Transdiagnostic pathways from early social stress to psychopathology: a 20-year prospective study

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Background: Adverse family environments confer susceptibility to virtually all psychiatric problems. This study evaluated two possible models to explain this diversity of associations. Stressful family circumstances during childhood could either activate general, transdiagnostic liabilities to mental disorder or promote numerous disorder-specific liabilities. Methods: We recruited a high-risk sample of 815 mother-offspring pairs and assessed social stressors in the family context prospectively from the perinatal period through offspring age 5. We factor analyzed offspring mental disorder diagnoses at age 20 to parse transdiagnostic and disorder-specific dimensions of psychopathology. Results: Structural analyses revealed nearly equivalent prospective effects of early family stress on overlapping Internalizing (β = .30) and Externalizing (β = .29) dimensions. In contrast, there was no evidence of disorder-specific effects. Conclusions: Social stressors early in life activate transdiagnostic, and not disorder-specific, liabilities to psychopathology. A focus on higher-order dimensions of psychopathology could accelerate etiological research and intervention efforts for stress-linked mental disorders. Keywords: Classification; comorbidity; early adversity; stress; transdiagnostic.

Introduction

One of the best-replicated findings in clinical psychology and psychiatry is that exposure to stressful family environments during childhood increases the risk of mental disorder. This association holds for virtually every psychiatric condition. Large-scale surveys show that early family stress (e.g. parental conflict and emotional abuse) has enduring associations with anxiety, depressive, substance use, antisocial behavior, and psychotic disorders (Green et al., 2010; Kessler et al., 2010). Developmental research over decades has illuminated a number of mechanisms thought to account for psychiatric disorders originating in stressful family and environmental contexts, shaping developing children’s insecure attachment styles, dysfunctional cognitive content and information processing, and behavioral and emotion regulation deficits (e.g. Dodge, Bates, & Pettit, 1990; Repetti, Taylor, & Seeman, 2002). Both animal and human research in developmental biology have helped to account for how stressful interactions and environmental conditions ‘get under the skin’ to promote neural, hormonal, genetic, and neuroendocrine processes that disrupt or impair responses to later challenges (e.g. Meaney, 2001; Miller & Chen, 2010; Sapolsky, 1996). However, these models generally yield relatively nonspecific findings, with few uniquely predictive associations between antecedent risks and particular forms of disorder (e.g. Shanahan, Copeland, Costello, & Angold, 2008).

The reasons for this nonspecificity are unclear, but at least two scenarios are possible (Nolen-Hoeksema & Watkins, 2011). First, stressors could act directly on numerous diagnostic categories (e.g. panic disorder and persistent depressive disorder). This model is consistent with the standard research design of examining life stress in relation to DSM-defined diagnoses. Second, early stressors could activate distal, transdiagnostic liabilities that in turn set the stage for various categorical diagnoses. This alternative is consistent with emerging etiological and nosological models that bypass DSM to frame research questions around dimensions of psychopathology that cut across traditional diagnostic entities (e.g. Cuthbert & Insel, 2013; Kotov et al., 2017).

The higher-order structure of psychopathology

Recent developments in structural models of psychopathology provide a useful framework for testing these competing hypotheses. Factor models of comorbidity among common mental disorders have revealed a small set of liability spectra that account for the co-occurrence of related conditions (reviewed by Lahey, Krueger, Rathouz, Waldman, & Zald, 2016). According to these models, depressive and anxiety disorders serve as indicators of a latent Internalizing dimension, whereas substance use and disruptive behavior disorders are indicators of a latent Externalizing dimension (Krueger, 1999). The two-factor structural model is hierarchical in that variability in each disorder is partitioned into (a) higher-order Internalizing and Externalizing dimensions that reflect common causes of related...
disorders, and (b) lower-order, specific dimensions that reflect pathology unique to a given disorder.

