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Comorbidity of Attention-Deficit/Hyperactivity Disorder and Early-Onset Conduct Disorder: Biological, Environmental, and Developmental Mechanisms

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Among boys, about one-third who exhibit severe attention-deficit/hyperactivity disorder (ADHD) in preschool follow a developmental trajectory to early-onset conduct disorder (CD) in later childhood and adolescence. Moreover, the vast majority of adolescent boys with early-onset CD also meet criteria for ADHD. Although trait impulsivity, a predisposing vulnerability to both ADHD and CD, is about 80% heritable, environmental risk factors play an important role in how impulsivity is expressed, including whether young children with ADHD eventually develop CD. In this article, we (a) describe how environmental risk potentiates early-onset conduct problems among trait-impulsive and therefore vulnerable individuals and (b) outline implications for conceptualizations of externalizing comorbidity. Although other pathways to CD exist, we focus on what is likely to be a common developmental trajectory to this costly psychiatric condition.

Key words: **ADHD, biology, comorbidity, conduct disorder, environment** ***[Clin Psychol Sci Prac 17: 327–336, 2010]***

In the three decades since publication of the *DSM-III* (American Psychological Association, 1980), which did away with decision trees limiting psychiatric diagnoses to one per individual, research on comorbidity has exploded. Nowhere is this more evident than in the literature on externalizing behavior disorders. A recent Medline database search (1/21/10) using the terms "comorbidity," "ADHD," and "conduct disorder" yielded 513 published studies. Given the complexity of this literature, we cannot provide even a cursory review. Rather, in anticipation of the forthcoming *DSM-V*, we briefly consider the developmental pathway from the *hyperactive/impulsive and combined* subtypes of attention-deficit/hyperactivity disorder (ADHD) to *early-onset* conduct disorder (CD). We do not consider the purely *inattentive* subtype of ADHD, which may be etiologically distinct, has different neural underpinnings, and is not part of the heterotypically continuous pattern of comorbidity discussed later (Gatzke-Kopp & Beauchaine, 2007; see also Diamond, 2005). Furthermore, we do not consider comorbidity of ADHD with *adolescent-onset CD*, which is distinct from childhood-onset CD and is also not part of the heterotypically continuous pattern of comorbidity discussed later (see, e.g., Moffitt & Caspi, 2001). Thus, in this article we discuss the specific pathway from early hyperactivity/impulsivity—expressed as ADHD—to early-onset CD. In the sections to follow, we (a) provide a description of comorbidity among early-onset externalizing disorders in general, focusing on trait impulsivity as a common predisposing vulnerability; (b) outline advantages of etiological models that specify biological

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vulnerabilities, environmental risk factors, and their interactions in shaping heterotypically continuous patterns of externalizing conduct among impulsive children; and (c) discuss limitations of focusing solely on behavioral symptoms in conceptualizing comorbidity.

COMORBIDITY OF EXTERNALIZING DISORDERS

As most readers are undoubtedly aware, comorbidity rates—both concurrent and lifetime—are exceedingly high for externalizing behavior disorders including ADHD, oppositional defiant disorder (ODD), and CD (see Angold, Costello, & Erkanli, 1999; Hinshaw, 1987; Maughan, Rowe, Messer, Goodman, & Meltzer, 2004). Furthermore, a sizable proportion of those who experience these conditions early in childhood suffer from antisocial tendencies and substance abuse and dependencies in adolescence and early adulthood (e.g., Kuperman et al., 2001; Myers, Stewart, & Brown, 1998). In fact, delinquent adult boys usually traverse a developmental pathway that begins with severe hyperactive/impulsive behaviors as early as toddlerhood, followed by ODD in preschool, early-onset CD in elementary school, substance use disorders (SUDs) in adolescence, and antisocial personality in adulthood (see, e.g., Loeber & Hay, 1997; Lynam, 1998). Thus, although continuity in externalizing conduct is common among those on an antisocial trajectory, the behavioral phenotype varies considerably across development (Hinshaw, Lahey, & Hart, 1993), a phenomenon referred to as *heterotypic continuity* by developmental psychopathologists. Although other routes to conduct problems exist (see, e.g., Gatzke-Kopp & Shannon, 2008), we focus on the heterotypically continuous pathway from ADHD early in life to childhood-onset CD, which often marks the beginning of life-course persistent antisociality (e.g., Moffitt & Caspi, 2001).

