availability, cost, shifting legal strictures, and the like. It is therefore conceivable that rates of violence and antisocial activity, which have precipitously increased in recent decades but which have leveled off and even declined during the 1990s, will again increase as the new millennium opens with a failing economy and the threat (and reality) of worldwide terrorism. Along this line, the ever-growing portrayal of violence in the public media, the increasing rates of blended families, and still-easy access to dangerous weapons in our society may also portend an increase in violence. Furthermore, following Moffitt's (1993) analysis, the disparity between biological and psychosocial maturity in our culture is likely to widen rather than narrow in future years, as a function of earlier physical maturity in an increasingly technological age. Such trends presage continuing escalations in adolescent-onset antisocial activity, particularly in societies with ever-widening gaps between the wealthiest and poorest segments of the population, and particularly as the earth's population reaches critical levels. It is also conceivable that the constellation of teratogenic and perinatal factors, disrupted attachments, and poor educational preparation that accrue to ever-escalating numbers of stressed, impoverished families will also propel an increase in multiproblem youths with early-onset ASB. Overall, to reiterate our closing words in 1996, it is not the time to rest on the laurels of the field's quite real scientific gains of recent decades, but rather to redouble scientific and policy-related efforts.

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NOTES

1. The term “undercontrolled” may be a misnomer, in the view of Block and Gjerde (1986), who contend that a disruptive, aggressive behavioral style may be associated with either an undercontrolled (impulsive) or an overcontrolled (planful, psychopathic) cognitive structure.

2. In addition, regarding the realm of attention deficits/hyperactivity, research has converged on the finding of a fundamental distinction between inattentive–disorganized and impulsive–hyperactive behaviors (see also Barkley, Chapter 2, this volume).

3. In adult psychopathology, for example, the ambiguously defined nature of Axis II personality disorders leads to extremely high rates of “comorbidity,” signified by the ascription of multiple personality disorders to the same individual. Such overlap of disorders may in part be an artifact of a lack of coherence of the definitional criteria.

4. Richters (1992) provides thoughtful commentary on the nature of the association between mothers’ depression and their often-noted tendency to rate their own children at high levels on scales measuring externalizing tendencies. Whereas definitive results await better-designed investigations, it appears that, rather than reflecting distorted or biased ratings, the linkage may well reflect accurate detection by mothers of independently corroborated acting-out behavior.

5. Space permits only brief mention of another seminal set of works regarding parent socialization and child aggression—namely, those by Wahler and colleagues. Over many years Wahler has emphasized the roles of maternal coercion and maternal attention/neglect in shaping aggressive behavior (e.g., Wahler & Dumas, 1987), with important consideration of such social-ecological variables as maternal isolation/insularity and family stress (e.g., Wahler & Hann, 1987; Wahler & Dumas, 1989). Wahler’s work provides an important counterpoint to the seminal model of Patterson.

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