More Than Three-Quarters of Young Adults with Psychiatric Disorders First Had a Diagnosis Between the Ages of 11 and 18 Years,
Indicating That We Must Consider Childhood and Adolescent Mental Illness as a Key Risk Factor for Later Psychiatric Problems. However, There Remain Important Questions About Diagnostic Prediction From Childhood and Adolescence to Adulthood. Herein, We Present New Evidence About Which Childhood and Adolescent Disorders Reliably Precede Later Disorders.

Homotypic Prediction Refers to a Disorder Predicting Itself Over Time (e.g., Earlier Depression Predicting Later Depression). This Supports the Idea That a Single Disease Process Expresses Itself Robustly Across Developmental Contexts. Homotypic Prediction Has Been Identified in Most Studies Predicting From Childhood to Late Adolescence and From Childhood and Adolescence to Young Adulthood. Indeed, Prior Disorder Status Is Typically the Strongest Predictor of Having That Disorder Later.

Heterotypic Prediction Refers to Different Disorders Predicting One Another Over Time (e.g., Earlier Oppositional Defiant Disorder [ODD] Predicting Later Depression). Such Patterns May Suggest That the Different Disorders Reflect a General Disease Process That Has Specific Phenotypic Expressions in Different Developmental Contexts. Although Typically Less Common Than Homotypic Prediction, 2 Pat-
terns of heterotypic prediction have received consistent support. First, anxiety and depression tend to be cross-predicted from childhood/adolescence to adulthood (anxiety predicting depression: full support\(^1,3,10,11,20\), partial support\(^9,13,16,19\); depression predicting anxiety: full support\(^1,3,10,11,20\), partial support\(^13,16\)). Second, childhood/adolescent conduct/oppositional problems tend to precede adult anxiety and depression\(^1,10,16,21-23\) but not vice versa\(^1,11,13\) (Hofstra et al\(^17\) present an exception).

Taken together, homotypic prediction appears to be common across a range of disorders, whereas heterotypic prediction is limited to a few specific pathways, but a number of substantive and methodological issues complicate the interpretation of this work. First, many studies collapse childhood and adolescent disorders to predict adult disorders, despite the fact that important changes in the prevalence of some disorders, such as depression and conduct disorder (CD), occur between childhood and adolescence.\(^25-28\) Such changes could indicate that disorders at different ages result from different etiologic pathways\(^27-30\) and so combining across childhood and adolescence could obscure important differences between childhood and adolescent prediction. Also, little attention has been paid to the fact that these developmental pathways may be different for males and females. For example, the prevalence of depression changes at puberty for girls but not for boys.\(^29\)

Second, most studies collapse multiple, potentially heterogeneous disorders into more general diagnostic groupings. For example, all anxiety disorders tend to be combined into one category, which could mask differences in prediction between individual anxiety disorders. Furthermore, ODD and CD are often combined, despite the fact that they are distinct in factor analytic studies\(^32,33\) in risk factors studies,\(^34\) and when tested as predictors of later problems.\(^35\) Indeed, ODD may be as likely to be linked with emotional disorders as CD.\(^10\)

Finally, studies of diagnostic predictors of later disorders have typically focused on pairwise associations: one earlier disorder predicting one later disorder. Yet, disorders tend to co-occur, and when comorbidity is not taken into account, pairwise associations may simply represent indirect effects rather than direct associations.\(^36,37\) For example, bivariate analyses may suggest that childhood anxiety disorders predict adolescent depression, but this association could be accounted for by comorbidity between childhood anxiety and depression.

Herein, we use Great Smoky Mountains Study (GSMS) data from middle childhood through young adulthood and stringent criteria to examine a broader range of patterns of homotypic and heterotypic prediction from middle childhood and adolescence to young adulthood.

**METHODS**

**SAMPLE AND PROCEDURES**

The GSMS is a longitudinal study of the development of psychiatric disorder and need for mental health services in rural and urban youth.\(^24,48\) A representative sample of 3 cohorts of children, aged 9, 11, and 13 years at intake, was recruited from 11 counties in western North Carolina. Potential participants were selected from the population of some 20,000 children using a household equal probability, accelerated cohort design.\(^50\) The accelerated cohort design means that over several years of data collection each cohort reaches a given age in a different year, thus controlling for cohort effects.\(^51\) Youth with behavior problems were oversampled. A screening questionnaire was administered to a parent (usually the mother) of the first-stage sample (n=3896). The questionnaire consisted mainly of the Externalizing (Behavioral) Problems Scale of the Child Behavior Checklist\(^22\) and was administered by telephone or in person. All children scoring higher than a predetermined cut point (the top 25% of the total scores), plus a 1-in-10 random sample of the rest (ie, the remaining 75% of the total scores), were recruited for detailed interviews. Ninety-five percent of families contacted completed the telephone screen.

