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Structure of Psychopathology in Romanian Preschool-Aged Children in an Epidemiological and a High-Risk Sample

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Abstract

Objective: Research on bifactor models of psychopathology in early childhood is limited to community samples with little longitudinal follow-up. We examined general and specific forms of psychopathology within 2 independent samples of preschool-aged Romanian children. Within a sample with children exposed to psychosocial deprivation, we also examined antecedents and longitudinal outcomes of the general factor.

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Dr. Plamondon served as the statistical expert for this research.

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Method: One sample consisted of 350 Romanian children (mean age = 39.7 months, SD = 10.9) from an epidemiological study; the second sample consisted of 170 Romanian children (mean age = 55.6 months, SD = 1.9) exposed to severe early-life deprivation, as well as community comparison children, with longitudinal follow-up at 8 and 12 years. Psychopathology symptoms were assessed through caregiver-reported structured clinical interviews.

Results: An SI-1 bifactor model of psychopathology was supported in both samples and included specific factors for externalizing, internalizing, and disturbed relatedness symptoms. In the second sample, longer duration of psychosocial deprivation and lower-quality caregiving were associated with higher scores on the general and all specific factors. Higher scores on the general factor were associated with later cognitive function, competence, and psychopathology symptoms. Considering all factors together, only the general factor explained variance in later childhood outcomes and was slightly stronger compared to a total symptom count for some, but not all, outcomes.

Conclusion: General psychopathology in early childhood explains meaningful variance in child outcomes across multiple domains of functioning in later childhood. However, important questions remain regarding its clinical utility and usefulness, given complex measurement and limited explanatory power beyond the more accessible approach of a total symptom count.

Clinical trial registration information: The Bucharest Early Intervention Project; https://clinicaltrials.gov/; NCT00747396.

Keywords

general psychopathology; preschool; bifactor modeling; psychosocial deprivation

Research on the structure of psychopathology in adulthood using bifactor models has rapidly expanded over the past decade.[1–8] Bifactor modeling (ie, items loading onto 2 or more factors simultaneously) is 1 methodological approach to examining the structure of psychopathology. Generally speaking, among children and adolescents, the bifactor structure of psychopathology consistently demonstrates adequate fit to the data with 1 overarching general factor of psychopathology and various ancillary specific factors, often internalizing- and externalizing-specific symptoms.[9–13] However, the literature on early childhood in particular (age <6 years) is limited to investigations of a few community or nationally representative samples,[14–19] with no reports in high-risk clinical samples.

Aside from the robust literature on more traditionally measured psychopathology across childhood and implications for lifespan development, the bifactor structure of psychopathology represents a fundamentally different methodological approach to psychopathology research. There is still much to learn about the structure of psychopathology in early childhood. For example, domains of psychopathology related to adversity in early childhood, such as reactive attachment disorder (RAD) and disinhibited social engagement disorder (DSED), have not been explored in this framework. Although they are rare in the general population, signs of RAD and DSED may be important for understanding variation in psychopathology for young children in high-risk contexts, such as those who experience severe psychosocial neglect. Exploring the addition of rare yet relevant forms of psychopathology to the structure of psychopathology in early childhood is

consistent with similar studies in adulthood that include psychosis and thought disorders.[8] Furthermore, samples with higher base rates of psychopathology and measurable outcomes of psychopathology can offer insight into antecedents and outcomes associated with general psychopathology.

It is important to acknowledge that a quantitative nosology of psychopathology has a rich 9-decade history of empirical work on constellations of signs and symptoms of psychopathology in children and youth that preceded studies of general and specific forms of psychopathology in adults.[6] It is only more recently that progress has been made to develop a nosology of psychopathology focused on adults, particularly using a bifactor framework, lending a new lens for re-examining the structure of psychopathology in young children. Thus, examination of bifactor models of psychopathology in young children is presently limited, with no known research attempting to integrate signs of RAD and DSED.

In the present study, we leveraged data from 2 samples of Romanian preschool-aged children—a cross-sectional epidemiological sample[20] and a longitudinal sample of children exposed to severe early-life deprivation with a never-institutionalized comparison group[21] (the Bucharest Early Intervention Project [BEIP])—to examine 3 primary objectives. Objective 1 tested the bifactor structure of psychopathology in early childhood against single and correlated factors models. Objective 2 tested whether and how RAD and DSED symptoms fit into the general and specific factors, including whether RAD and DSED might form their own specific factor. Finally, using the longitudinal data available in the BEIP sample, objective 3 examined antecedents and outcomes associated with general and specific psychopathology. In the BEIP sample, a bifactor model of psychopathology has been previously demonstrated, with strong to moderate stability from middle childhood to adolescence.[22] We extended these findings by examining the bifactor structure of psychopathology within this sample during the preschool period. In addition, we explored whether early psychosocial deprivation and caregiving quality were associated with general and specific psychopathology in early childhood, and whether general and specific psychopathology in early childhood were associated with psychopathology, competence, and cognitive ability at ages 8 and 12 years.

Finally, the bifactor model of psychopathology has been questioned. In particular, concerns related to statistical issues such as over-fitting and difficulty replicating models across samples (particularly in regard to the specific factors)[23,24] have been cited. There are also questions about the value of bifactor models in relation to traditional models of psychopathology for clinical practice.[24–27] Thus, in a final exploratory analysis, we examined whether the effect sizes for the general factor were meaningfully stronger than a total sum of symptoms in explaining variation in later childhood outcomes at ages 8 and 12 years.

METHOD

Participants

Epidemiological Sample.—Participants included 350 Romanian children aged 18 to 60 months (mean = 39.7 months, SD = 10.9) recruited from a public outpatient pediatric

clinics in Bucharest and enrolled in a study examining the epidemiology of psychiatric disorders among preschool-aged children.[20] Initially, a total of 1,003 children were screened. The original and present analysis used 350 children selected for follow-up psychiatric interviews, a subsample enriched for psychopathology (Figure S1, available online). A majority were male (n = 185, 53%) and of Romanian ethnicity (93%). Expanded details about the epidemiological sample are provided in Supplement 1, available online.

