THE CHILDREN IN THE COMMUNITY
STUDY OF DEVELOPMENTAL COURSE
OF PERSONALITY DISORDER

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The Children in the Community (CIC) Study is an ongoing investigation of
the course of psychiatric disorders including personality disorders
(PDs) in an epidemiological sample of about 800 youths. In addition to
tracking developmental trajectories over 20 years from adolescence into
adulthood, the CIC Study has used prospective data to investigate early
risks for Axis II disorders and symptoms (including both environmental
factors and early characteristics), implications of comorbidity with Axis
I disorders, and associated negative prognostic risk of adolescent PDs
into adulthood. The substantial independent impact of PD on subse-
quent Axis I disorders, suicide attempts, violent and criminal behavior,
interpersonal conflict, and other problematic adult outcomes confirms
the importance of attention to these problems when they manifest in
early adolescence. The implications of study findings for potential
changes in the DSM are discussed.

The CIC Study began as a follow-up to a 1975 study of a large random
sample of children (ages 1–10 years) living in households in 100 residen-
tial areas sampled in 2 upstate New York counties. The original study was
designed to assess the level of need for children's services and validate
social indicators of that need (Kogan, Smith, & Jenkins, 1977). Data came
from maternal interviews and covered a wide range of developmental, tem-
perament, health, and environmental variables. When first followed-up in
1983, study goals shifted to focus on predictors of Axis I psychiatric disor-
ders in early adolescence (mean age = 14) (see Cohen & Cohen, 1996 for a
full description of sampling method, retention, and characteristics). Axis I
disorders were assessed in interviews with children and mothers using the
Diagnostic Interview Schedule for Children (DISC-1; Costello, Edelbrock,
Dulcan, Kalas, & Klaric, 1984). The protocol also covered theoretically and
empirically plausible risks for disorders assessed with multiple measures of
family, peer, neighborhood, and school environment. Health, personal-

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ity, attitudes, values, and behaviors were reported by both parent and child.

From this first assessment Cohen was interested in exploring the development of PDs in children and adolescents. However, despite DSM-III recognition of likely early life origins of PDs, there were no existing age-appropriate PD measures for adolescents.

PD MEASUREMENT
To avoid lengthening the interview with unnecessary repetition, we used plausible symptom indicators already available in the protocol. Most of these child- and parent-reported items covered the “normal” range as well as extremes on traits or behaviors that approximated some Axis II symptoms (e.g., distrusting others, difficulty controlling anger, reckless disregard for safety, abandonment fears, etc.). To provide coverage of remaining symptoms, Cohen selected items from the Personality Disorder Questionnaire (PDQ; Hyler, Rieder, Spitzer, & Williams, 1982) with age-appropriate revisions when needed. A few new items were written when no alternative seemed age-appropriate. DISC-1 items were not used in PD scales in order to ensure independent assessment of Axis I and II disorders.

The CIC sample was reassessed in mid-adolescence (mean age = 16). Preparation for this follow-up permitted Drs. Cohen and Schwab-Stone to augment the previous protocol with additional items better suited to the PD criteria, especially as revised for the DSM-III-R. New items were adapted from a prototype of the Structured Clinical Interview for DSM-III-R Personality Disorders (Spitzer & Williams, 1986). Bernstein reviewed all non-DISC youth- and parent-reported items to create PD symptom scales and diagnostic algorithms. This work led to the first CIC publications on PD (Bernstein, Cohen, Velez, Schwab-Stone, Siever, & Shinsato, 1993; Bezirganian, Cohen, & Brook, 1993) based on the best information available in early adolescence and more complete data from the mid-adolescent assessment.

The next data collection took place in early adulthood (mean age = 22) and was largely modeled on the earlier protocols. Although data on the developmental course of PDs was potentially available from age 9 (the youngest participants in the first assessment) to age 27 (the oldest in the young adult assessment), investigation of age changes in symptom levels required consistent measurement in repeated assessments. Furthermore, the DSM-IV came out with several changes in diagnostic criteria. These considerations led us to revise our item selection and algorithms. Working with Cohen, Jeffrey G. Johnson reviewed the parent and youth items in the protocol and produced consistent symptom scales and algorithms in all three assessments that covered almost all DSM-IV criteria. Diagnoses were made using scaled diagnostic criteria counts and standard DSM-IV thresholds.

The reassessment of the CIC sample at mean age = 33 did not include
maternal interviews, thus preventing use of the existing PD scales. New measures were developed by Thomas Crawford based on self-report items alone. All items were part of early and later adult assessments (mean ages 22 and 33) and thus constituted a consistent measure of PD across the 11 years. At age 33 a clinical assessment of PDs using the Structured Clinical Interview for DSM-IV Personality Disorders (SCID-II: First, Gibbon, Spitzer, Williams, & Benjamin, 1997) permitted evaluation of concordance between the CIC measures and the more standard assessment. Clusters A and B diagnoses based on CIC algorithms both demonstrated higher concordance with SCID-II diagnoses than most comparable self-report instruments (Crawford et al., 2005). Concordance for Cluster C diagnoses was closer to the published average. Unlike other self-report instruments, algorithms for the CIC Study do not overestimate diagnoses when compared with SCID-II clinical diagnoses. Scaled measures of PD showed surprisingly good prediction of CIC and SCID-II scales and associated disability at mean age 33 from CIC measures recorded 11 years earlier.

As would be expected, there is substantial correlation between these different versions of our measures. The most significant changes came when adolescent and young adult measures combining youth and parent reports were compared to measures using self-report alone. Agreement between corresponding PD scales was still high (mean \( r = .63 \)) and higher at the cluster level (mean \( r = .72 \)) even though parent and youth reports are often discordant. We thus have found little reason to be concerned about problems in generalizing findings from one PD version to another.