Behavioral genetics studies indicate that comorbidity among externalizing disorders results largely from heritable mechanisms. Across several large twin samples of children and adults, a common latent factor accounts for much of the covariation among ADHD, ODD, CD, and related externalizing constructs (e.g., Krueger et al., 2002; Tuvblad, Zheng, Raine, & Baker, 2009; Young, Stallings, Corley, Krauter, & Hewitt, 2000). This factor is impressively heritable, with additive genetic effects (h^2) approaching and sometimes exceeding 0.80. As we have described elsewhere (Beauchaine, Klein, Crowell, Derbidge, & Gatzke-Kopp, 2009; Beauchaine & Neuhaus, 2008), the vulnerability underlying this factor might best be described as trait impulsivity—expressed early in life as hyperactive-impulsive symptoms of ADHD[1]—which predisposes to disorders across the externalizing spectrum as affected individuals mature (see also Young et al., 2000).

Yet even though ADHD is highly heritable (see, e.g., Price, Simonoff, Waldman, Asherson, & Plomin, 2001), whether it develops into more severe conduct problems depends considerably on exposure to potentiating environmental risk factors. Indeed, a fundamental principle in developmental psychopathology is that predisposing vulnerabilities may not be linked closely to risk factors that maintain, exacerbate, or attenuate risk (see Beauchaine & Hinshaw, 2008; Hinshaw, 2008). For example, progression from childhood ADHD to early-onset CD is accounted for in part by ineffective/coercive parenting (see Meier, Slutske, Heath, & Martin, 2009; Patterson, DeGarmo, & Knutson, 2000). Furthermore, children with ADHD who experience maltreatment are more likely than those who do not to develop SUDs later in life (De Sanctis et al., 2008). In adolescence, exposure to neighborhood violence and criminality potentiates delinquency among impulsive boys (Lynam et al., 2000; Meier, Slutske, Arndt, & Cadoret, 2008). Similarly, adolescent initiation of alcohol use is determined in large part by environmental risk, even though maintenance of drinking is affected more by genetic vulnerabilities (see Viken, Kaprio, Koskenvuo, & Rose, 1999). Finally, aggregation of at-risk youth in treatment settings often increases their antisocial behavior through exposure to deviant peers (Dishion, McCord, & Poulin, 1999). Thus, a picture emerges in which accumulating environmental risk exposure facilitates progression along the externalizing spectrum among trait-impulsive, genetically vulnerable individuals. Consistent with this interpretation, recent findings from genome-wide association studies indicate no additional genetic burden for children with ADHD+CD compared with those with ADHD alone (e.g., Anney et al., 2008). Despite conflicting findings from other methods (e.g., Zhou et al., 2008), if these

association studies are correct, the emergence of CD among children with ADHD is almost certainly promoted by environmental influences.

Although impulsive and therefore vulnerable individuals are especially likely to develop CD in high-risk environments, it is important to note that the converse also holds: impulsive children who are raised in protective environments are at lowered risk of developing more serious externalizing conduct. For example, Lynam et al. (2000) demonstrated that impulsive boys—as defined both behaviorally and through their performance on various psychological tests—were at no more risk for delinquency than their nonimpulsive peers when reared in high-SES, low-criminality neighborhoods. In contrast, similarly impulsive boys were at alarmingly high risk for delinquency—including serious crimes such as theft and robbery—when reared in high-criminality neighborhoods. These findings have since been replicated in a large population-based study (Meier et al., 2008). Thus, although high-risk environments potentiate delinquency among impulsive individuals, protective environments promote desistence from heterotypically continuous externalizing trajectories.

A model of externalizing conduct that accommodates correlations and interactions among heritable vulnerabilities and environmental risk factors may help to explain several observations. For example, a number of research groups have described difficulties in recruiting children/adolescents who meet clinical criteria for CD yet do not exhibit elevated ADHD scores (e.g., Gatzke-Kopp et al., 2009; Klein et al., 1997). If ADHD and CD are separate disorders, this should not be the case. However, when trait impulsivity is viewed as a core predisposing vulnerability to CD, such findings are expected. In other words, even though only a plurality of boys with early trait impulsivity/ADHD progress along a CD trajectory, the majority of boys with early-onset CD exhibit core features of ADHD, with many if not most meeting diagnostic criteria.[2] In addition, psychostimulants, which act on reward-sensitive brain structures that are functionally compromised in both ADHD and CD (Gatzke-Kopp et al., 2009), consistently reduce impulsivity *and* reactive forms of aggression (see Hinshaw, 1991).