About 8% of the area residents and the sample were African American, and fewer than 1% were Hispanic. American Indian individuals made up only about 3% of the population of the study area, which is overwhelmingly white, but were oversampled from school records to constitute 25% of the study sample. This was done by using the same screening procedure but recruiting everyone irrespective of screen score. Of the 456 American Indian children identified, 96% were screened, and 81% (n=350) participated in the study. All subjects were given a weight inversely proportional to their probability of selection so that the results presented are representative of the population from which the sample was drawn. Of all subjects recruited, 80% (n=1420) agreed to participate.

**Table 1** presents the study design and participation rates at each wave. Data were collected on 1 cohort at ages 9 and 10 years, 2 cohorts at ages 11, 12, and 13 years, and all 3 cohorts at ages 14, 15, 16, 19, and 21 years. This article presents data on 8806 parent/child pairs of interviews carried out across the age range 9 through 21 years. Participants were interviewed as closely as possible to their birthday each year. Funding constraints prevented our interviewing the youngest cohort from January 1997 through June 1998.

Interviews were completed with the children and their primary caregiver at their home or a convenient location until age 16 years and with the young adults only thereafter. Before the interviews began, interviewees signed informed consent forms approved by the Duke institutional review board. Across waves, an average of 82% of all possible interviews were completed, ranging from 75% to 94% at individual waves.

**MEASURES**

Psychiatric disorders were assessed using (1) the Child and Adolescent Psychiatric Assessment (CAPA)\(^38-40\) until age 16 years and (2) the upward extension of the CAPA, the Young Adult Psychiatric Assessment (YAPA)\(^40\) at ages 19 and 21 years. Scoring programs for the CAPA and YAPA, written in SAS,\(^41\) combined information about the date of onset, duration, and intensity of each symptom to create diagnoses according to the DSM-IV.\(^42\) With the exception of attention-deficit/hyperactivity disorder (ADHD), for which only parental reports were counted, a symptom was counted as present if it was reported by either the parent or the child until age 16 years or by the young adult at ages 19 and 21 years, as is standard clinical practice. Two-week test-retest reliability of CAPA diagnoses in children aged 10 to 18 years is comparable with that of other highly structured interviews (as for individual disorders range from 0.6–1.0).\(^39\) To minimize recall bias, the time frame of both interviews for determining the presence of most psychiatric symptoms was the preceding 3 months. A previous publication suggested that there was little evidence of symptom attenuation (lower reported symptom levels in subsequent data waves), cohort differences, or differential dropout in this sample.\(^15\)
In the current study, disorder status was aggregated across childhood (ie, ages 9-12 years), adolescence (ie, ages 13-16 years), and young adulthood (ie, ages 19-21 years). Childhood and adolescent diagnostic groupings included depression (including major depressive disorder [MDD], dysthymia, and depressive disorder, not otherwise specified), separation anxiety disorder in childhood, generalized anxiety disorder (GAD), CD, ADHD, and ODD. DSM-III-R overanxious disorder (OAD) was also included because we had previously found that it predicted several adolescent disorders when diagnosed in childhood. Disorders with a prevalence of less than 1% in a given developmental period were not included in analyses (eg, separation anxiety in adolescence, social phobia, posttraumatic stress disorder). Substance disorders (including those meeting abuse or dependence criteria) were only sufficiently common for inclusion beginning from adolescence. Young adult diagnostic groups included depression (same as childhood and adolescence groups), GAD, panic disorder without agoraphobia, agoraphobia without panic, and antisocial personality disorder (ASPD).

**ANALYSES**

Weighted logistic regression models were estimated using generalized estimating equations implemented by SAS PROC GENMOD. Sampling weights were inversely proportional to selection probability. Robust variance (sandwich-type) estimates were used to adjust the standard errors of the parameter estimates for the stratified design effects. Therefore, the resulting parameters are representative of the population from which the sample was drawn.