However, two related problems have become apparent over time that are not unique to this study or our assessment instruments. The first pertains to arbitrary criterion-based cutoffs demanded by the DSM. Despite the utility of diagnoses for clinical and policy purposes, we agree that on the whole diagnostic cutoffs have little empirical justification. Future taxonomic and latent class work by our group or others may examine potential empirical justification for such thresholds. A related problem occurs when individuals do not meet criteria for any single PD but nevertheless have many symptoms from a combination of PDs. Our analyses show that the adverse outcomes for these people with personality disorder not otherwise specified (PDNOS) are as serious as those in people who met full diagnostic criteria for one or more PDs (Johnson et al., in press).

The second problem is determining just when a given criterion has been met. Because symptom items have graded response options (e.g., always, most of the time, sometimes, never), there are multiple choices about which item cut-points will best define when a diagnostic criterion has been met. Response extremity and relative low endorsement rates are logically expected for a behavior or other manifestation to be considered a symptom in the general population. However, definitions of “extremity” or “low rate endorsement” will vary from one diagnostic algorithm to another. These decisions can have substantive effects on the conclusions drawn from an empirical investigation. For instance, extremity of cutoff can have large
effects on the correlations with the other variables in an analysis. We have found that this problem is minimized by analyses using scaled symptom measures as well as categorical diagnoses, with attention to consistency across empirically based conclusions.

Another issue pertains to the DSM-IV’s requirement that adolescent PD symptoms persist for at least 1 year. Most PD items in our assessment and other self-reports do not specify duration, but are phrased as descriptions of the person and presumed reasonably stable. Nevertheless, some of our analyses of long-term outcomes consider adolescents with PDs to be only those meeting or nearly meeting criteria at both mean ages 14 and 16.

SUBSTANTIVE QUESTIONS AND METHODOLOGICAL ISSUES
Given prospective data accumulated over 20 years, the CIC Study is unique in being able to address questions about developmental trajectories of adolescent PDs. The Study’s comprehensive assessment of Axis I and II disorders has also permitted investigation of the developmental significance of comorbidity between these two sets of disorders. Comorbidity estimates based on our community sample are especially valuable because they are not inflated by ascertainment bias typically associated with referred samples (Berkson, 1946). One of the drawbacks, however, is that community samples yield relatively few cases of individual PDs, thereby limiting statistical power. To increase power we often analyze PDs grouped into diagnostic clusters, generally checking to ensure that findings are not thereby distorted.

Prospective data have permitted investigation of adult outcome variables (e.g., Axis I and II disorders, social impairment, suicide, violence, etc.) that can be predicted by adolescent PDs. Moreover, CIC data have been used to show how early risk factors (e.g., childhood abuse, neglect, maladaptive parenting, school experiences) predict PDs in adolescence and adulthood. Insofar as prospectively reported data have been used, our findings were not distorted by recall bias that undermines confidence in retrospective reports. Analyses also generally include consideration of probable or established correlated risks as covariates. When testing effects for a given PD, we have learned the importance of controlling for other co-occurring disorders. For example, adolescent PD initially appeared to be predictive of early adult eating and weight problems (Johnson, Cohen, Kotler, Kasen, & Brook, 2002). However, its effect disappeared once adolescent depressive disorder and earlier eating problems were added to the model.

STUDY FINDINGS RELATED TO AGGREGATED PD SYMPTOMS OR DISORDERS
Change and Stability in Symptom Levels

We have shown that mean PD symptoms overall and virtually all disorders are consistently highest in early adolescence and are followed by an essentially linear decline from age 9 to 27 (Johnson, Cohen, Kasen et al., 2000).
Some of this decrease can be attributed to established developmental declines in impulsivity, attention seeking, and dependency. Declines undoubtedly also correspond to increases in social competence and goal-related self-control. Not everyone declined in symptoms, of course: approximately 21% of the sample showed an overall increase in PD symptoms over the decade of repeated assessment (Johnson, Cohen, Kasen et al., 2000). Nevertheless, there is a general uniformity in overall decline in symptoms (amounting in aggregate to about 1% per year) and in the corresponding diagnoses. Current data suggest that this decline ends for most PDs by the mid- to late-20s. In the assessment spanning age 28 to 38 we observed no age differences in our cohort except for declines in histrionic and narcissistic PDs.

Whether measured in adolescence or adulthood, stability coefficients for PD symptoms are highly comparable across diagnostic clusters and developmental stages. When measured with combined mother and youth reports, stability coefficients for Cluster A, B, and C disorders all ranged between .42 and .65 from mean age 13 to 16 and from mean age 16 to 22 (Johnson, Cohen, Kasen et al., 2000). Surprisingly, correlations over 9 years following early adolescence were almost as large as those over 6 years from mid-adolescence. Moreover, correlational stability in PD symptoms across adolescence (that is, stability ignoring the average age-associated decline in symptoms) is comparable to stability observed in adults. Using self-reported symptom criteria assessed at mean age 22 and 33, stability coefficients for all 3 clusters were around .55. These estimates are similar to other stability coefficients for PDs (e.g., Ferro, Klein, Schwartz, Kasch, & Leader, 1998; Lenzenwager, 1999; Trull, 1993) and fall within the .4 and .7 range typically observed for normal personality traits over comparable intervals (McCrae, Costa, & Schneewind et al., 1989).