In recent years, researchers have searched for other dimensions of this trait hierarchy, at both superordinate and subordinate levels, relative to the Internalizing and Externalizing spectra. One line of evidence supports the existence of a general factor of psychopathology located at the apex of the hierarchical model (Lahey et al., 2012; Tackett et al., 2013). This overarching trait—termed the $p$-factor because it conceptually parallels the $g$-factor found in intelligence research—bridges the Internalizing and Externalizing dimensions and theoretically reflects vulnerability to any mental disorder (Caspi et al., 2014). At a more fine-grain level, the Internalizing dimension has been dissected in some datasets into more homogeneous Fear (panic and the phobias) and Distress (depression, posttraumatic stress disorder, and generalized anxiety disorder) subfactors (Krueger & Markon, 2006). Indeed, the structural model of psychopathology is a work in progress, and efforts are underway to expand the hierarchy ‘outwards’ by uncovering other higher-order dimensions that account for comorbidity alongside Internalizing and Externalizing, as well as ‘downwards’ by establishing more homogeneous syndromes and symptom components at lower levels of the trait structure (Kotov et al., 2017).

A number of investigations have examined specific early stressors, in particular child abuse and neglect, in association with higher-order dimensions of psychopathology. In a large national sample, retrospective reports of maltreatment were linked with these broad liabilities to mental disorders, but not disorder-specific pathology (Keyes et al., 2012). Subsequently, Vachon, Krueger, Rogosch, and Cicchetti (2015) demonstrated in a high-risk youth sample that childhood maltreatment predicted elevations on Internalizing and Externalizing dimensions. Again, after adjusting for the broad liability factors, specific diagnoses had no connection with early maltreatment. In studies that modeled the $p$-factor as an explanation for the overlap among Internalizing and Externalizing dimensions, childhood adversity was significantly associated with general risk for psychopathology, but not with the more specific Internalizing and Externalizing traits once their shared variance—captured by the $p$-factor—was partialled out (Caspi et al., 2014; Lahey et al., 2012).

Current study

We investigated the prospective effects of stressful family environments during early childhood on higher-order dimensions of psychopathology in a high-risk community sample followed from birth to age 20. We extended prior studies by contemporaneously assessing life stressors, all of which occurred before offspring age 5, to avoid bias attendant to retrospective reporting (Hardt & Rutter, 2004). Moreover, we moved beyond the evaluation of child maltreatment in prior research to examine a broad range of stressful family circumstances, some of which do not as immediately and directly impinge on the child but may nevertheless have pathological consequences. That is, we aimed to capture a more general trait of maladaptive family functioning. Past research has shown that indicators of family dysfunction (e.g., parental substance abuse, criminality, marital conflict, and instability) are particularly strongly linked with the onset of later psychopathology (Green et al., 2010).

On the basis of diagnostic interviews administered in adolescence and young adulthood, we evaluated a structural model of mental disorders anchored by Internalizing and Externalizing dimensions. We then related those liability spectra to exposure to our index of maladaptive family functioning, which included parental separation, parental conflict, family legal issues, maternal stress, and maternal depression. Previous research has implicated all of these stressors in diverse psychiatric conditions (Wolfe & Mash, 2006) and demonstrated that they regularly co-occur (Green et al., 2010).

We hypothesized that family stress during early childhood would prospectively predict standing on broad liability dimensions, but not disorder-specific factors. Further, based on prior findings, we expected similar associations of family stress with Internalizing and Externalizing dimensions, and we predicted that effect sizes would not vary across sex.

Methods

Participants

Our sample was recruited as a subsample of a birth cohort of over 7,000 mothers and their offspring who participated in the Mater-University Study of Pregnancy (MUSP) in Brisbane, Australia (Keeping et al., 1989). Mothers had been assessed for depression using the Delusions-Symptoms-States Inventory (DSSI; Bedford & Foulds, 1978) during pregnancy, postpartum, 6 months after birth, and 5 years after birth. This study selected 815 of the original families when the child reached age 15, oversampling for mothers endorsing elevated depression symptoms (see Keenan-Miller, Hammen, & Brennan, 2007, for sampling details). At offspring age 20, all families were re-contacted regarding participation in a second follow-up assessment, with 705 youth and mothers consenting to complete further interviews and questionnaires. The sample was largely Caucasian (92.1% White, 3.6 Asian, 1% Australian Aborigine, 0.8% Maori) and lower-middle class.

Procedure

In the initial phase of the MUSP, researchers invited women to participate in a study on pregnancy upon arrival for their first antenatal hospital visit (Keeping et al., 1989). Women completed self-report questionnaires on health problems, psychosocial constructs, daily activities, and attitudes toward pregnancy. Approximately 3–5 days after their child’s birth, mothers completed an additional questionnaire that assessed changes occurring during pregnancy. At child age 6 months,
and again at age 5 years, mothers were mailed further questionnaires assessing health and psychosocial changes for themselves and their children.