Ideally, all diagnoses from all developmental periods would be combined into a single path-type multivariate analysis. Because such complex models did not converge despite our reasonable sample size, we examined psychiatric status across 2 developmental periods at a time: from adolescence to young adulthood and from childhood to young adulthood. Homotypic and heterotypic patterns were determined by predicting each later disorder (eg, depression in young adulthood) from each earlier disorder in a series of 3 models. In the bivariate, unadjusted model, a prior disorder was the single predictor of the later diagnosis. The unadjusted odds ratios (ORs) resulting from model 1 are reported in **Table 2** and **Table 3**. In the sex differences model, model 2, the prior disorder, sex, and the sex × prior disorder interaction were included (full results are available on request). In the final comorbidity or adjusted model, the prior disorder that corresponded to the outcome variable and all other prior disorders were included. For example, childhood depression and all other childhood disorders were entered to predict young adult depression in the childhood/young adulthood model. The adjusted ORs resulting from the final model are reported in **Table 2** and **Table 3**. Inclusion of comorbid disorders provides a stringent test of homotypic and heterotypic prediction patterns. Where evidence of a sex × disorder interaction was detected, separate results for adjusted models were provided for males and females. Analyses involving childhood were based on 2 GSMS cohorts (n=1008; <13 years at intake) and those involving adolescence were based on all 3 GSMS cohorts (N=1420).

As with any longitudinal study, not all assessments were completed at each data wave. Such missing assessments may affect prediction estimates if individuals with missing observations were more or less likely to have a psychiatric disorder than individuals with complete observations. To test for such effects, each individual's total number of missed assessments adjusted for the total number of expected assessments was predicted by the individual's psychiatric status at his or her first assessment (because all subjects had at least 1 assessment). Initial rates of psychiatric disorder did not predict the likelihood of missing assessments (z 1.5; P=.23), suggesting no effect of differential dropout. Therefore, observations missing within a given developmental period were excluded from analyses involving that development period. Because subjects were interviewed multiple times within each developmental period, subjects could miss 1 interview and still be included in analyses for that period.

**RESULTS**

**HOMOTYPIC PREDICTORS**

Table 2 and Table 3 display the results from unadjusted and adjusted models for each pair of developmental groups (adolescence to young adulthood, childhood to young adulthood). Separate adjusted results are provided for males and females where a significant sex × disorder interac-
In unadjusted models, homotypic prediction was found for antisocial personality disorder (from adolescent CD), depression, and substance disorders (Table 2). Both generalized anxiety and panic disorders were predicted from OAD. The apparent association of adolescent depression with young adult depression was completely attenuated in adjusted models, whereas the homotypic prediction of ASPD, GAD, and substance and panic disorders was undiminished. Overanxious disorder predicted later GAD and panic disorders more strongly for males than females.

Because the attenuation of homotypic prediction of depression in the comorbidity-adjusted model was unexpected, possible informant effects were tested by running the adjusted models separately by parent and self-reports. The results were the same regardless of informant (parent report, OR, 0.6; 95% confidence interval [CI], 0.2-2.7; self-report, OR, 1.0, 95% CI, 0.4-3.1). To clarify whether homotypic prediction for CD predicting ASPD was an artifact of the diagnostic criterion for ASPD re-
quiring prior evidence of CD before age 15 years, the adjusted model was rerun using an ASPD diagnosis in which subjects were not required to have displayed prior evidence of CD before age 15 years. Again, CD alone predicted ASPD (OR, 5.2; 95% CI, 1.4-19.1).

Childhood to Young Adulthood

As in the adolescence/young adulthood models, the link between CD and ASPD was also found after adjusting for comorbidity (Table 3). This link remained if the alternative form of the ASPD diagnosis (requiring no prior CD symptoms) was used (OR, 3.2; 95% CI, 1.0-9.6).

There was evidence of prediction between various anxiety disorders in unadjusted models, although only 3 associations were significant after adjustment: OAD predicted panic disorder, separation anxiety disorder predicted agoraphobia without panic disorder, and GAD predicted agoraphobia without panic disorder.