BEIP Sample.—Participants included 158 children from the BEIP.[21] The original BEIP trial consisted of 136 institutionalized children and 72 never-institutionalized community comparison children (never-institutionalized group [NIG]). The NIG sample was recruited from the same hospitals in which the children in institutional care were born. The 136 ever-institutionalized children were randomly assigned at baseline to care-as-usual in the institution (care-as-usual group [CAUG], n = 68) or placement into a foster care system created by the BEIP (foster care group [FCG], n = 68). We used a policy of non-interference across the study, meaning that if an opportunity became available for a child randomized to institutional care to enter a family (adoptive, biological, or foster), we did not interfere. Thus, children were allowed to freely move from institutional care to family placements. Upon entry to the study, all children were assessed at baseline, which occurred when children were on average 22 months of age (range, 6–31 months). Randomization occurred immediately after the baseline assessment. During the trial, assessments were conducted at ages 30, 42, and 54 months. For the current study, analyses were completed on 158 children with complete data at age 54 months. Follow-up data were collected at ages 8 and 12 years to assess the long-term effects of psychosocial deprivation and the impact of the foster care intervention on outcomes across development.

Procedures

Epidemiological Sample.—Parents consented and then completed a demographics questionnaire and screening measure. Caregiver interviews of psychiatric symptoms were completed by female primary caregivers.

BEIP Sample.—Foster parents were recruited and trained by BEIP personnel to provide high-quality care to the children, which included explicit encouragement to make a psychological commitment to the children in their care.[21,28] Consent for participation was obtained at the beginning of the study and at each subsequent assessment from each child's legal guardian. Assent was obtained from each participant at ages 8, 12, and 16 years.

Data for the present study were drawn from caregiver reports of psychopathology at child age 54 months. Caregiver interviews were completed by each child's primary caregiver: biological, foster, or adoptive mothers, or by the institutional staff member who best knew the child. In anticipation of reporting challenges related to familiarity with the child, we ensured that the institutional staff providing reports on a given child were those who knew the child well and interacted with them frequently. At the 8- and 12-year follow-up assessments, data on multiple constructs were collected to determine long-term risk conferred by early deprivation and random assignment to foster care.

Measures: Epidemiological and BEIP Sample

Preschool Age Psychiatric Assessment.—The Preschool Age Psychiatric Assessment (PAPA)[29,30] is a structured caregiver report of psychiatric symptoms of *DSM-IV* psychiatric disorders among preschool-aged children. The PAPA can be used to make categorical diagnoses and also provides symptom counts within each disorder, the latter of which was used in the present study. The PAPA has demonstrated test—retest reliability comparable to those of other psychiatric interviews designed to assess older children and adults.[30] This was used in the current study at age 54 months.

PAPA items were translated into Romanian, then back translated into English and assessed for meaning at each step by bilingual research staff. Symptom counts for the following were used in analyses: oppositional defiant disorder (ODD), conduct disorder (CD), attention-deficit/hyperactivity disorder (ADHD), depression, separation anxiety, generalized anxiety disorder (GAD), specific phobia, and posttraumatic stress disorder (PTSD). Although social phobia symptoms were assessed, a very low base rate was observed and was thus excluded from all models.

Disturbances of Attachment Interview.—Signs of RAD and DSED at age 54 months were assessed using the Disturbances of Attachment Interview (DAI),[31–34] a validated caregiver-reported semi-structured interview of signs of disordered attachment. The DAI was translated into Romanian, back-translated into English, and assessed for meaning at each step by bilingual research staff. Five items assessed RAD and 3 items assessed DSED. Higher scores indicated more signs of RAD and DSED.

Measures: BEIP Sample—Specific Measures

The following were measured in the BEIP sample only. Given the large number of antecedents and outcomes included, detailed information is provided in Supplement 1, available online.

Early Childhood Antecedents.—A range of early childhood antecedents pertaining to child characteristics (eg, birth weight), aspects of the institutional care environment (eg, age of initial placement into institutional care and later placement into family care), and early caregiving experiences (eg, caregiving quality [30 and 42 months], security of attachment [42 months]) were assessed. See Supplement 1 available online for more information.

Later Childhood Outcomes.—Cognitive ability at 8 and 12 years, a composite of competent functioning across multiple domains at 8 and 12 years, including peer and family relations, academics, physical health, etc, and psychopathology at 12 years were assessed. Supplement 1, available online, provides more information.

Data Analysis

Objective 1: To Test the Structure of Psychopathology in Samples 1 and 2.—We used confirmatory factor analyses to compare the same series of competing models in both samples. The first 2 models were first-order models with either 2 (model 1A) or 3 (model 1B) correlated factors. In model 1A, the factors were for externalizing (ODD, CD,

inattention, and hyperactivity/impulsivity) and internalizing (depression, GAD, separation anxiety, specific phobia, and PTSD) symptoms. In model 1B, the same externalizing factor was modeled with separate fear (separation anxiety, specific phobia, and PTSD) and distress (depression and GAD) factors.

We then tested multiple bifactor models[35,36] with factors constrained to be uncorrelated (ie, orthogonal). Two "traditional" (ie, symmetrical) bifactor models (models 2A and 2B) were tested. Model 2A comprised 1 general factor and 2 specific factors: externalizing and internalizing (similar to model 1A). Model 2B comprised 1 general factor and 3 specific factors: externalizing, fear, and distress (similar to model 1B). Symmetrical bifactor models present statistical and conceptual challenges.[23,37,38] Conceptually, the meaning of the general factor can vary depending on the specific constructs (ie, symptoms, syndromes, or disorders) included in each study. One strategy to avoid these issues is to have at least 1 indicator that loads only on the general factor and not on any specific factor. One of these models is the bifactor SI-1 model. Models 3A and 3B are similar to models 2A and 2B, respectively, with the exception of having 1 indicator (ie, inattention) that loads only on the general factor.

Objective 2: To Test the Addition of RAD and DSED to the Best-Fitting Model in Samples 1 and 2.—Using the model identified as preferable in objective 1, we tested whether signs of RAD and DSED are best conceptualized as forming their own specific factor, which we called "disturbed relatedness."

Objective 3: To Examine Antecedents and Outcomes of General and Specific Forms of Psychopathology in the BEIP Sample.—We used the optimally identified model in Objective 2 to perform correlations and regressions. We also compared the mean levels of psychopathology across the 3 groups (ie, NIG, FCG, CAUG).