Mothers and offspring completed independent diagnostic interviews at age 15. At offspring age 20, offspring were again interviewed and completed a questionnaire battery in their homes. Procedures were approved by the Institutional Review Boards of the University of Queensland; University of California, Los Angeles; and Emory University. Participants provided written informed consent and were compensated for their time.

**Measures**

**Early childhood stress.** Mothers completed questionnaires related to various family adversities at multiple time points from pregnancy through offspring age 5. (a) At offspring age 5, mothers reported on whether they had experienced a divorce, separation, or partner change during the previous 5 years. (b) At all four time points before offspring age 5 (prenatal, birth, 6 months, 5 years), mothers completed the eight-item satisfaction scale of the Dyadic Adjustment Scale (DAS; Spanier, 1976). We averaged these four reports of relationship discord (alpha range .85 to .97) to create an index of parental conflict. (c) Also at offspring age 5, mothers reported whether they or their partners had been arrested or incarcerated over the previous 5 years. A positive response to either of these items for either partner was considered evidence of legal troubles. (d) Mothers completed a checklist of nine interpersonal, health, or occupational problems that occurred over the past 6 months at prenatal and postnatal assessments. (e) Mothers’ depressive disorder diagnoses between offspring birth and age 5 were ascertained using the lifetime version of the Structured Clinical Interview for DSM-IV (SCID; First, Spitzer, Gibbon, & Williams, 1995) administered to mothers when offspring reached age 15. The SCID diagnoses have demonstrated good reliability and validity in the present sample (see Keenan-Miller et al., 2007, for details). For continuous measures of adversity (maternal stress, relationship discord), responses were averaged, and offspring falling in the upper tertile of the resulting distribution were scored as experiencing that stressor, following prior work (Hazel, Hammen, Brennan, & Najman, 2008).

**Offspring mental disorders.** The Schedule for Affective Disorders and Schizophrenia in School-Aged Children (K-SADS-E; Orvaschel, 1995) was administered during the age 15 assessment wave to ascertain current and lifetime diagnoses of DSM-IV Axis I psychiatric disorders. The K-SADS-E is a widely used and validated semistructured interview for assigning Axis I diagnoses in children and adolescents. Trained clinicians interviewed adolescents and their mothers separately and privately. Diagnoses were assigned if either the adolescent or maternal interview indicated that the adolescent qualified for a given syndrome. Interrater reliability was assessed using a random sample of 75 K-SADS-E interview recordings evaluated by clinicians blind to the original diagnostic ratings. Weighted kappas were in the acceptable range (i.e. > .75) for depressive, anxiety, substance use, and disruptive behavior disorders. The SCID was administered to offspring at age 20 to assess for onsets of DSM-IV Axis I disorders since the age 15 wave. To determine interrater reliabilities for diagnoses, a sample of 55 interviews were selected and reviewed by a second trained clinician blind to the original diagnoses. Weighted kappas for depressive, anxiety, and substance use, and disruptive behavior disorders were all above 0.79. We aggregated information across the age 15 and 20 assessments to ascertain lifetime histories of disorder, which served as the indicators for our factor model of psychopathology.

**Data analytic procedures**

The first step of our analysis was to evaluate the fit of a two-factor structural model of psychopathology found to provide good fit to diagnostic correlations in prior research. In this model, the Internalizing factor was defined by major depressive disorder, dysthymia, generalized anxiety disorder, posttraumatic stress disorder, panic disorder, social phobia, and specific phobia, whereas the Externalizing factor was defined by conduct disorder, oppositional-defiant disorder, alcohol abuse/dependence, and drug abuse/dependence diagnoses. We compared the fit of this two-factor model to other structures supported in prior investigations. In one of these alternate models, the Internalizing factor bifurcates into Distress and Fear subfactors, where Fear is defined by panic disorder and the phobias and Distress by the remaining anxiety and depressive disorders. This lower-order organization of the Internalizing domain has been supported in some, but not all, datasets (e.g. Krueger & Markon, 2006). We also tested the fit of a bifactor model, in which all diagnoses load onto a general factor (termed the p-factor by Caspi et al., 2014) and either an Internalizing or Externalizing specific factor. All factors are constrained to be orthogonal. This hierarchical model has been tested in various studies of children and adults (see Lahey et al., 2016).