Based on stable but declining symptoms, individuals thus maintain similar rankings in PD symptoms relative to age peers even as average levels decline over time. Recent findings, however, suggest a gradual shift in the meaning of PD symptoms during the early adult years. Individuals with the highest PD scores differ increasingly from normative symptom levels at successive assessments (Crawford et al., 2005). Thus personality disturbances represent increasing deviance as normative PD symptoms decline throughout adolescence and early adulthood. Given this broad developmental pattern of persistence and change, we have learned the importance of asking not only which risks are associated with early elevations in PD symptoms but also which factors delay or prevent symptom declines over time.

**GENERAL RISKS FOR PD DISORDERS AND SYMPTOMS**

There is relatively high comorbidity and correlation among the criteria counts for the PDs. Noting the substantial fraction of item covariance reflected in the first principal component of the symptom correlation matrix,
Cohen (1996) created an outcome variable representing the sum of all Axis II symptoms assessed in the young adult interviews. Low family SES, family welfare support, single parent, parental conflict, paternal and maternal sociopathy, and parental illness and death were each independently related to later PD symptoms. Parenting and parent-child relationships—including low closeness to mother, low closeness to father, power assertive punishment, maternal control through guilt, and having been the result of an unwanted pregnancy—were predictive of later PD symptoms. Childhood characteristics including behavior problems, social isolation, and poor health at mean age 6 also predicted young adult PD symptoms assessed some 16 years later. In adolescence, low social competence, introversion, low self-esteem, not being attractive, high emotionality, and abrasiveness predicted elevated symptoms, including those scoring two SDs above the mean. The strongest long-term predictors were earlier PD, disruptive disorder, and depressive symptoms, thus paralleling findings observed separately for each of the three clusters (Bernstein, Cohen, Skodal, Bezirganian, & Brook, 1996). Other strong predictors included low IQ, poor achievement, having been suspended or expelled from school, having repeated at least one grade, and not being goal directed (Cohen, 1996). An important point of these analyses was that alternative indices of effect size (magnitude of the predictive relationship) would lead to very different conclusions about which of these risks was more important. For example, it was first shown in these analyses and replicated in many other individual studies that a history of child abuse substantially elevated odds ratios for subsequent serious PD problems. Such a finding might lead some to the unwarranted conclusion that many or even most PDs were a consequence of childhood maltreatment. In contrast, these analyses show that other childhood characteristics and environments accounted for much higher fractions of the cases and higher proportions of the variance in the symptom sum.

These findings were elaborated in analyses showing that maladaptive parenting behaviors significantly increased the risk for “any PD” in early adulthood (Johnson, Cohen, Kasen, Smailes, & Brook, 2001) independently of earlier childhood difficult behavior and psychiatric disorder. Parental psychiatric disorder also predicted offspring PD in early adulthood but not independently of maladaptive parental behaviors. The maltreatment effect, in contrast, remained significant even when parental disorder was included in the model.

COMORBIDITY WITH AXIS I DISORDERS
Although the high comorbidity with Axis I disorders at any given age has not been the focus of our scientific reports on PD, it has been reported incidentally in many publications. In general comorbidity with depressive, anxiety, and disruptive disorders was high for all three PD clusters, whether indexed by symptoms or by diagnosis. New onset of disorders is
a more complex question, complicated in this study by the gaps in assessment intervals over which disorders were identified. Assessment of disorders was generally confined to the year prior to interview, a strategy that improves the validity of the assessment according to a long history of empirical research. Nevertheless, this strategy leaves gaps in the life history record.

To facilitate consideration of their relevance to potential diagnostic system changes, the following summary of findings is organized by diagnostic cluster. For each cluster we examine specific findings on stability and trajectory, comorbidity, prospective risks, and dysfunctional outcomes.

Cluster A PDs

Stability and Trajectory. Much of the existing information on the course of Cluster A disorders (paranoid, schizoid, and schizotypal PDs) comes from studies that follow relatives of people with schizophrenia (Kendler et al., 1993) because individuals with Cluster A disorders rarely present for treatment and may be atypical when they do (Loranger, 1990). Most reports involve people with schizotypal or schizoid PDs and few if any studies have tracked the course of paranoid PD. Although Cluster A disorders in adult clinical samples show a more chronic or even increasing symptomatic course (Paris, 2003), data from our community sample show declining average symptoms across adolescence and early adulthood. As noted above, the increasing deviance from the norm of those whose symptoms persist or increase is syntonic with findings based on clinical samples.

In the CIC Study combined youth and parent reports showed that stability of Cluster A symptoms equaled .57 from mean age 13 to 16 and dropped to .49 across the longer interval from mean age 16 to 22 (Johnson, Cohen, Kasen et al., 2000). From mean age 22 to 33, stability of symptom criteria based on self-report was .56. Overall, adolescent paranoid symptoms were more stable than other Cluster A symptoms. Paranoid and schizotypal symptoms were both more stable than schizoid symptoms in adulthood.

Comorbidity with Axis I Disorders. Given criteria for Cluster A PDs, one primarily expects diagnostic overlap with schizophrenia and other psychotic illnesses that are low in prevalence and usually emerge in late adolescence or adulthood. As such, we did not expect to be able to detect significant comorbid psychotic disorders in this sample. We documented instead that adolescents with stable Cluster A disorders (i.e., diagnosed in both adolescent assessments) had elevated rates of other Axis I disorders—35% had disruptive disorders, 25% had anxiety disorders, and 20% had depressive disorders (Kasen, Cohen, Skodol, Johnson, & Brook, 1999). Comorbid Cluster A and Axis I disorders in adolescence are noteworthy because of sharply increased rates of Cluster A disorder persistence into adulthood (Kasen et al., 1999). Odds of a Cluster A disorder in early adulthood were 24.6 times higher when adolescent Cluster A disorders and disruptive disorders co-occurred. When adolescent Cluster A and depressive
disorder co-occurred, odds ratios were 20.5 times higher for Cluster A PDs in young adults. When Cluster A and anxiety disorders co-occurred, the odds of a Cluster A diagnosis in early adulthood were 16.9 times higher. Moreover, this risk for adolescent Cluster A disorders persisting into adulthood was four times greater than the risk for Cluster B and C disorders extending into adulthood when they co-occurred with anxiety disorders.