Model Estimation, Evaluation, and Analysis.—Models were estimated in Mplus (Version 8.4). In all analyses, symptom counts for all syndromes were specified as categorical. We specified to the software that the data are categorical (which applies to ordinal data) rather than to treat the scores as continuous. This is useful here because treating the data as continuous would lead to bias such as floor effects (ie, many zeroes), as is the case here. For objectives 1 and 2, we used Bayesian estimation. Estimation problems were encountered for some models when using 2 commonly used estimators, the weighted least-squares estimator with means and variance adjustment (WLSMV) and maximum likelihood estimation with robust standard errors (MLR). This was probably due to the small sample sizes. Model convergence was determined by ensuring that the potential scale reduction (PSR) reached the default value in Mplus.[39] Because we used Bayesian estimation and treated the data as categorical, model fit was evaluated with the posterior predictive p (PPP) value, which indicates whether the model does not fit the data well.[39] Values below 0.05 would indicate that the model does not fit the data well, whereas values above 0.05 and closer to 0.50 are preferred. The Bayesian PPP value is not interpreted exactly like the typical χ^2 test of model fit. A model should be rejected if its PPP value is below 0.05, whereas a model with excellent fit should have a PPP value close to 0.5, with a wide confidence interval where zero is close to the midpoint.[40] In other words, a PPP value

that is larger than 0.05 and closer to 0.50 indicates that the model fits the data well. For objective 3, we used the maximum likelihood estimation with robust standard errors (MLR) estimator because convergence problems were encountered when using Bayesian estimation. To compare the differences in the means of the latent factors, we used multiple imputation with 20 imputations to obtain the plausible values of the latent variables.

RESULTS

Objective 1

Fit indices and factor loadings are reported in Tables S1 and S2, available online. In both samples, models 1A and 1B did not fit the data well (PPP < 0.05). Fit indices and factor loadings for symmetrical bifactor models 2A and 2B are reported in Tables S1 and S3, available online. The PPP values for these models were above 0.05 in both samples, indicating acceptable fit. Albeit not statistically significant, inattention loaded negatively on the specific externalizing factor. For model 2A, factor loadings on internalizing were moderate in both samples but statistically significant only in the epidemiological sample, which could be due to the smaller sample size in the BEIP. In model 2B, the factor loadings on the distress factor were small and nonsignificant in both samples. Thus, neither the fit indices nor the factor loadings favored model 2B, and model 2A was deemed preferable over 2B.

Bifactor SI-1 models 3A and 3B were similar to models 2A and 2B, respectively, with the exception that inattention loaded only on the general factor and not on the externalizing factor. Fit indices and factor loadings (Tables S1 and S4, available online) were similar to models 2A and 2B, although there were no longer any negative loadings now that inattention was no longer required to load onto the externalizing factor. Overall, models 2A and 3A were deemed preferable to models 2B and 3B. Because of the potential limitations of symmetrical bifactor models, we selected model 3A for objectives 2 and 3.

Objective 2

Building on model 3A, we tested 2 variants that included signs of RAD and DSED. Model 4A included signs of RAD and DSED on the general factor and the internalizing-specific factor (preliminary analyses showed that they were more strongly associated with internalizing compared to externalizing symptoms). Model 4B included RAD and DSED on the general factor and on a “disturbed relatedness” specific factor.

Evidence supporting the benefit of the disturbed relatedness factor was mixed. The fit of models 4A and 4B were virtually identical in both samples. Except for RAD and DSED, factor loadings were also almost identical across models 4A and 4B in both samples (Table S5, available online). The most notable difference between samples was that the addition of a disturbed relatedness factor to account for an association between RAD and DSED appeared to be pertinent only in the BEIP sample, where such conditions are directly tied to a history of deprivation. Thus, model 4A (Table 1) was the most appropriate model for the epidemiological sample, and model 4B was most appropriate in the BEIP sample (Table 2)

and thus was the model selected for objective 3, which was evaluated using only the BEIP sample.

Objective 3

Convergence problems were encountered when using Bayesian estimation. For antecedents (ie, differences in means among NIG, GCF, and CAUG), we used a 2-step approach: (1) multiple imputation with 20 imputations to obtain the plausible values of the latent variables; and (2) analysis of the multiply imputed data with MLR. Dummy variables indexing group membership were entered as covariates to test differences in means. For outcomes, we used a single-step approach in which the best-fitting model was allowed to correlate with the longitudinal outcomes. Bivariate associations between early childhood antecedents and the general and specific factors are presented in Table 3. Associations with outcomes at ages 8 and 12 years are shown in Table 4. Post hoc regression analyses indicated that the general factor was significantly associated with all 8-and 12-year outcomes, and was often the only statistically significant latent factor score associated with the outcomes (Table 5).

We then compared psychopathology across the latent means in each group (Table S6, available online). Differences were interpreted using the Cohen *d*.[41] Significant differences emerged only on the general psychopathology factor, with large to very large differences between the NIG compared to FCG ($d = -0.90$) and CAUG ($d = -1.12$), which did not differ from one another. For disturbed relatedness, a moderate effect size difference (albeit not statistically significant) emerged between the NIG and CAUG ($d = -0.47$, $p = .065$).

Finally, we examined associations between outcomes at 8 and 12 years with a simple sum of all psychopathology symptoms (Table S7, available online). The effect sizes and significance for the simple and score were similar to, but slightly smaller than, those based on the general psychopathology factor. When included as simultaneous independent variables in a regression model, the general psychopathology factor score concealed the effect of the simple sum, although some confidence intervals overlapped.

DISCUSSION

Examining data across 2 samples of preschool-aged Romanian children, the present study adds to the growing body of literature on the structure of psychopathology in early childhood. The best-fitting model in both samples was one that included the placement of inattention symptoms on the general factor only, thus specifying a bifactor SI-1 model. A bifactor SI-1 model with inattention on the general factor mirrors past work that also supported this structure of childhood psychopathology.[18] Overall, the standardized factor loadings for the best -itting models in both samples mirrored past work in similar samples.[18,19] This was particularly true for the epidemiological sample, which is not surprising, given that past works included normative/community level samples more similar to the epidemiological sample.