MOLECULAR GENETIC AND NEURAL SUBSTRATES OF IMPULSIVITY

Given their respective scopes, literatures addressing the molecular genetic and neural bases of impulsivity cannot be reviewed here. However, we have summarized these literatures elsewhere (Beauchaine et al., 2009; Gatzke-Kopp & Beauchaine, 2007), and they point toward dopaminergic and to a lesser extent serotonergic vulnerabilities. Most candidate genes implicated in the expression of externalizing conduct affect dopamine (DA) neurotransmission via synthesis, reuptake, catabolism, or indirect gating mechanisms (see Beauchaine et al., 2009; Thapar, Langley, Owen, & O'Donovan, 2007). These include DA receptor (DRD2, DRD4, and DRD5) and transporter genes (DAT1), the monoamine oxidase (MAOA) gene, and the catechol-*O*-methyltransferase (COMT) gene. Collectively, these genes affect neurotransmission in (a) phylogenetically old brain systems implicated in reward responding and associative learning, including the ventral striatum, and (b) phylogenetically newer brain structures involved in error monitoring and executive functioning, including the anterior cingulate cortex (ACC).

It was once thought that vulnerability to externalizing psychopathology was conferred by excessive DA activity, particularly within the ventral striatum, which resulted in reward-seeking behavior (e.g., Quay, 1993). However, modern imaging studies point overwhelmingly toward dampened responding within, and reduced connectivity between, the ventral striatum and ACC among those with ADHD, both with and without comorbid CD (e.g., Durston et al., 2003; Gatzke-Kopp et al., 2009; Shannon, Sauder, Beauchaine, & Gatzke-Kopp, 2009; Volkow et al., 2009). Low central DA activity—particularly in striatal regions—is experienced as unpleasant (Laakso et al., 2003), which motivates affected individuals to seek external rewards to upregulate their chronically aversive mood state. However, given compromised functioning within these brain regions, large rewards are required to achieve satiating activation levels, which predisposes affected individuals to both impulsive behavior and more serious externalizing conduct in high-risk settings. Methylphenidate and other psychostimulants normalize activation levels within the striatum (Volkow, Fowler, Wang, Ding, & Gatley, 2002), thereby reducing reward-seeking

behaviors and impulse-control problems, as well as improving attentional performance.

Following from this discussion, the theoretical model that guides our research on the development of early-onset conduct problems is summarized in Figure 1. Genetic vulnerability to impulsivity/ADHD appears at the top, including a number of susceptibility loci, all of which affect DA neurotransmission (including the serotonin transporter that modulates both serotonin and DA activity). The left side of the figure depicts a series of high-risk environments, beginning with family coercion early in life and extending to deviant peer group affiliations, neighborhood violence, and exposure to criminality and drug use. Individuals confronted with these accumulating risk factors are likely to develop comorbidity of ADHD and early-onset ODD/CD in childhood, with an increased likelihood of emerging antisocial personality traits later in life. In contrast, the right side of the figure depicts a series of protective environments, beginning with skilled parenting and extending to healthy peer group affiliations and neighborhood cohesion. Although afflicted with ADHD given its strong heritability, individuals in these environments are far less likely to develop more severe externalizing conduct problems.