Second, we established a unidimensional factor model for the five family adversities. We focused on processes or events that reflected social dysfunction in the family context, such that each manifest variable theoretically is a function of levels of a social dysfunction trait characterizing the family unit. We labeled this latent variable – which underlies the parental separation, parental conflict, family legal issues, maternal stress, and maternal depression – Maladaptive Family Functioning.

We evaluated a common factor model based on evidence from prior studies that these adverse circumstances tend to co-occur at above-chance levels (e.g. Dong et al., 2004; McLaughlin, 2016). Indeed, a growing number of studies have reported the psychometric validity of addressing the covariation among childhood adversities with latent variable modeling (e.g. Green et al., 2010; Vachon et al., 2015). We specified a unifactorial model partly based on cumulative risk hypothesis, which supports a global view of environmental stressors, as opposed to examining them in isolation (e.g. Felitti et al., 1998). Also, a parallel analysis of the family adversity correlatedness matrix indicated that no more than 1 factor should be extracted.

Third, we regressed the higher-order dimensions from Step 1 onto Maladaptive Family Functioning. Fourth, we inspected model modification indices from Step 3 to determine whether estimating any paths from Maladaptive Family Functioning to disorder-specific dimensions (i.e. diagnosis residual terms) would improve model fit. Following prior conventions, we set a modification index threshold of 3.84 (i.e. critical chi-square value for expected model improvement corresponding to a .05 alpha level) to evaluate for the presence of statistically significant disorder-specific associations (Keyes et al., 2012).

We analyzed the data in Mplus (version 7.11; Muthén & Muthén, 1998–2014) using the weighted least squares means and variance adjusted (WLSMV) estimator. We evaluated model goodness of fit using comparative fit indices (CFI), the Tucker-Lewis index (TLI), the root mean square error of approximation (RMSEA), and the weighted root mean square residual (WRMR). Acceptable fit was defined according to guidelines offered by Hu and Bentler (1999): RMSEA values close to 0.06 or below, CFI and TLI values close to .95 or above, and WRMR values close to 1.00 or below. There were some missing data – 9% missing for maternal stress, 14% for maternal depression, 9% for legal trouble, and <1% for parental separation and conflict – for the various early adversities. Missingness was accommodated in all analyses using direct ML (see Allison, 2003).
Results

Common factor model of social stress

The correlations among the five early adversity risk factors are presented in Table 1. All stressors were significantly intercorrelated (median \( r = .20 \)). A one-factor model offered a good fit to the observed correlations, \( \chi^2(5) = 2.79, \ p = .73; \ CFI = 1.00; \ TLI = 1.01; \ RMSEA = .00; \ WRMR = .36. \) Modification indices suggested that there were no localized areas of strain (i.e. outstanding residual correlations).

Higher-order structure of psychopathology

A correlation matrix of all diagnoses is reported in online supplementary material (Table S1). We initially fit an oblique two-factor model to the diagnostic correlations, as described above. That model provided a good fit to the data, \( \chi^2(29) = 37.18, \ p = .14; \ CFI = .98; \ RMSEA = .02; \ WRMR = .89. \) All factor loadings were moderate-to-large and statistically significant at the .001 alpha level. The two latent factors were moderately correlated (\( r = .37 \)).

Regarding alternate dimensional structures, the higher-order model involving Fear and Distress sub-factors fit adequately, \( \chi^2(41) = 63.90, \ p < .05; \ CFI = .96; \ RMSEA = .03; \ WRMR = .93, \) but ultimately was not appropriate for our data due to a negative residual variance estimate for the Fear sub-factor. An exploratory three-factor model revealed that the factor correlation between Distress and Fear was not different from unity. Additionally, we rejected the bifactor solution because the model-implied variance for the Externalizing factor was nil. Fit statistics were not available because the model did not converge properly. In an ad hoc analysis, we specified that the externalizing variance should be >0 using the model constraint command in Mplus, but the variance estimate remained small and not statistically significant, and there were out-of-range factor loading estimates on the Externalizing factor (full results available upon request).\(^1\) Therefore, we carried forward the two-factor (correlated Internalizing and Externalizing) structural model for tests of the association of early family adversity with higher-order liabilities.