### Table 3. Young Adult Diagnoses Predicted From Childhood Diagnoses

<table>
<thead>
<tr>
<th>Childhood dx</th>
<th>Young Adult Disorders, OR (95% CI), %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dx absent, %</td>
<td>GAD 2.6 (0.7-4.4)</td>
</tr>
<tr>
<td>Dx present, %</td>
<td>2.6 3.0 1.2 0.6 0.6 0.8 19.8</td>
</tr>
<tr>
<td>Unadjusted</td>
<td>7.4 (1.2-46.5)</td>
</tr>
<tr>
<td>Adjustedb</td>
<td>5.3 (1.3-22.1)</td>
</tr>
</tbody>
</table>

### HETEROTYPIC PREDICTORS

Adolescence to Young Adulthood

In adjusted models, heterotypic patterns were found for depression and all anxiety disorders. Specifically, GAD and OAD predicted depression (in males for the depression-OAD link). Adolescent depression also predicted agoraphobia without panic disorder. Adolescent ODD predicted later GAD, panic disorder without agoraphobia (in males only), and depression. Finally, adolescent substance disorders predicted later depression.
Childhood to Young Adulthood

Compared with adolescent/young adulthood models, limited support for heterotypic prediction emerged in the childhood to young adulthood adjusted models: childhood ODD predicted young adult depression and childhood depression predicted panic disorder without agoraphobia and GAD.

COMMENT

A review of previous studies suggested the following conclusions with respect to the continuity of disorders from childhood and adolescence to young adulthood: (1) Homotypic prediction is the norm from childhood/adolescence to young adulthood. (2) Generalized anxiety and depression cross-predict. (3) Childhood/adolescent combined disruptive disorders (ODD and/or CD treated as a single entity) predict adult anxiety and depressive disorders in addition to ASPD. This study provided developmentally differentiated and stringent tests of these patterns by (1) separating childhood from adolescent diagnostic predictors, (2) disaggregating specific anxiety and disruptive disorders, and (3) adjusting for comorbid conditions. Our results indicate that prediction patterns are actually more developmentally and diagnostically nuanced than the previous literature suggests.

In summary, although homotypic patterns were common, the path from adolescent to young adult depression was entirely accounted for by other comorbidities. Of childhood and adolescent anxiety disorders, DSM-III-R OAD was most likely to predict later young adult anxiety disorders. Generalized anxiety and depression cross-predicted, although these effects were not uniform across childhood and adolescence. Finally, a single behavioral disorder, adolescent ODD, preceded anxiety and depressive disorders.

CAVEATS

Before considering these findings in more detail, the following methodological considerations should be kept in mind. First, the GSMS participants lived in a rural area, and the study oversampled American Indian children, with very few African American (8%) and no Latino or Asian American children. Thus, the sample is not representative of the US population. However, comparison of the GSMS with other studies indicates that there are similar rates of cumulative childhood disorders in representative samples from other counties, other regions of the United States, and samples involving higher levels of Hispanic and African American youth. While the subjects were followed up to 12 years (age 9-21 years), cases will have been missed because subjects may have met criteria for disorders prior to our study, between assessments, or after their last assessment.

Second, some associations of moderate to large magnitude were nonsignificant because of the limited number of youth with the particular disorder. However, studies of homotypic and heterotypic predictors from child to adult psychiatric disorder that deal with a range of disorders are rare because they depend on large, longitudinal community-based samples carefully characterized over many years. We are unaware of any currently existing studies with greater power to address these questions. Finally, our research aim was to determine which childhood and adolescent disorders reliably precede young adult disorders. This did not allow us to examine the chronological order of disorders within a developmental period. For the current analysis, the order of the disorders within a developmental period, however, had no effect on the strength of the association with the young adult outcome.

A terminological note is also in order. We have referred herein to homotypic and heterotypic prediction rather than continuity, despite the latter term’s common usage. We believe that typical measurement schedules in prospective studies cannot adequately capture (dis) continuity, because observations of disorders tend to be intermittent (rather than continuous).

HOMOTYPIC PREDICTION

Although homotypic patterns were identified (eg, CD to ASPD and substance-related disorders), homotypic patterns were less common than previously reported by other studies when accounting for comorbidity between disorders. There was no evidence of homotypic prediction for depression and homotypic prediction of young adult anxiety disorders was primarily accounted for by DSM-III-R OAD, rather than by DSM-IV GAD.