IMPLICATIONS FOR MODELS OF COMORBIDITY AND THE STUDY OF EXTERNALIZING PSYCHOPATHOLOGY

As noted by others (e.g., Angold et al., 1999; Klein & Riso, 1993), a number of different sources of

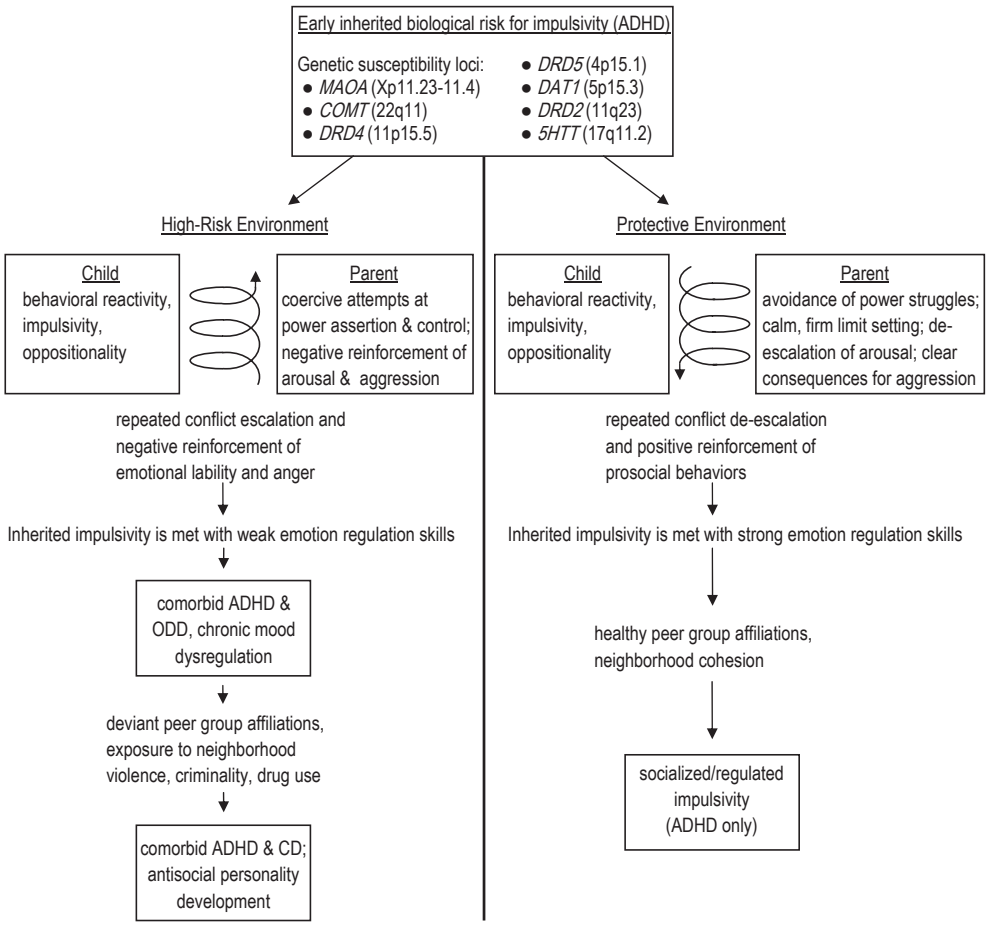

Figure 1. A Biological Vulnerability × Environmental Risk interaction model of heterotypic continuity and comorbidity of externalizing disorders, including attention-deficit/hyperactivity disorder (ADHD) and early-onset conduct disorder. On the left, genetic vulnerability interacts with environmental risk to produce lifelong antisocial behavior. On the right, a series of protective environments buffers vulnerable children with ADHD from developing more serious externalizing disorders. Adapted from Beauchaine, Gatzke-Kopp, and Mead (2007), Copyright 2007 Elsevier. Adapted with permission.

comorbidity—some "real" and some artifactual—can be identified. Two that merit discussion here are (a) heterotypic continuity and (b) primary versus secondary disorders. As we have already seen, for heterotypically continuous disorders, a single trait can manifest differently across development. In the case of early-onset externalizing spectrum disorders, genetically transmitted propensities toward impulsivity confer lifelong vulnerability to psychopathology, but the topography of behavior changes across childhood, adolescence, and adulthood, varying systematically in protective versus amplifying environments.

Given the role of trait impulsivity in the etiology of early-onset externalizing spectrum disorders, certain questions emerge over the practice of parsing symptoms into discrete diagnostic entities such as ADHD and CD. On the one hand, considering ADHD and CD as distinct disorders provides a degree of criterion-related validity, as CD portends greater functional impairment and risk for violence and criminality. On the other hand, assuming the two disorders are always discrete can obscure the etiological connections outlined previously. Indeed, shared genetic susceptibilities, common neural vulnerabilities, studies of environmental risk factors, and impressive longitudinal work by Moffitt and others suggest considerable continuity in early-onset externalizing behavior. However, when parsed based solely on symptoms and validated against differential cross-sectional correlates, distinctions between ADHD, ODD, and CD may become reified—even when the conditions are connected etiologically. We return to this issue later.