The placement of inattention on the general factor only indicates that symptoms of inattention may be a broader or more general indicator of psychiatric difficulties in multiple domains in early childhood. Furthermore, hyperactivity/impulsivity loaded onto the

externalizing specific factor in the best-fitting model for both samples, but the loading was not statistically significant. This finding mirrors past work, both in the bi-factor literature,[40] and, more broadly,[42] that ADHD and externalizing disorders (eg, CD, ODD) are considered separate disorders. Furthermore, evidence from the BEIP indicates differential response to the intervention.[43]

Research on the structure of psychopathology in higher-risk samples of young children is extremely limited. Our findings in the BEIP sample, a sample of children exposed to severe early psychosocial deprivation with high levels of psychopathology, are somewhat dissimilar to findings in community samples regarding the composition of the specific factors. Specifically, we did not find support for symptoms of inattention loading onto the externalizing factor. Methodological differences (ie, in the measurement of symptoms) may partially explain the differences in the structure of psychopathology across different studies. Furthermore, for the BEIP sample, many of the internalizing disorder symptoms did not significantly load onto the internalizing specific factor but did load significantly onto the general factor. Thus, it could be that in early childhood, internalizing symptoms are less differentiated and largely captured by the general factor.

The decision to include RAD and DSED symptoms in the models originated from our aim to explore general and specific forms of psychopathology in the BEIP sample, in which RAD and DSED are particularly relevant. In addition, RAD and DSED are disorders of early childhood that are relevant to this period of development. The issue of a low base rate in the community (reviewed earlier) is reflected in RAD not loading significantly onto the general factor in the epidemiological sample. In contrast, DSED loaded significantly onto the general factor in the epidemiological sample, suggesting that it shares variance with other disorders of early childhood, which is consistent with the strong association between DSED and impulsivity seen in ADHD in clinical samples.[34] Neither RAD nor DSED loaded significantly onto the disturbed relatedness factor in the epidemiological sample, suggesting that a disturbed relatedness specific factor may not be supported in non-clinical samples. There is much future work to be done examining the addition of RAD and DSED to the structure of psychopathology in early childhood. One interesting future direction is exploring whether RAD and DSED might be considered within the HiTOP framework along the lines of the work by Wright *et al.*[44] on personality disorders as interpersonal disorders.

Providing enriched early caregiving experiences for those with a history of early deprivation and neglect may reduce risk for general and specific forms of psychopathology. Moreover, Forbes *et al.*[45] have suggested that preventing the development of P requires early intervention (ages 3–6 years) through targeting cross-cutting skills such as executive function, emotional reactivity, parenting behavior, and peer relationship skills. Similarly, Carver *et al.*[26] suggested that impulsive reactivity to emotion may be a core underlying process in the development of the general factor during childhood, and Olino *et al.*[17] showed that emotion regulation difficulties were associated with the general factor in 3-year-old children. The development of emotion regulation skills begins in infancy with the co-regulation of emotions by primary caregivers (see Humphreys *et al.*[46] for a review). Thus, interventions to prevent risk for the development of general psychopathology should begin early, through building positive caregiver—infant relationships that help to

scaffold the executive and self-regulatory skills that guard against the development of later psychopathology.

Scores on the general factor were highest among children randomized to CAUG followed by children randomized to the FCG, and were markedly lower among community comparison children with no exposure to institutional care (ie, NIG). There were no statistically significant differences in scores on the specific factors between groups; however, the difference in scores on the disturbed relatedness factor between the NIG and CAUG groups was close to the significant range. This is consistent with prior work on the structure of psychopathology by our group using teacher and caregiver ratings, in which differences in the general factor were not observed at 8 years but began to manifest at 12 years and were apparent by 16 years.[22] Thus, there do not appear to be meaningful differences in the general factor as a function of the foster care intervention prior to the adolescent transition, which might represent a period of heightened plasticity in which the benefits of family care following early deprivation become more fully realized. This is consistent with the idea of a "sleeper" effect, which, interestingly, has also been observed for self-control in the BEIP, a phenotype that mediates the effects of the foster care intervention on general psychopathology at 16 years.[47]

Previously, in an examination of foster care intervention effects on psychopathology at age 54 months using total symptom counts (ie, not bifactor modeling), we found higher rates of internalizing disorders among the CAUG compared to the FCG.[48] The internalizing specific factor in the present study was "weak" (ie, only symptoms of depression and PTSD loaded significantly on this factor), likely due to little variance in internalizing symptoms after being accounted for on the general factor, which may have limited our ability to detect similar differences between groups on rates of internalizing disorders.

The difference between the CAUG and FCG groups on the disturbed relatedness factor was marginally significant, mirroring previous symptom count—based work in the BEIP, particularly for RAD.[31] Some of the variance in DSED and RAD was shared with other domains of psychopathology (via the general factor), whereas additional variance was specific to these disorders. This suggests that, in addition to generalized mechanisms of symptom improvement, family-based intervention may ameliorate some of the difficulties that are a consequence of early deprivation. Characterizing the mechanisms accounting for these improvements should be a focus of subsequent research.

Finally, it is worth noting that findings were inconclusive regarding whether the general factor measured in early childhood is a stronger or "better" indicator of outcomes in late childhood compared to a standard total symptom count. This is notable, given ongoing criticism and uncertainty about the clinical utility of "P" scores.[26,27] One possibility is that the general factor is capturing something about the extent or severity of psychiatric difficulties as opposed to a particular shared etiology.[1] Thus, some have suggested that scores on the general factor may shed light on the level of mental health impairment and thus the need for mental health treatment, such as how long and intense treatment may need to be to have an impact.[49] Future work comparing the bifactor structure to more traditional

measures of psychopathology in regard to outcomes will be helpful in understanding the utility of bifactor models for clinical practice.

This study has several strengths, including 2 different samples of preschool-aged children that used the same assessment of psychopathology, one of which was a clinical sample, allowing for the examination of antecedents and outcomes of general and specific factors in a sample at risk for poor psychiatric outcomes. One limitation is that both samples were collected in Romania, and thus results may not generalize to other cultures. Next, in the BEIP sample, the institutional caregivers who were asked to report on children's behavior did change over the course of the study in both the care-as-usual group and the foster care group, which is a limitation of our measurement of psychopathology. Our sample sizes were on the smaller side, and although we used rigorous methods given this issue, our results should be interpreted with caution. However, we are not aware of a comparable sample, and thus we acknowledge both the limitations related to sample size and that imperfect information can still be useful. More work with larger samples is needed to further this line of work. Furthermore, the age range in sample 1 was quite large and may have contributed to differences between studies. Previous work has demonstrated consistency for the general factor across early childhood,[16,19] but more work is needed to understand change in this factor across early development. Finally, severe psychosocial deprivation is a rare experience in the general population, and therefore the antecedents and outcomes associated with a general factor, and disturbed relatedness specifically, may differ in non-deprived samples. Future work across clinical samples will provide greater context to antecedents and outcomes across development.