More specifically, adolescent Axis I disorders were associated with later schizoid and paranoid PDs in early adulthood (Kasen et al., 2001). Disruptive behavior disorder in adolescence was associated with a six-fold increase in risk for schizoid PD in early adulthood even after earlier schizoid problems were taken into account. Anxiety disorder in adolescence quadrupled the risk for paranoid PD in early adulthood and its effect was independent of earlier paranoid PD. These findings were observed after taking co-occurring Axis I disorders, childhood maltreatment, and family adversity into account.

**Prospective Risk Factors.** For young adult participants who reported prior experiences of childhood sexual abuse, the odds of a Cluster A diagnosis were significantly increased (Cohen, Brown, & Smailes, 2001). Moreover, growth curves across adolescence and early adulthood showed that developmental trajectories for Cluster A symptoms were significantly higher in abused participants than in those who reported no abuse. (However, these analyses did not control for co-occurring PDs, especially borderline PD, thus leaving it uncertain whether childhood sexual abuse predicts Cluster A disorders independently of other PDs in early adulthood.) Childhood verbal abuse predicted elevated rates of Cluster A PD in prospective analyses even after controlling for all other types of abuse and co-occurring psychiatric disorders (Johnson, Cohen, Smailes et al., 2001). Childhood verbal abuse also predicted elevated rates of paranoid PD net of other forms of abuse and co-occurring disorders.

**Prospective Outcomes of Cluster A.** Adolescent Cluster A disturbances often represent the prodrome for schizophrenia spectrum illnesses; thus their developmental trajectories may be particularly problematic in the late second and third decades of life. Consistent with this expectation, we found the greatest functional delay in the transition to adulthood among adolescents with high Cluster A symptoms (Cohen et al., 2005). In detailed narratives describing how individuals function in different roles and social settings from age 17 to 27, these adolescents had lower trajectories of education and achievement and abnormal trajectories in other roles, including childbearing.

Adolescent Cluster A disorders increased the risk of subsequent mood disorders and disruptive behavior disorders in early adulthood net of co-occurring Axis I and Axis II disorders (Johnson et al., 1999). As noted below, however, increased risk for these Axis I disorders was not unique to Cluster A. In a separate study, adolescent Cluster A disorders and symptoms prospectively predicted violent acts and criminal behavior (Johnson, Cohen et al., 2000). More specifically, paranoid symptoms were indepen-
dently associated with subsequent violent acts and criminal behavior. Paranoid cognitions may mobilize aggressive behaviors that are secondary to the perceived threats and suspiciousness that characterize both paranoid and schizotypal PDs. Alternatively, paranoid ideation and aggressive behavior may mutually reinforce each other and thus lead both to persist at elevated levels over time.

Adolescent Cluster A disorders also doubled the risk for anxiety disorders in early adulthood net of co-occurring adolescent Axis I and II disorders (Johnson, Cohen, Skodol et al., 1999). Interestingly, once these covariates were taken into account, a similar effect was not observed in Cluster B or C disorders. This long-term association with anxiety may reflect perceived threat that prevents people with Cluster A disturbances from seeking or accepting treatment. Clinical interventions ignoring this aspect of Cluster A disorders may be unlikely to alleviate the distress and impairment associated with these PDs.

**Interpersonal Dysfunction.** In an unexpected finding, we observed that adolescents with Cluster A disorders spent a relatively high percentage of time involved in romantic relationships and were more likely to be teenage parents than age peers without PD (Chen et al., 2004; Cohen et al., 2005). Partner conflict also was higher in youth with elevated Cluster A symptoms up until age 23, perhaps reflecting interpersonal deficits and in some cases stress associated with early parenting (Chen et al., 2004). These outcomes are consistent with other evidence of early parenthood among the more severely mentally ill. However, there were indications that some young people with high symptoms in adolescence found a more adaptive niche in early adulthood that permitted a more timely assumption of adult roles and a subsequent decline in symptoms (Cohen et al., 2005) as well as a decline in partner conflict after age 23 (Chen et al., 2004).

### Cluster B PDs

Most of our studies of adolescent Cluster B disorders have investigated borderline, histrionic, and narcissistic PDs. Antisocial PD was not included with other Cluster B disorders because the DSM does not recognize this disorder until age 18. Also, we generally have not focused on antisocial PD because a wealth of information about childhood precursors and developmental trajectories for this disorder already exists elsewhere in the literature. Nevertheless, it is increasingly our view that stable childhood conduct disorder is best viewed as a disorder of personality.

**Stability.** The stability of adolescent Cluster B symptoms was .65 from mean age 14 to 16 (Johnson, Cohen, Kasen et al., 2000). Stability dropped to .50 across the longer interval from mean age 16 to 22. In more recent data, the corresponding stability coefficient (when antisocial symptoms were excluded) was .55 from mean age 22 to mean age 33. Stability coefficients for symptoms of specific Cluster B disorders are somewhat lower.
across these age ranges and generally averaged about .40. Narcissistic symptoms, however, had notably elevated stability from mean age 14 to 16 \( r = .57 \) (Johnson, Cohen, Kasen et al., 2000).