In comorbidity research, it is often assumed—sometimes explicitly and sometimes implicitly—that diagnostic co-occurrence results from distinct disorders with *different* etiologies (see, e.g., Kopp & Beauchaine, 2007). As we have seen, however, although environmental influences on etiology may differ across externalizing syndromes, genetic and neural etiological factors may be rather unified among early-onset individuals. Nevertheless, we have traditionally treated externalizing disorders such as ADHD and CD as distinct. There was merit to this perspective several decades ago, because the assumption that all "externalizing" behaviors were indistinguishable obscured more specific cross-sectional and longitudinal associations, which slowed progress in the field (e.g., Hinshaw, 1987, 1992). For example, in childhood, there is little specificity in linkages between aggressive conduct problems and academic underachievement; the more specific predictive association is between attention problems and indicators of school failure (Hinshaw, 1992).

However, given current knowledge, routinely separating externalizing syndromes may serve to fractionate the scientific literature (see Beauchaine, Neuhaus, Brenner, & Gatzke-Kopp, 2008). Investigations into ADHD have traditionally focused largely on heritability, genetics, and cognitive functioning, whereas reports on early-onset CD have focused much more on family, peer, and neighborhood processes. A similar fractionation is evident in treatment and treatment development. Consider an adult who presents for treatment of substance dependence. His or her specific SUD is almost certain to be treated as the primary condition, even if predated by severe externalizing conduct. In fact, if ADHD or CD or both are assessed at all, they are likely to be considered as comorbidities, not primary disorders, and they are likely to go untreated. In this manner, we often overlook essential aspects of etiology—which may adversely affect translation into more effective interventions (see Beauchaine & Marsh, 2006).

It is instructive to compare this approach to practices in medicine, where successive syndromes arising from a common vulnerability are rarely treated as distinct disorders. For example, as with externalizing disorders, type 2 diabetes is largely heritable (e.g., Medici, Hawa, Ianari, Pyke, & Leslie, 1999), but its course is affected strongly by environmental influences. The disease manifests differently over time, beginning with milder symptoms including frequent urination, thirst, and weight loss, and progressing—usually across many years—to life-threatening conditions including heart disease, renal failure, blindness, and eventual loss of limbs through amputation. Again, as is the case with externalizing disorders, many of those who are vulnerable genetically do not progress along the "spectrum" of illness, especially in protective environments. Yet in contrast to mental disorders, regardless of how far along the illness progresses, type 2 diabetes is *always* considered the primary condition. Thus, if and when renal failure

occurs, it is a secondary condition, and it does not change the primary diagnosis. Furthermore, although renal failure must be treated, this does not occur at the expense of treating the primary condition, type 2 diabetes. Of course treatment progresses differently if the cause of renal failure is not diabetes.

A likely explanation for the difference in conceptualization and treatment of disorders in mental health versus medical settings is that the etiologies of diseases such as type 2 diabetes are relatively well understood (see Beauchaine & Marsh, 2006). In contrast, the etiologies of psychopathology often are not. As described previously, however, the etiology of early-onset externalizing disorders—at least for many affected individuals—is becoming far better understood. Accordingly, conceptualizing successive manifestations of early-onset externalizing conduct as secondary to trait impulsivity might better reflect reality than our current diagnostic system, which specifies different externalizing disorders based on successive, cross-sectional topographies of symptoms. For this reason and others, some have suggested replacing the *DSM* with a dimensional approach to diagnosis that specifies a limited number of traits—including impulsivity—that interact with environmental risk to potentiate psychopathology (e.g., Clark, 2005; Watson, Clark, & Chmielewski, 2008).