In conclusion, we have provided support for a bifactor structure of psychopathology in early childhood in a low-risk epidemiological sample and a high-risk sample of children exposed to early deprivation. We further demonstrated the importance of including RAD and DSED symptoms in the modeling, particularly for samples of children exposed to early caregiving adversity. Much remains to be learned about the structure of psychopathology in early childhood and its utility for clinical practice, including what it might add beyond a more traditional and easier-to-compute total symptom count. Still, it is clear that general psychopathology factor scores are a powerful early indicator of risk for poor developmental outcomes, and that supporting healthy caregiver–child relationships remains an important target for interventions that aim to reduce risk for psychopathology across development.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Disclosure:

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REFERENCES

1. Caspi A, Houts RM, Belsky DW, et al. The p factor: one general psychopathology factor in the structure of psychiatric disorders? Clin Psychol Sci. 2014;2(2):119–137. 10.1177/2167702613497473 [PubMed: 25360393]
2. Caspi A, Moffitt TE. All for One and One for All: Mental Disorders in One Dimension. American Psychiatric Association; 2018:831–844.
3. Kotov R, Waszczuk MA, Krueger RF, et al. The Hierarchical Taxonomy of Psychopathology (HiTOP): a dimensional alternative to traditional nosologies. J Abnorm Psychol. 2017;126(4):454–477. 10.1037/abn0000258 [PubMed: 28333488]
4. Lahey BB, Applegate B, Hakes JK, Zald DH, Hariri AR, Rathouz PJ. Is there a general factor of prevalent psychopathology during adulthood? J Abnorm Psychol. 2012;121(4): 971–977. 10.1037/a0028355 [PubMed: 22845652]
5. Lahey BB, Krueger RF, Rathouz PJ, Waldman ID, Zald DH. A hierarchical causal taxonomy of psychopathology across the life span. Psychol Bull. 2017;143(2):142–186. 10.1037/bul0000069 [PubMed: 28004947]
6. Kotov R, Krueger RF, Watson D, et al. The Hierarchical Taxonomy of Psychopathology (HiTOP): a quantitative nosology based on consensus of evidence. Ann Rev Clin Psychol. 2021;17(1):83–108. 10.1146/annurev-clinpsy-081219-093304 [PubMed: 33577350]
7. Kotov R, Krueger RF, Watson D, et al. The Hierarchical Taxonomy of Psychopathology (HiTOP): a dimensional alternative to traditional nosologies. J Abnorm Psychol. 2017; 126:454–477. 10.1037/abn0000258 [PubMed: 28333488]
8. Kotov R, Jonas KG, Carpenter WT, et al. Validity and utility of Hierarchical Taxonomy of Psychopathology (HiTOP): I. Psychosis superspectrum. World Psychiatry. 2020; 19(2):151–172. 10.1002/wps.20730 [PubMed: 32394571]
9. Deutz MHF, Geeraerts SB, Belsky J, et al. General Psychopathology and dysregulation profile in a longitudinal community sample: stability, antecedents and outcomes. Child Psychiatry Hum Dev. 2020;51(1):114–126. 10.1007/s10578-019-00916-2 [PubMed: 31359330]
10. Laceulle OM, Vollebergh WAM, Ormel J. The structure of psychopathology in adolescence: replication of a general psychopathology factor in the TRAILS study. Clin Psychol Sci. 2015;3(6):850–860. 10.1177/2167702614560750
11. Patalay P, Fonagy P, Deighton J, Belsky J, Vostanis P, Wolpert M. A general psychopathology factor in early adolescence. Br J Psychiatry. 2015;207(1):15–22. 10.1192/bjp.bp.114.149591 [PubMed: 25906794]
12. Sallis H, Szekely E, Neumann A, et al. General psychopathology, internalising and externalising in children and functional outcomes in late adolescence. J Child Psychol Psychiatry. 2019;60(11):1183–1190. 10.1111/jcpp.13067 [PubMed: 31049953]

13. Shields AN, Reardon KW, Brandes CM, Tackett JL. The p factor in children: relationships with executive functions and effortful control. J Res Personal. 2019;82: 103853–103853. 10.1016/j.jrp.2019.103853

14. Carter AS, Briggs-Gowan MJ, Jones SM, Little TD. The Infant-Toddler Social and Emotional Assessment (ITSEA): factor structure, reliability, and validity. J Abnorm Child Psychol. 2003;31(5):495–514. 10.1023/A:1025449031360 [PubMed: 14561058]

15. Grotzinger AD, Cheung AK, Patterson MW, Harden KP, Tucker-Drob EM. Genetic and environmental links between general factors of psychopathology and cognitive ability in early childhood. Clin Psychol Sci. 2019;7(3):430–444. 10.1177/2167702618820018 [PubMed: 31440427]

16. McElroy E, Belsky J, Carragher N, Fearon P, Patalay P. Developmental stability of general and specific factors of psychopathology from early childhood to adolescence: dynamic mutualism or p-differentiation? J Child Psychol Psychiatry Allied Discip. 2018; 59(6). 10.1111/jcpp.12849

17. Olino TM, Dougherty LR, Bufferd SJ, Carlson GA, Klein DN. Testing models of psychopathology in preschool-aged children using a structured interview-based assessment. J Abnorm Child Psychol. 2014;42(7):1201–1211. 10.1007/s10802-014-9865-x [PubMed: 24652485]

18. Wade M, Plamondon A, Jenkins JM. A family socialization model of transdiagnostic risk for psychopathology in preschool children. Res Child Adolesc Psychopathol. 2021;49(8): 975–988. 10.1007/s10802-021-00789-x [PubMed: 33687647]

19. Olino TM, Bufferd SJ, Dougherty LR, Dyson MW, Carlson GA, Klein DN. The development of latent dimensions of psychopathology across early childhood: stability of dimensions and moderators of change. J Abnorm Child Psychol. 2018;46(7):1373–1383. 10.1007/s10802-018-0398-6 [PubMed: 29359267]

20. Gleason MM, Zamfirescu A, Egger HL, Nelson CA, Fox NA, Zeanah CH. Epidemiology of psychiatric disorders in very young children in a Romanian pediatric setting. Eur Child Adolesc Psychiatry. 2011;20(10):527–535. 10.1007/s00787-011-0214-0 [PubMed: 21866415]