Crawford, Cohen, and Brook (2001a) used latent variables in structural equation models to examine the stability of Cluster B symptoms in a subsample of youth ages 10 to 14. When latent variables reflecting the “true” variance in Cluster B symptoms were estimated, the stability over 2½ adolescent years was notably higher \( r = .73 \) than unadjusted stability estimates reported above. Across 9 years stability was .63 and .69 in adolescent boys and girls, respectively. Crawford et al. (2001a) also contrasted latent Cluster B symptoms with latent variables for internalizing symptoms (depression and anxiety) and externalizing symptoms (ADHD, oppositional defiant, and conduct disorder) associated with Axis I disorders. The stability of internalizing and externalizing symptoms each were consistently lower than Cluster B symptoms, thereby supporting expectations that PD symptoms should be more enduring than episodic disturbances normally assigned to Axis I.

Hamigami, McArdle, and Cohen (2000) estimated the reciprocal effects of narcissistic and borderline symptoms on each other from early adolescence into the mid-20s. A dynamic growth curve analysis of data from age 9 to 27 showed that borderline symptoms predicted subsequent increases in narcissistic symptoms, whereas narcissistic symptoms predicted lower subsequent borderline symptoms. One interpretation of this finding is that borderline problems are more maladaptive and thus inhibit the expected developmental decline in narcissism. Narcissistic symptoms, on the other hand, decline sharply throughout this age period and may be indicators of a more benign developmental delay.

**Comorbidity with Axis I Disorders.** In the first three successive assessments, cross-sectional associations were found between latent variables for Cluster B symptoms and externalizing symptoms (ranging from .37–.68), and for Cluster B symptoms and internalizing symptoms (ranging from .15–.61) (Crawford et al., 2001a). In models assessing comorbidity over time, correlations appear to be due to common causes and a causal effect of early Cluster B symptoms on subsequent externalizing symptoms in middle adolescence (Crawford, Cohen, & Brook, 2001b). For girls there was more evidence of reciprocal effects as well as common causes, with externalizing symptoms predicting elevated Cluster B symptoms in late adolescence. For girls internalizing symptoms in early adolescence also significantly predicted increased Cluster B symptoms in middle adolescence.

At the diagnostic level, a full 47% of the adolescents with Cluster B disorders also had a comorbid disruptive behavior disorder (Kasen et al., 1999). The strong association between these disorders in adolescence is theoretically compelling, especially given how antisocial PD is classified as a Cluster B disorder in adulthood. Kasen et al. (1999) also observed that 38% of adolescents with Cluster B disorders had an anxiety disorder and 28% had a depressive disorder.
Prospective Risk Factors. Given the high salience of parent-child relationships in early adolescence, Bezirganian et al. (1993) investigated earlier disturbances in maternal and paternal parenting as predictors of Cluster B PD observed later in adolescence. Maternal inconsistency significantly predicted offspring borderline PD, and maternal overinvolvement predicted offspring histrionic PD. These analyses suggest that these mothering behaviors may mediate effects of maternal borderline symptoms (as assessed by a measure of ego integration).

As with other PDs, Cluster B disorders in our community sample have been clearly linked with experiences of childhood abuse. Uniquely, elevated symptoms of borderline PD in early adulthood were predicted by official records of sexual abuse (Johnson, Cohen, Brown, Smailes, & Bernstein, 1999). At the cluster level, risks for Cluster B disorders in young adulthood were 14.5 times higher among those with self-reported or recorded childhood sexual abuse, for whom the expected declining symptom trajectory was delayed (Cohen et al., 2001). Childhood sexual abuse also predicted elevated trajectories of anxiety, depressive, and disruptive behavior disorders in early adulthood, perhaps thus contributing in part to the overlap between these Axis I disorders and Cluster B disorders.

Official records of childhood physical abuse were also associated with elevated Cluster B disturbances in adulthood (Cohen et al., 2001; Johnson, Cohen, Brown et al., 1999). However antisocial PD was the only Cluster B disorder showing such a relationship independently of co-occurring disorders. Physical neglect increased odds of young adult borderline and narcissistic disorders (Johnson, Smailes, Cohen, Brown, & Bernstein, 2000) and elevated symptoms of antisocial PD as well (Johnson, Cohen, Brown et al., 1999). Verbal abuse assessed in childhood predicted elevated borderline and narcissistic PD even net of effects of other types of abuse and of co-occurring psychiatric disorders (Johnson, Cohen, Smailes et al., 2001).

Adolescent school settings characterized by high levels of academic aspirations and achievement reduced the odds of antisocial PD in early adulthood and thereby offset some of the effects of harsh parental punishment, deviant peer relationships, and earlier conduct problems (Kasen et al., 1998).

Odds of adult Cluster B diagnoses were 12.5 times higher when stable adolescent Cluster B disorders co-occurred with disruptive disorder (Kasen et al., 1999). More specifically, adolescent disruptive behavior disorders increased risks for narcissistic and antisocial PD in early adulthood (Kasen et al., 2001). Major depression in adolescence increased risks for antisocial PD in early adulthood even after taking earlier disruptive behavior and anxiety disorders into account. When adolescent depressive disorder co-occurred with Cluster B disorders, the risk for subsequent Cluster B disorders was 19.1 times higher (Kasen et al., 1999). Comorbidity with anxiety disorders in adolescence resulted in a four-fold increase in risk for Cluster
B disorders in adulthood. In these analyses antisocial PD was included in Cluster B disorders in young adulthood but not in adolescence.

**Prospective Outcomes of Cluster B.** Systematic information on the course of narcissistic PD based on clinical samples or nonclinical samples is extremely sparse (Paris, 2003). In our community sample, mean levels of narcissistic symptoms showed the greatest relative decline (73%) of all the PDs we analyzed from ages 9 to 27 (Johnson, Cohen, Kasen et al., 2000). In contrast to most other PDs, narcissistic symptoms continued to decline between ages 28 and 38. At the symptom level, idealized fantasies about success and romance continued to subside across this age range as did haughty and arrogant attitudes. In normative terms, life experience and maturation probably lead people to gradually accept more realistic appraisals of themselves and their prospects, perhaps fostering greater humility as well.