REIFYING DISTINCTIONS AMONG EXTERNALIZING SYNDROMES

Despite accumulating evidence for a heterotypically continuous spectrum of early-onset externalizing psychopathology, considerable effort continues to be expended toward differentiating among externalizing syndromes. Three approaches to doing so include (a) partialling the effects of one disorder from another to determine which contributes independently to future adverse outcomes, (b) conducting genetic association studies to search for genes that are distributed unequally across externalizing disorders, and (c) factor analyzing symptom criteria to extract separate latent constructs that may link to different disorders. Unfortunately, such studies are often misinterpreted or overinterpreted or both, further reifying distinctions among externalizing syndromes.

Misapplication of Statistical Control

The assumption—whether implicit or explicit—that ADHD and CD are always distinct or etiologically dissociable has led to a large number of studies in which symptoms are parsed using analysis of covariance (ANCOVA) or related multiple regression techniques to determine which disorder or symptom cluster "explains" current or future functioning. For example, several authors have explored predictive associations between externalizing disorders and future SUDs. As noted by Elkins, McGue, and Iacono (2007), links between ADHD and future substance use often disappear when symptoms of CD are "controlled" statistically. Yet how we interpret such findings depends on whether we view ADHD and CD as independent disorders versus stemming from a common heritable vulnerability. If we believe that ADHD and CD are independent, we must conclude from the results of these studies that only CD confers risk for SUDs, the typical interpretation.

However, because ADHD and early-onset CD are often not independent etiologically, this is a clear misuse of ANCOVA. Indeed, in such analyses, the common heritable mechanism tying ADHD to CD has been removed statistically, creating a "trait" that does not exist in reality (i.e., ADHD without externalizing vulnerability; see Figure 2). In their incisive critique outlining misapplications of ANCOVA, Miller and Chapman (2001) considered a parallel problem in research on internalizing disorders, concluding that

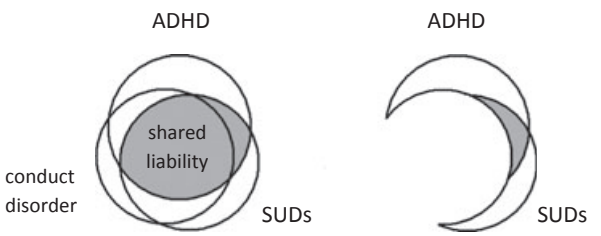

Figure 2. Interrelations among attention-deficit/hyperactivity disorder (ADHD), early-onset conduct disorder (CD), and substance use disorders (SUDs) for those on a heterotypically continuous externalizing trajectory (left panel). Shared liability for ADHD and SUDs is indicated by the gray shaded region. The right panel depicts residual overlap between ADHD and SUDs, with shared liability to CD removed (i.e., partialled out). Given common heritable risk for all three conditions, such partialling leads to incorrect conclusions regarding causal relations among disorders (see text).

''statistical methods cannot remove the 'effect' of anxiety from depression if conceptually they are overlapping constructs'' (p. 44). Using ANCOVA in this manner artificially obscures meaningful etiological connections between early impulsivity and downstream externalizing outcomes. Furthermore, given the temporal sequence of syndrome expression (ADHD → CD → SUDs), patterns of autocorrelation (the phenomenon by which temporally proximal measures of the same construct correlate more strongly than temporally distal measures) virtually guarantee better prediction of SUDs by CD than by ADHD—*even if* ADHD and CD share a common etiological substrate.

Overinterpreting Genetic Association Studies

A second issue that may obscure etiological connections among externalizing disorders stems from overinterpreting findings from and misunderstanding limitations of genetic association studies. For example, it is common in psychiatric genetics to compare patterns of gene expression across externalizing groups in association studies (e.g., ADHD vs. ADHD+CD). Resultant group differences are then interpreted as supporting the diagnostic distinction used, even when the genes identified bear little or no theoretical relevance to underlying disease processes. This practice is especially problematic given the high false positive rates and low replication rates inherent in association studies (see Lux, 2009). Furthermore, association studies are not true experiments given the absence of random assignment to groups. As a result, stratification on unmeasured third variables may explain differences in gene expression (see Neale et al., 2008). Similarly, if the phenotype is specified poorly, artifactual group differences in gene expression may emerge, leading to incorrect conclusions about diagnostic distinctions. For instance, if primarily inattentive children are allowed into an association study comparing gene expression among those with ADHD alone versus ADHD+CD, discrepancies in allelic frequency distributions may reflect different etiologies of ADHD across groups. This is because very few children with ADHD *and* CD have the primarily inattentive subtype of ADHD. Thus, primarily inattentive children will be overrepresented in the ADHD-only group, inducing spurious differences in genetic etiology.