21. Zeanah CH, Nelson CA, Fox NA, et al. Designing research to study the effects of institutionalization on brain and behavioral development: the Bucharest Early Intervention Project. Dev Psychopathol. 2003;15(04):885–907. 10.1017/S0954579403000452 [PubMed: 14984131]

22. Wade M, Fox NA, Zeanah CH, Nelson CA. Effect of foster care intervention on trajectories of general and specific psychopathology among children with histories of institutional rearing. JAMA Psychiatry. 2018;75(11):1137–1137. 10.1001/jamapsychiatry.2018.2556 [PubMed: 30267045]

23. Eid M, Geiser C, Koch T, Heene M. Anomalous results in G-factor models: explanations and alternatives. Psychol Methods. 2017;22(3):541. [PubMed: 27732052]

24. Watts AL, Poore HE, Waldman ID. Riskier tests of the validity of the bifactor model of psychopathology. Clin Psychol Sci. 2019:216770261985503–216770261985503. 10.1177/2167702619855035

25. Greene AL, Eaton NR, Li K, et al. Are fit indices used to test psychopathology structure biased? A simulation study. J Abnorm Psychol. 2019;128(7):740–764. 10.1037/abn0000434 [PubMed: 31318246]

26. Carver CS, Johnson SL, Timpano KR. Toward a functional view of the p factor in psychopathology. Clin Psychol Sci. 2017;5(5):880–889. 10.1177/2167702617710037 [PubMed: 29057170]

27. Levin-Aspenson HF, Watson D, Clark LA, Zimmerman M. What is the general factor of psychopathology? Consistency of the p factor across samples. Assessment. 2021;28(4): 1035–1049. 10.1177/1073191120954921 [PubMed: 32909442]

28. Smyke AT, Zeanah CH, Fox NA, Nelson CA. A new model of foster care for young children: the Bucharest Early Intervention Project. Child Adolesc Psychiatr Clin North Am. 2009;18:721–734. 10.1016/j.chc.2009.03.003

29. Egger HL, Angold A. The preschool age psychiatric assessment (PAPA): a structured parent interview for diagnosing psychiatric disorders in preschool children. In: DelCarmen-Wiggins R, Carter A, eds. Handbook of Infant, Toddler, and Preschool Mental Health Assessment. Oxford: Oxford University Press; 2004:223–243.

30. Egger HL, Erkanli A, Keeler G, Potts E, Walter BK, Angold A. Test-retest reliability of the Preschool Age Psychiatric Assessment (PAPA). J Am Acad Child Adolesc Psychiatry. 2006;45(5):538–549. 10.1097/01.chi.0000205705.71194.b8 [PubMed: 16601400]

31. Gleason MM, Fox NA, Drury S, et al. Validity of evidence-derived criteria for reactive attachment disorder: indiscriminately social/disinhibited and emotionally withdrawn/inhibited types. J Am Acad Child Adolesc Psychiatry. 2011;50(3):216–231. 10.1016/j.jaac.2010.12.012 [PubMed: 21334562]

32. Smyke AT, Dumitrescu A, Zeanah CH. Attachment disturbances in young children. I: the continuum of caretaking casualty. J Am Acad Child Adolesc Psychiatry. 2002;41(8): 972–982. 10.1097/00004583-200208000-00016 [PubMed: 12162633]

33. Zeanah CH, Smyke AT, Dumitrescu A. Attachment disturbances in young children. II: indiscriminate behavior and institutional care. J Am Acad Child Adolesc Psychiatry. 2002;41(8):983–989. 10.1097/00004583-200208000-00017 [PubMed: 12162634]

34. Zeanah CH, Smyke AT, Koga SF, Carlson E. The Bucharest Early Intervention Project Core Group. Attachment in institutionalized and community children in Romania. Child Dev. 2005;76(5):1015–1028. 10.1111/j.1467-8624.2005.00894.x [PubMed: 16149999]

35. Holzinger KJ, Swineford F. The bi-factor method. Psychometrika. 1937;2(1):41–54. 10.1007/BF02287965

36. Schmid J, Leiman JM. The development of hierarchical factor solutions. Psychometrika. 1957;22(1):53–61. 10.1007/BF02289209

37. Watts AL, Lane SP, Bonifay W, Steinley D, Meyer FA. Building theories on top of, and not independent of, statistical models: the case of the p-factor. Psychol Inquiry. 2020; 31(4):310–320.

38. Burns GL, Geiser C, Servera M, Becker SP, Beauchaine TP. Application of the bifactor S–1 model to multisource ratings of ADHD/ODD symptoms: an appropriate bifactor model for symptom ratings. J Abnorm Child Psychol. 2020;48(7):881–894. [PubMed: 31834589]

39. Muthén B, Asparouhov T. Bayesian structural equation modeling: a more flexible representation of substantive theory. Psychol Methods. 2012;17(3):313. [PubMed: 22962886]

40. Hand ED, Lonigan CJ. Examining the relations between preschoolers' externalizing behaviors and academic performance using an S-1 bifactor model. Res Child Adolesc Psychopathol. 2022;50(5):577–589. 10.1007/s10802-021-00861-6 [PubMed: 34460050]

41. Sawilowsky SS. New effect size rules of thumb. J Modern Appl Stat Methods. 2009; 8(2):26.

42. Nigg JT, Sibley MH, Thapar A, Karalunas SL. Development of ADHD: etiology, heterogeneity, and early life course. Ann Rev Dev Psychol. 2020; 2(1):559. [PubMed: 34368774]

43. Humphreys KL, Gleason MM, Drury SS, et al. Effects of institutional rearing and foster care on psychopathology at age 12 years in Romania: follow-up of an open, randomised controlled trial. Lancet Psychiatry. 2015;2(7):625–634. 10.1016/S2215-0366(15)00095-4 [PubMed: 26303560]

44. Wright AG, Ringwald WR, Hopwood C, Pincus AL. It's time to replace the personality disorders with the interpersonal disorders. PsyArXiv Preprints. 2022. 10.31234/osf.io/7syvf

45. Forbes MK, Rapee RM, Krueger RF. Opportunities for the prevention of mental disorders by reducing general psychopathology in early childhood. Behav Res Ther. 2019; 119:1–8. 10.1016/j.brat.2019.103411

46. Humphreys KL, King LS, Guyon-Harris KL, Zeanah CH. Caregiver regulation: a modifiable target promoting resilience to early adverse experiences. Psychol Trauma. 2022;14(S1):S63. [PubMed: 34460282]