When romantic relationships gain importance in adolescence and early adulthood, the negative influence of Cluster B disturbances becomes increasingly apparent. Consistent with Erikson’s (1950, 1968) developmental theory, identity disturbances that manifest in Cluster B symptoms had an inverse relationship with intimacy (Crawford, Cohen, Johnson, Sneed, & Brook, 2004) and this effect gained strength as young people entered adulthood. In another study assessing the impact of PD on changing levels of conflict between romantic partners from age 17 to 27, Chen et al. (2004) found that adolescent Cluster B disorders were associated with sustained elevations in partner conflict throughout this 10-year span. In contrast, adolescent Cluster A and Cluster C disorders were associated with elevated partner conflict only up until about age 23.

Adolescent Cluster B disorders significantly predicted subsequent disruptive behavior disorders (Johnson, Cohen, Skodol et al., 1999). Once co-occurring psychiatric disorders in adolescence were taken into account, youths with stable Cluster B disorders were 3.9 times more likely to have early adult disruptive behavior disorders. Similarly, adolescent Cluster B PDs independently predicted later aggressive and criminal behavior net of adolescent conduct disorder, oppositional defiant disorder, and other co-occurring PDs (Johnson, Cohen, Smailes et al., 2000). In particular, adolescent narcissistic PD symptoms predicted adult violence and criminal behavior.

The strong association between substance abuse, conduct disorder, and antisocial PD is well established. In our data, substance abuse disorder in early adulthood reflected a ten-fold increase in the odds of co-occurring antisocial PD and was independent of adolescent predictors in longitudinal models (Kasen et al., 2002). Much less is known about long-term risks for substance abuse associated with other PDs in adolescence. Borderline, histrionic, and narcissistic PDs in early adolescence were all associated with increased risk for substance abuse disorders in later adolescence and early adulthood, even after controlling for other well-known risk factors.
(male gender, conduct disorder, and parental substance abuse) (Cohen, Chen, Crawford, Gordon, & Brook, 2004; Johnson, Cohen, Skodol et al., 1999). When evaluated in separate models, PDs from Clusters A and C initially showed elevated risk for substance abuse but these effects dropped out in more complete models that included Cluster B effects. These data suggest that the impulsivity and perhaps self-destructiveness associated with early Cluster B disorders may represent a specific risk for subsequent substance abuse.

Cluster C PDs

Our research has focused less on Cluster C disorders (avoidant, dependent, and obsessive-compulsive PD) than other PDs, and very few studies elsewhere have investigated the developmental course of Cluster C disorders. In our sample avoidant and dependent PD are clearly associated with psychosocial impairment and low overall functioning in adolescence and adulthood (Bernstein et al., 1993; Crawford et al., 2005). In contrast, we have often failed to find similar results for obsessive-compulsive PD. It could be that functional impairment is sometimes obscured in obsessive-compulsive PD, for instance, when excessive devotion to school or work is ego-syntonic and even adaptive in achieving academic or occupational goals. Despite these limitations, we have accumulated the following information on the developmental course of Cluster C disorders.

**Stability.** The stability of Cluster C symptoms in the CIC sample was .48 from mean age 13 to 16 and then .42 from mean age 16 to 22 (Johnson, Cohen, Kasen et al., 2000). In more recent data, the stability of self-reported symptom criteria was .54 from mean age 22 to 33. Avoidant and dependent PD symptoms declined from adolescence to middle adulthood and showed comparable stability over time (Johnson, Cohen, Kasen et al., 2000). In contrast, obsessive-compulsive PD symptoms did not decline and stability levels were lower than other Cluster C disorders. Avoidant or dependent PD have been reported to be more stable in adult patients and may even increase over time in those with an Axis I disorder. Once again, developmental trajectories in clinic patients may differ from those in community samples.

**Comorbidity with Axis I Disorders.** Among adolescents with stable Cluster C diagnoses, Kasen et al. (1999) found that just over half (51%) had comorbid anxiety disorders. Such frequent co-occurrence probably reflects limits to the official nosology for anxiety disorders as much as real comorbidity between independent diagnostic entities (Caron & Rutter, 1991). In this context, generalized social phobia and avoidant PD probably reflect a single spectrum disorder rather than fully independent Axis I and Axis II disorders. Cluster C disorders also co-occurred with other Axis I disorders during adolescence: 34% of adolescents with stable Cluster C diagnoses had disruptive behavior disorders and 23% had depressive disorders.
Prospective Risk Factors. Kasen et al. (1999) found that the odds of new onset adult Cluster C disorder were 4 times higher following adolescent anxiety disorder and about 16 times higher when adolescent Cluster C disorders co-occurred with disruptive disorder or with depressive disorder. Once again, the increase in adult PD associated with comorbid disruptive and depressive disorders characterized all three Axis II clusters. With regard to individual PDs, major depressive disorder in adolescence was significantly associated with adult dependent PD net of effects of earlier dependent PD (Kasen et al., 2001).

Officially recorded physical abuse predicted early adult avoidant and dependent PD symptoms, the latter independently of co-occurring PD (Johnson, Cohen, Brown et al., 1999). Official neglect records predicted avoidant PD symptoms in early adulthood net of symptoms of other PDs. Childhood verbal abuse also predicted elevated rates of obsessive-compulsive PD in early adulthood (Johnson, Cohen, Smailes et al., 2001), thus representing one of the few prospective risks we observed for this disorder.