Misunderstanding of Factor Analysis

Finally, many arguments for distinctions among externalizing constructs follow the results of factor analyses of symptoms. When such analyses yield multiple factors, evidence for dissociable externalizing syndromes is often claimed. However, as behavioral genetics studies of shared externalizing liability demonstrate (e.g., Krueger et al., 2002; Tuvblad et al., 2009; Young et al., 2000), a single *second-order* factor (impulsivity) can account largely for comorbidity among various *first-order* factors (ADHD, CD). Furthermore, the finding that several first-order factors emerge in externalizing psychopathology research in no way precludes a common etiological substrate among disorders. In fact, ordinary factor analysis is not an appropriate statistical method for identifying ''clusters,'' ''types,'' or ''diagnostic entities'' that differentiate among people. Rather, factor analysis identifies individual differences (i.e., dimensions) on which people vary (see, e.g., Waller & Meehl, 1998).

This distinction is illustrated in research on personality. Five-factor models, which emerge repeatedly in factor analytic studies, in no way suggest five *types* of people. On the contrary, one can score high or low on one or all personality dimensions, producing a vast number of individual differences. Similarly, factor analyses of externalizing behavior that identify multiple *dimensions* of conduct do not necessarily imply multiple *types* of disorders. Indeed, externalizing syndromes are highly correlated, so individuals who score high on one factor usually score high on others (e.g., Hinshaw, 1987).

CONCLUDING REMARKS

Those who study psychological and psychiatric phenomena have sometimes been dichotomized as ''lumpers'' versus ''splitters'' (e.g., Meehl, 1987). The former are apt to group together any phenomena (e.g., cognitive abilities, behavioral patterns, and emotional tendencies) with even slight linkages, whereas the latter accentuate any and all differences, however small. Which is the better strategy? There is a clear analogy here with investigations into cognitive performance or personality: Is there a single cognitive ability (i.e., ''g'') or a multitude of separable skills (e.g., Catell's 120 cognitive abilities)? Is there better predictive validity from

an overall personality trait such as neuroticism versus its potentially myriad subfacets (e.g., distress intolerance, impulsivity, and pessimism)?

The answer, ultimately, depends on the scientific framework under consideration as well as the criterion measures that are used to evaluate the validity of competing conceptual models (for a parallel on whether psychopathology is better conceptualized as dimensional versus categorical, see Pickles & Angold, 2003). Our contention here is that when development is considered explicitly, there is more continuity and overlap between the supposedly differentiable constructs of trait impulsivity (operationalized as ADHD-combined type) and aggressive conduct problems than would be assumed from the current diagnostic nomenclature. From this perspective, genes are not separable from environments but are inextricably linked with them, and strong genetic vulnerability to trait impulsivity may well be molded by contextual influences—whether high risk or protective—to shape differential outcomes. Future models of comorbidity need to consider far more than symptom overlap or independence per se. To understand etiology, maintenance, and life trajectories toward psychopathology, a broader and more conceptually based understanding of ''comorbidity'' is required.

NOTES

1. Debate over the optimal definition of impulsivity has existed for decades (see Beauchaine & Neuhaus, 2008). Alternative definitions range from very specific deficits on psychological tests to very broad response tendencies such as frequent engagement in socially inappropriate behaviors. However, based on consistently high reliabilities ($\alpha \approx 0.80$) and heritabilities ($h^2 \approx 0.80$) of ADHD scale scores, the most reliable and construct valid definition likely comprises symptoms of hyperactivity and impulsivity.
2. As noted by an anonymous reviewer, observed comorbidity rates of ADHD and CD are usually lower in epidemiological samples than in clinical samples (e.g., Angold et al., 1999; Maughan et al., 2004). To date, however, few epidemiological studies have distinguished between early-onset and adolescent-onset CD. Rather, both subtypes have been subsumed into a single CD group. However, the hyperactive/impulsive developmental pathway that we describe is specific to early-onset cases. In contrast, temperamental impulsivity is not associated with adolescent-onset CD (Moffitt & Caspi, 2001).

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