47. Mukerji CE, Wade M, Fox NA, Zeanah CH, Nelson CA. Growth in self-regulation over the course of adolescence mediates the effects of foster care on psychopathology in previously institutionalized children: a randomized clinical trial. Clin Psychol Sci. 2021; 9(5):810–822. 10.1177/2167702621993887 [PubMed: 34888125]

48. Zeanah CH, Egger HL, Smyke AT, et al. Institutional rearing and psychiatric disorders in Romanian preschool children. Am J Psychiatry. 2009;166(7):777–785. 10.1176/appi.ajp.2009.08091438 [PubMed: 19487394]

49. Smith GT, Atkinson EA, Davis HA, Riley EN, Oltmanns JR. The general factor of psychopathology. Annu Rev Clin Psychol. 2020;16(1). 10.1146/annurev-clinpsy-071119-115848

TABLE 1

Factor Loadings and Cross-Loadings for the Best-Fitting Model in the Epidemiological Sample

Symptom	General factor		Externalizing		Internalizing	
ODD	**0.62**	[0.51; 0.72]	**0.46**	[0.28; 0.64]		
CD	**0.4**	[0.24; 0.55]	**0.63**	[0.38; 0.80]		
Inattention	**0.81**	[0.73; 0.87]				
Hyperactivity/impulsivity	**0.80**	[0.73; 0.86]	0.03	[−0.12; 0.18]		
Depression	**0.75**	[0.65; 0.82]			**0.23**	[0.05; 0.39]
GAD	**0.88**	[0.82; 0.92]			**0.21**	[0.08; 0.33]
Separation anxiety	**0.37**	[0.22; 0.50]			**0.41**	[0.21; 0.60]
Specific phobia	**0.31**	[0.10; 0.50]			**0.75**	[0.50; 0.90]
PTSD	**0.37**	[0.19; 0.54]			**0.45**	[0.23; 0.66]
RAD	0.20	[−0.11; 0.49]			**0.46**	[0.05; 0.77]
DSED	**0.56**	[0.37; 0.71]			0.13	[−0.15; 39]

Note: Values in square brackets are 95% CIs. The best-fitting model in the epidemiological sample was model 4A (ie, model with reactive attachment disorder and disinhibited social engagement disorder included on the internalizing factor). Values in boldface type are statistically significant based on the 95% CI. CD = conduct disorder; DSED = disinhibited social engagement; GAD = generalized anxiety disorder; ODD = oppositional defiant disorder; PTSD = posttraumatic stress disorder; RAD = reactive attachment disorder.

TABLE 2

Factor Loadings and Cross-Loadings for the Best-Fitting Model in the Bucharest Early Intervention Project Sample

Symptoms	General factor		Externalizing		Internalizing		Disturbed relatedness	
ODD	**0.70**	[0.57; 0.80]	**0.44**	[0.26; 0.63]				
CD	**0.49**	[0.30; 0.65]	**0.72**	[0.47; 0.87]				
Inattention	**0.87**	[0.79; 0.92]						
Hyperactivity/impulsivity	**0.86**	[0.79; 0.91]	0.11	[−0.04; 0.26]				
Depression	**0.83**	[0.73; 0.89]			**0.25**	[0.00; 0.47]		
GAD	**0.93**	[0.87; 0.96]			0.04	[−0.14; 0.23]		
Separation anxiety	**0.21**	[−0.02; 0.42]			0.34	[−0.07; 0.73]		
Specific phobia	**0.52**	[0.23; 0.74]			0.31	[−0.13; 0.66]		
PTSD	**0.41**	[0.16; 0.62]			**0.56**	[0.12; 0.82]		
RAD	**0.40**	[0.19; 0.58]					**0.33**	[0.05; 0.78]
DSED	**0.59**	[0.43; 0.72]					**0.68**	[0.17; 0.84]

Note: Values in brackets are 95% CIs. The best-fitting model in the Bucharest Early Intervention Project Sample was model 4B (ie, model with reactive attachment disorder and disinhibited social engagement disorder included on the disturbed relatedness factor). Values in boldface type are statistically significant based on the 95% CI. CD = conduct disorder; DSED = disinhibited social engagement; GAD = anxiety disorder; ODD = oppositional defiant disorder; PTSD = posttraumatic stress disorder; RAD = reactive attachment disorder.

TABLE 3

Bivariate Associations Between Model Factors and Early Childhood Antecedents in the Bucharest Early Intervention Project Sample

	General psychopathology		Externalizing		Internalizing		Disturbed relatedness	
	r	**95% CI**	***r***	**95% CI**	***r***	**95% CI**	***r***	**95% CI**
Birth weight	**−0.21**	[−0.40; −0.03]	0.09	[−0.15; 0.33]	−0.04	[−0.29; 0.20]	−0.04	[−0.34; 0.25]
Age at institutionalization	−0.13	[−0.36; 0.11]	−0.07	[−0.33; 0.19]	−0.06	[−0.34; 0.23]	−0.06	[−0.40; 0.27]
Age at entry into foster care	**0.46**	[0.27; 0.64]	0.02	[−0.27; 0.31]	0.17	[−0.24; 0.57]	0.36	[−0.37; 1.08]
Age at first family placement	0.11	[−0.10; 0.31]	0.11	[−0.12; 0.33]	0.08	[−0.17; 0.32]	**0.52**	[0.08; 0.97]
Percentage of time in institutional care at baseline	0.11	[−0.13; 0.35]	0.33	[−0.12; 0.78]	−0.05	[−0.38; 0.28]	0.16	[−0.27; 0.60]
Percentage of time in institutional care through 30 mo	**0.40**	[0.21; 0.60]	0.19	[−0.12; 0.49]	**0.30**	[0.03; 0.57]	**0.53**	[0.11; 0.94]
Percentage of time in institutional care through 42 mo	**0.36**	[0.17; 0.54]	0.10	[−0.18; 0.38]	**0.30**	[0.07; 0.53]	**0.66**	[0.23; 1.09]
Percentage of time in institutional care through 54 mo	**0.33**	[0.14; 0.52]	0.04	[−0.23; 0.31]	**0.29**	[0.06; 0.52]	**0.72**	[0.26; 1.19]
Caregiving disruptions through 30 mo	−0.03	[−0.30; 0.24]	−0.21	[−0.47; 0.04]	0.06	[−0.30; 0.41]	−0.21	[−0.58; 0.16]
Caregiving disruptions through 42 mo	0.22	[−0.05; 0.49]	**−0.28**	[−0.55; −0.01]	0.24	[−0.12; 0.60]	0.17	[−0.10; 0.44]
Caregiving disruptions through 54 mo	0.23	[−0.05; 0.50]	**−0.31**	[−0.58; −0.03]	0.32	[−0.11; 0.74]	0.15	[−0.18; 0.49]
Staff-rated caregiving quality at 54 mo	**−0.24**	[−0.40; −0.08]	0.00	[−0.21; 0.20]	**−0.41**	[−0.60; −0.21]	**−0.81**	[−1.18; −0.44]
ORCE caregiving quality at baseline	**−0.37**	[−0.52; −0.21]	0.03	[−0.19; 0.25]	**−0.25**	[−0.48; −0.02]	**−0.38**	[−0.69; −0.07]
ORCE caregiving quality at 30 mo	**−0.27**	[−0.41; −0.12]	−0.20	[−0.42; 0.03]	**−0.31**	[−0.54; −0.08]	−0.30	[−0.62; 0.02]
ORCE caregiving quality at 42 mo	−0.10	[−0.28; 0.08]	−0.01	[−0.22; 0.21]	**−0.32**	[−0.57; −0.07]	**−0.41**	[−0.77; −0.05]
Security score at 42 mo	**−0.57**	[−0.68; −0.46]	0.05	[−0.24; 0.34]	−0.34	[−0.73; 0.06]	**−0.69**	[−1.07; −0.30]