In preparation for DSM-IV, Klein and colleagues reviewed taxonomic issues related to the DSM-III-R anxiety disorders. Overanxious disorder was the focus of particular attention because it included a group of heterogeneous worries (eg, about the future, academic performance, self-consciousness) and was highly comorbid with other anxiety disorders (particularly GAD). Despite these concerns, it was recommended that it be retained as a childhood anxiety disorder, but with modified criteria to reduce overlap with other disorders. Instead, it was eliminated with the rationale that these children would likely receive a diagnosis of DSM-IV GAD. In a prior study, our group compared the relative predictive validity of childhood OAD as compared with DSM-IV GAD in predicting adolescent disorders. Overanxious disorder not only predicted later anxiety disorders but also predicted adolescent depression and CD. In contrast, DSM-IV GAD only predicted later CD. In this study predicting young adult disorder status, OAD again predicted both anxiety disorders and depression. This is in line with findings from the New York Child Longitudinal Study in which OAD predicted young adult depression, social phobia, and generalized anxiety. Together, these findings suggest that the DSM-IV GAD criteria are insufficient for assessing the full range of “generalized anxiety” in children and adolescents and fail to identify anxious children at risk for a range of later disorders. It seems that Klein and colleagues were right to suggest that OAD should have been retained in the DSM-IV. We recommend its rehabilitation in the DSM-V.

The example of depression illustrates the importance of separating childhood from adolescent predictors and controlling for comorbidities. The significant bivariate prediction from adolescent to young adult
depression (OR, 3.3) was entirely accounted for by co-

morbidty of adolescent depression with adolescent ODD, GAD, OAD, and substance disorders (OR for depression reduced to 0.8), whereas there was no direct pre-
diction from childhood depression to young adult depression even in the bivariate models. This suggests that the apparent association between adolescent depression and young adult depression is epiphenomenal, result-

ing from the direct associations between comorbid ado-

lescent disorders and later depression.

This may appear to be a clear departure from the con-
sensus of previous research, but actually it is not. Many

studies looking at the adolescence–young adult depres-
sion link have used highly selected or clinical samples

and/or failed to account for common comorbid disor-
ders. While these studies can demonstrate that ado-

lescent depression precedes young adult depression, they are insufficient, on their own, to provide evidence of di-
rect prediction. Such evidence can only come from com-

munity samples that assess for a range of disorders, in

addition to depression, at multiple points in both ado-

lescence and young adulthood.

To date, 3 such studies have been published. The first,
a community sample of adolescents followed up into young

adulthood by Lewinsohn and colleagues, concludes that their results "clearly illustrate a strong pattern of conti-
nuity for depression." Their initial analyses demon-
strate higher risk of later depression for a group of ado-

lescents with MDD as compared with groups with either

no depression or a nonaffective disorder. In this compar-

ison, however, adolescent MDD cases were allowed to have

other Axis I nonaffective disorders (and 51.0% did). A more

stringent test compared risk for depression between a group

with "pure" adolescent depression and a group with co-
morbid depression and found no difference. This might

seem to suggest that even after controlling for comorbid-

ty there is a link from adolescent to young adult depres-
sion, but rates of young adult depression are not pro-

vided for either of these 2 groups. It is entirely plausible

then that the "pure" MDD group could both only be mar-
ginally associated (or not associated) with later MDD and,
at the same time, not be statistically different from the co-
morbid MDD group. Without knowledge of the rates of

young adult depression in these 2 groups, one cannot draw

any conclusions about the role of comorbidity in the ado-

lescent–young adult MDD link.

The second study tested this link in a birth cohort of

1265 children and concluded that there was a “direct and specific” link from adolescent depression to later depres-
sion. The study design provides a rather stringent test for

the outcomes of adolescent depression by account-

ing for the effects of anxiety disorders, early cigarette

smoking, CDs, alcohol abuse, and a range of other pu-
tative risk factors. At the same time, the negative out-

comes (including depression) are assessed for ages 16 to

21 years and thus overlap both with late adolescence and

young adulthood. If there were a rather punctuated shift

in depression between adolescence and young adult-

hood, it would not be detected by this design. But is this

likely? In fact, such a striking shift occurs in depression

a few years earlier in the pubertal transition from child-

hood to adolescence, so this possibility cannot be re-

jected a priori. While the pubertal shift is associated with

significant biochemical changes, the shift to young adult-

hood and the associated transition to independent liv-

ing may be similarly substantial in the social domain.

The final study by Pine and colleagues found that the

best-fitting multivariable prediction model of young adult

depression did not include adolescent depression, after

accounting for comorbidities. As with our findings, there

was evidence of significant prediction from adolescent
to adult depression in bivariate analyses. This signifi-
cant effect was primarily attenuated by inclusion of CD

in the best-fitting adjusted model; ODD was not in-
cluded in their analysis.