Prospective Outcomes. Stable adolescent Cluster C disorders did not predict adult anxiety disorders net of earlier Axis I adolescent disorders, but were independently associated with a nearly six-fold increase in odds for disruptive behavior disorders (Johnson, Cohen, Skodol et al., 1999). Unlike Cluster A and B disorders, however, adolescent Cluster C disorders were not associated with adult violent or criminal behavior (Johnson, Cohen, Smailes et al., 2000), suggesting that the disruptive behavior reflects more oppositional and defiant behavior than actual deviance. As such, aggression is not infused in Cluster C personalities to the same degree as observed in Cluster A and B personalities.

To our surprise, only adolescent Cluster C disorders and more specifically dependent symptoms in adolescence predicted subsequent suicidal ideation or suicide attempts in early adulthood (Johnson, Cohen, Skodol et al., 1999, p. 809), both with and without statistical controls for prior suicidal ideation and behavior and co-occurring Axis I and Axis II disorders. Insofar as aggression in Cluster C disturbances does not manifest toward others, it may be more likely to be targeted against the self.

Although most adolescents with Cluster C disorders did not have early romantic relationships, those who were involved with a partner had elevated rates of conflict up until about age 23 (Chen et al., 2004). Partner conflict declined in this group after age 23 and even dropped below normative levels. These anxious young adults may maintain romantic relationships by avoiding conflict that might jeopardize their relationship.

QUESTIONS TO BE ADDRESSED IN FUTURE WORK ON THE CIC
Much of our work has addressed prior gaps in knowledge about the prevalence of PDs in adolescence and uncertainty about how they persist and change over time. As our research goes forward we hope to extend informa-
tion on the course of PD and comorbid Axis I disorders into midlife. The fourth decade of life is distinguished from previous developmental stages by fewer age-graded markers. Individual differences and life experiences may cumulate, including effects of unique opportunities or influences, positive and negative life events, and the extent and timing of prior roles (e.g., years of schooling, career selection and career shifts, marriage and divorce, number and spacing of children, residential relocations, etc.). There are thus a wider array of social role markers in midlife (Lachman & James, 1997; Brooks-Gunn & Kirsch, 1984). Although earlier PD affects the attainment and timing of these status variables (Cohen, Chen et al., 2005), we also expect that life experiences will have reciprocal effects influencing the course and continuity of PD over the fourth decade of life.

We are interested in resilience and recovery factors that may be related to enduring basic strengths of the individual and stable aspects of the environment. By midlife we expect that some individuals find a lifestyle and setting niche that is reasonably well-suited to the management of their vulnerabilities. Among these people we anticipate a decline of PD during midlife (Oldham & Morris, 1990). Others may take on new tasks, especially childrearing, that pose significant new risks insofar as they reduce personal control over one’s time and divert attention away from one’s own needs (Cowan & Cowan, 1992). Among these individuals PD symptomatic expression may increase.

We remain interested in the relationship between parental PD and symptoms and disorders in offspring, and we are just beginning to examine the impact of PD on fathering. There is an increasing sophistication of thinking about reciprocal and interactive impacts of heredity and environment on the development of disorders. Such improvements in our theoretical models, however, make it more and more obvious that much developmental information will be needed in the ongoing effort to sort out general and specific etiological relationships for different disorders.

Etiological Factors vs. Exacerbating Factors

We expect that our future work will pull together more information about the substantive meaning of risk factors and course issues that have emerged in prospective research on PD. Can we discriminate basic risks that create an ongoing vulnerability from “kindling” risks that lead to variation in symptom expression over time? This question has critical clinical, policy, and potentially diagnostic implications. Empirical studies have indicated that certain risks predict subsequent increases in symptoms. Negative life events, for instance, increase subsequent symptoms of depression, anxiety, and other psychiatric disorders, including elevations meeting diagnostic criteria. Should life events be thought of as an etiological factor for these disorders? Probably no more than exercise-induced cardiac strain should be thought of as a cause of cardiac disease. Rather, physical exertion may exacerbate symptoms in a person with cardiac disease whose
underlying illness more accurately reflects biological sensitivity to cholesterol-inducing fatty acids in combination with unhealthy behaviors such as diet and lack of regular exercise. Similarly, the etiology of PD and other psychiatric disorders is more likely to involve ongoing vulnerabilities that include genetic influences or more specific gene-environment interactions. Etiological risk factors may involve early life trauma, severe deprivation, or extremely maladaptive environmental factors that shape the individual’s world view and behavioral patterns in ways that are resistant to change. Exacerbating factors may involve acute events (e.g., loss of a loved one) or reflect more chronic disturbances (e.g., dysfunctional and abusive relationships in adulthood). Discrimination between exacerbation and etiological factors may have important treatment and prevention implications in the future.

Comorbid Axis I and Axis II Disorders

The meaning of the high rate of psychiatric comorbidity within and across Axes represents another ongoing challenge. Because each diagnostic group predicts the other across adolescence and early adulthood, we cannot conclude that Axis I disorders “cause” the Axis II disorders or vice versa. At minimum, we can observe that anxiety, depressive, disruptive behavior, and Axis II disorders appear to mutually reinforce each other when they co-occur. Shared genetic influences, for instance, may account for the observed overlap between impulsivity and aggression that characterize Cluster B and disruptive behavior disorders.

IMPLICATIONS OF LONGITUDINAL FINDINGS ON PERSONALITY DISORDER

Findings from the CIC Study make it clear that PD symptom constellations identified in adulthood have their origins in childhood and can be reliably assessed in combined youth and parent reports. Symptoms become much less prevalent with age, at least between age 10 and 25, thus making it clear that maturational factors influence PD symptoms during adolescence and probably preadolescence as well. Nevertheless the stability of these problems relative to age peers is roughly equivalent to that found in adulthood. In addition, PD problems that were previously at the extreme ends of more nearly normal distributions become increasingly deviant relative to peers as they persist into early adulthood.