Note: Model factors come from the best-fitting model (model 4B). Values in boldface type are statistically significant based on the 95% CI. ORCE = Observational Record of the Caregiving Environment.

TABLE 4

Bivariate associations Between Model Factors and Outcomes at Ages 8 and 12 Years in the Bucharest Early Intervention Project Sample

	General psychopathology		Externalizing		Internalizing		Disturbed relatedness	
	r	**95% CI**	***r***	**95% CI**	***r***	**95% CI**	***r***	**95% CI**
Age 8 competence[a]	**−0.51**	[−0.65; −0.37]	−0.12	[−0.37; 0.12]	−0.17	[−0.39; 0.05]	**−0.44**	[−0.85; −0.02]
Age 12 competence[a]	**−0.57**	[−0.69; −0.45]	−0.09	[−0.39; 0.20]	−0.32	[−0.54; −0.09]	**−0.55**	[−0.90; −0.20]
Age 8 IQ	**−0.61**	[−0.75; −0.47]	−0.09	[−0.40; 0.22]	**−0.41**	[−0.67; −0.15]	**−0.47**	[−0.84; −0.10]
Age 12 IQ	**−0.58**	[−0.72; −0.45]	0.02	[−0.34; 0.37]	**−0.31**	[−0.58; −0.05]	−0.40	[−0.83; 0.04]
Age 12 Internalizing symptoms	**0.20**	[0.03; 0.37]	−0.26	[−0.58; 0.07]	**0.17**	[0.00; 0.33]	**0.35**	[0.02; 0.67]
Age 12 ADHD symptoms	**0.48**	[0.33; 0.63]	0.04	[−0.25; 0.34]	0.12	[−0.11; 0.35]	**0.38**	[0.11; 0.64]
Age 12 CD and ODD symptoms	**0.28**	[0.14; 0.43]	**0.31**	[0.07; 0.55]	0.18	[−0.03; 0.38]	**0.35**	[0.01; 0.69]

Note: Model factors come from the best-fitting model (model 4B). Values in boldface type are statistically significant based on the 95% CI. ADHD = attention-deficit/hyperactivity disorder; CD = conduct disorder; IQ = intelligence quotient; ODD = oppositional defiant disorder.

[a]Ages 8 and 12 competence composite included 7 domains (peer relations, family relations, academics, physical health, mental health impairment, risk taking, and substance use).

TABLE 5

Regression Models With General and Specific Factors Predicting Outcomes at Ages 8 and 12 Years in the Bucharest Early Intervention Project Sample

	β	95% CI
Age 8 competence[a]		
General psychopathology	**−0.51**	[−0.65; −0.36]
Externalizing	−0.01	[−0.21; 0.19]
Internalizing	**0.27**	[0.06; 0.49]
Disturbed relatedness	−0.23	[−0.57; 0.10]
Age 12 competence[a]		
General psychopathology	**−0.56**	[−0.68; −0.44]
Externalizing	0.02	[−0.21; 0.24]
Internalizing	−0.04	[−0.26; 0.18]
Disturbed relatedness	−0.33	[−0.78; 0.11]
Age 8 IQ		
General psychopathology	**−0.61**	[−0.76; −0.46]
Externalizing	0.06	[−0.19; 0.30]
Internalizing	0.21	[−0.04; 0.47]
Disturbed relatedness	−0.24	[−0.51; 0.03]
Age 12 IQ		
General psychopathology	**−0.60**	[−0.75; −0.46]
Externalizing	0.16	[−0.13; 0.45]
Internalizing	0.19	[−0.05; 0.42]
Disturbed relatedness	−0.16	[−0.46; 0.15]
Age 12 Internalizing symptoms		
General psychopathology	**0.21**	[0.04; 0.38]
Externalizing	**−0.29**	[−0.57; 0.00]
Internalizing	0.11	[−0.11; 0.34]
Disturbed relatedness	**0.29**	[0.01; 0.57]
Age 12 ADHD symptoms		
General psychopathology	**0.48**	[0.32; 0.64]
Externalizing	−0.03	[−0.29; 0.22]
Internalizing	−0.03	[−0.26; 0.19]
Disturbed relatedness	0.18	[−0.09; 0.45]
Age 12 CD and ODD symptoms		
General psychopathology	**0.24**	[0.09; 0.39]
Externalizing	**0.28**	[0.04; 0.52]
Internalizing	−0.02	[−0.24; 0.20]
Disturbed relatedness	0.30	[−0.03; 0.63]

Note: ADHD = attention-deficit/hyperactivity disorder; CD = conduct disorder; IQ = intelligence quotient; ODD = oppositional defiant disorder. Values in boldface type are statistically significant based on a 95% CI.

[a]Ages 8 and 12 competence composite included 7 domains (peer relations, family relations, academics, physical health, mental health impairment, risk taking, and substance use).