We suggest, therefore, that the early conclusions about

the link between adolescent and young adult depression

may have been premature. This putative link may be

attenuated by comorbid adolescent disorders, particu-

larly anxiety and behavioral disorders. It may also be

the case that there is a rather punctuated shift in the natu-

ral course of depression around age 17 or 18 years. This

hypothesized shift is consistent with an emerging litera-
ture that suggests heterogeneity in childhood/adolescent and adult depressions with respect to biologi-
cal correlates and psychosocial predictors.30,55

HETEROTYPIC PREDICTION

Each heterotypic pattern identified from previous re-

search was extended by the current study. An emerging

body of literature has suggested that generalized anx-

iety not only reliably precedes depression, but vice

versa. By disaggregating childhood and adolescent di-
agnoses, the current study found that this pattern was

developmentally nuanced: only childhood depression pre-
dicted young adult GAD and only adolescent GAD pre-
dicted later depression. In addition, adolescent OAD was

a stronger predictor of later depression in males than GAD.

Furthermore, the cross-prediction was stronger than ho-

motypic prediction for these 2 disorders (a finding also

previously reported by Pine and colleagues).

Together with evidence that GAD and depression oc-
ccur more often with one another than with other disor-
ders, and have shared genetic etiology, this lends support to the notion of grouping these disorders more closely than is currently reflected in the DSM-IV. Cross-
prediction (or sequential comorbidity) is not, however,

very strong evidence of diagnostic unity. Childhood and

adolescent GAD and MDD predicted different adult dis-
orders and young adult GAD and MDD were predicted

differently by childhood and adolescent disorders. Our find-
ings, together with those of other longitudinal epidemi-
logic samples, suggest that GAD and MDD, while

closely related, are distinct both in terms of their natu-

ral courses and developmental histories.

A recent study by Kim-Cohen and colleagues found

that CD/ODD “was a part of the developmental history of
every adult disorder.” Because CD and ODD were

combined in that study, it was unclear, for any given out-
come disorder, whether it was preceded by ODD, CD, or
:both. We found that young adult depression and anx-

iety disorders were preceded by adolescent ODD, but not

CD. This finding is at odds with the traditional “failure

HETERO
model,\textsuperscript{22,60} which suggests that depression results from the social and educational failures that often follow CD. As with homotypic patterns of depression, the bivariate link between adolescent CD and young adult depression in our study was entirely accounted for by comorbid disorders (here, adolescent GAD, ODD, and substance disorders). If it is indeed ODD, rather than CD, that predicts later depression, then this might suggest an amended failure model that emphasizes the social and emotional consequences of irritability and interpersonal difficulties rather than the legal and social sequelae of delinquency and overt aggression.

As part of the research agenda for DSM-V, questions have been raised about the diagnostic and predictive validity of ODD after accounting for comorbid disorders (eg, ADHD, CD).\textsuperscript{61} Our findings suggest that ODD is a singular disorder in being part of the developmental history of many young adult affective and anxiety disorders. No other childhood or adolescent disorder demonstrated such pleiotropic effects. In DSM-IV, ODD is ruled out if criteria for CD are met. In International Statistical Classification of Diseases, 10th Revision, ODD is a mere subtype of CD. Our data suggest that this subordination of ODD may be misguided. One accepted measure of the utility of a psychiatric diagnosis is the extent to which it predicts future psychiatric functioning.\textsuperscript{62,63} On this measure, ODD may be in a class by itself.

Submitted for Publication: September 21, 2009; final revision received January 25, 2009; accepted February 2, 2009.

Correspondence: William E. Copeland, PhD, Center for Developmental Epidemiology, Department of Psychiatry and Behavioral Sciences, Duke University Medical Center, Box 3454, Durham, NC 27710 (william.copeland@duke.edu).

Author Contributions: All authors had full access to all the data in this study and Dr Copeland takes responsibility for the integrity of the data and the accuracy of the data analysis.

Financial Disclosure: None reported.

Funding/Support: This work was supported by National Institute of Mental Health grants MH63970, MH63671, and MH48085, National Institute on Drug Abuse grant DA/MH11301, and the William T. Grant Foundation.

REFERENCES


30. Kaufman J, Martin A, King R, Chaney D. Are child-, adolescent-, and adult-