Second, elevated symptoms even in early adolescence have negative prognostic implications over the next 10 to 20 years, often more serious or pervasive than those associated with Axis I disorders. In many cases, Axis II disorders may account for the long-term impairment associated with the Axis I disorders that often co-occur with PDs. Again, these findings are consistent with those based on adult samples, including clinical cases.

Third, there are a number of environmental insults that predict PDs
both in adolescence and adulthood. Although our findings clearly show that early trauma or abuse increases the risk of PD, it is important to emphasize that they do not account for all, or even most cases of PD observed in our longitudinal cohort. More likely, environmental effects may be conditional on genetic factors (and vice versa) and the interplay between the two must be clarified before we can advance our understanding of the origins of PD and of comorbidity both among PDs and with Axis I disorders. In addition, we need to discriminate environmental settings or experiences that result in short-term exacerbation or reductions in symptoms from those that lead to long-term increase or decrease in vulnerability.

**IMPLICATIONS OF THESE FINDINGS FOR POTENTIAL CHANGES IN HOW THE DSM DEFINES OR CLASSIFIES PERSONALITY DISORDER**

First, these cumulative findings make it clear that PDs are significant mental health problems. Thus changes in the DSM should be considered only if they are not likely to have a negative impact on future research or treatment. In particular, the absence of insurance reimbursement for treatment of PD in much of the U.S. is deplorable. If DSM changes such as the integration of Axis I and II would improve this situation, we would tend to support it, especially in light of articulated treatment alternatives for these disorders (e.g., Sperry, 2003).

Second, despite the higher-than-adult symptom levels of adolescents, the long-term negative prognostic implications of high-for-age PD symptoms and PD diagnoses shown in this study indicate the importance of early attention to these problems. Thus the DSM-V should emphasize more clearly that adolescent PD represents significant short- and long-term risk for ongoing psychopathology and functional impairment. However, to avoid premature labeling, it may be worthwhile to distinguish adolescent PD from adult PD, just as conduct disorder is distinguished from antisocial PD. Such diagnoses should not carry any unwarranted and possibly stigmatizing expectation of necessary persistence into adulthood.

Third, our findings and those of other researchers show that comorbidity among disorders is highly prevalent in the general population as well as in clinical populations, is present both within and between diagnostic axes, and is an indicator of elevated impairment and negative prognosis. A strong consensus statement by the mental disorder professions that comorbidity is very high and that there is clear evidence that such comorbidity bodes ill for long-term recovery and adjustment would be a good starting point for diagnostic considerations. It is possible that retention of the Axis I—Axis II distinction may encourage attention to comorbidity.

On the other hand, despite some historical advantages of distinction between Axis I and II, it may be time for reconsideration of this separation for at least some disorders. For some disorders it may be more useful to group in a single axis disorders for which criterion and empirical overlap
often suggest a common spectrum with potential etiological commonalities. In particular, it is difficult to justify placement of disruptive disorders of childhood on Axis I and impulsive and antisocial Cluster B PDs on Axis II. Either conduct and oppositional/defiant disorders should be viewed as childhood PDs (our preference) or some combination of these disorders should appear as an Axis I spectrum. Just as some distinction between major depression in adolescence and major depression in adulthood is often proposed, in this case and potentially for some other disorders, a different set of criteria may be needed for childhood or adolescent manifestations as compared to adult criteria. Subtypes of conduct disorder (CD) may be identified that may be usefully discriminated in terms of treatment and prognostic implications (e.g., Moffitt, 1993; Quay, 1993). On the other hand, there is little reason to require childhood CD as a prerequisite to adult antisocial PD diagnosis any more than any other childhood disorder is an adult PD criterion. Adults meeting other criteria are likely to be prognostically equivalent with or without such an identified history. Some adolescents may be protected from such a childhood diagnosis by family, by their ability to hide relevant behaviors and attitudes, or by the sexual bias of the CD criteria.

We believe that it is premature to incorporate current perspectives on the potential etiology or meaning of PD into the diagnostic nomenclature. It is clear that basic personality dimensions, and even second-order personality factors will be part of our eventual understanding of many Axis I and II disorders. Nevertheless, this approach underestimates the importance of the extremity/deviance aspect of disorders in our view. Similarly, other potential etiologically or treatment-relevant factors, including attachment and identity issues (Cohen & Crawford, 2005), are extremely important. Nevertheless, their etiological roles are not so well understood as to justify current formal adoption into the diagnostic system except as criteria for individual disorders.

Although we are reluctant to rely on our data for advice about individual PDs, we may note that schizoid disorder in these data appears to be distinctly different from the other two Cluster A disorders, and may be more related to asocial disorders such as Asperger’s syndrome or avoidant anxiety. On the whole, our prospective data suggest that diagnostic criteria may work essentially similarly for most PDs in adolescents and adults. We have found that these criteria counts serve reasonably well as symptom scales. Despite the desirability of a more empirical basis for diagnostic cutoffs, current requirements apparently indicate reasonable clinical levels of problem in our epidemiological sample.

This rather conservative view, on the whole, reflects our experience in using the current system. We find the broad coverage and flexibility of the International Classification of Diseases (ICD) very attractive. Nevertheless, the utility of formal diagnostic criteria in maximizing the match between diagnostic labels and actual problems is absolutely essential to the accumulation of empirical epidemiological, biological, etiological, and treatment evidence.
REFERENCES