General and disorder-specific pathways from social stress to psychopathology

We next regressed Internalizing and Externalizing factors on the Maladaptive Family Functioning factor (see Figure 1). We adjusted for sex in the structural model, and it was associated with the liability factors in expected ways. That is, males were characterized by greater Externalizing (\( b = .46, \ SE = .08, \ p < .001, \ \beta = .63 \)) and smaller Internalizing (\( b = -.46, \ SE = .08, \ p < .001, \ \beta = -.60 \)) levels. Maladaptive Family Functioning had statistically significant effects (\( p < .001 \)) on the higher-order dimensions, such that a standard unit increment in Maladaptive Family Functioning predicted an increase of 0.30 standard deviation in trait Internalizing and .29 standard deviation in trait Externalizing. Chi-square difference tests confirmed that the effects of social stress on Internalizing versus Externalizing were not significantly different, and that no regression coefficient varied significantly by sex, \( \chi^2(\text{diffs}(1)) < 1.00, \ ps > .10. \) Collectively, these predictors explained 18% of variability in both the Internalizing and Externalizing spectra.\(^2\)

Finally, we examined the effects of Maladaptive Family Functioning on disorder-specific dimensions. We inspected the modification indices to determine whether Maladaptive Family Functioning had a statistically significant association with residual variation in any disorder. No regression modification index exceeded our predetermined threshold for significance. Therefore, the association between Maladaptive Family Functioning and psychopathology was accounted for by the higher-order Internalizing and Externalizing liabilities.

Discussion

Stressful family environments during early childhood portend a wide array of psychiatric disorders. Most research has examined diagnostic outcomes individually, or in small clusters, in relation to early adversities, so explanations for this multifinality remain elusive (Kessler et al., 2010). We took advantage of a hierarchical structural model of psychopathology to test two competing accounts. We found that maladaptive family functioning during childhood (e.g. parental discord and legal trouble) conferred risk to two broad liability spectra, as opposed to numerous discrete diagnostic entities. Thus, family adversity early in life appears to be associated with higher-order dimensions that cut across traditional diagnostic boundaries.

Our finding is in strong agreement with prior investigations that examined childhood maltreatment (i.e. abuse and neglect) in relation to transdiagnostic spectra (Caspí et al., 2014; Keyes et al., 2012;
Vachon et al., 2015). For instance, in one study of high-risk youth, documented cases of child maltreatment predicted latent Internalizing and Externalizing liabilities, but not disorder-specific pathology (Vachon et al., 2015). Indeed, those investigators reported effect sizes (i.e., standardized effects of .19 and .28 on Internalizing and Externalizing, respectively) comparable to, if not slightly smaller than, ones observed here.

Our study advances existing work in this area through a more inclusive approach to social adversities during childhood. Instead of concentrating on severe cases of child maltreatment, we assessed a broader range of social stressors that reflected family dysfunction more comprehensively. Thus, we captured more common and diverse family circumstances, which past research has shown to be particularly closely linked with disorder risk (e.g., Green et al., 2010). Indeed, those investigators reported effect sizes (i.e., standardized effects of .19 and .28 on Internalizing and Externalizing, respectively) comparable to, if not slightly smaller than, ones observed here.

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The causal mechanisms that account for the association between early family adversity and Internalizing and Externalizing pathology were not evaluated in our study and remain uncertain. One theoretical formulation of this causal pathway holds that deficits in emotion processing and executive function mediate the pathogenic effects of stressful childhood environments (McLaughlin, 2016). This model posits that exposure to threatening events instigates attention, attribution, and memory bias; limbic system hyperresponsivity to negative emotional cues; and exaggerated autonomic nervous system arousal (e.g., Dodge et al., 1990; Gee et al., 2013; Pollak, Cicchetti, Hornung, & Reed, 2000). In turn, these disrupted emotional systems set the stage for the full spectrum of mental disorders, accounting for the multifinal downstream effects of early threatening and uncontrollable experiences (Aldao, Nolen-Hoeksema, & Schweizer, 2010). At the same time, a related line of research indicates that deprivation experiences during childhood, such as institutionalization or poverty, predict mental disorders via executive functioning impairments. That is, deprived environments (e.g., lack of attachment figures and no age-appropriate cognitively enriching activities) are linked with diminished planning, inhibition, and working memory (e.g., Farah et al., 2006; Noble, McCandliss, & Farah, 2007). These cognitive control deficits are in turn linked with risk for psychopathology, especially externalizing conditions (McLaughlin, Sheridan, & Lambert, 2014). With continued research into these intermediate phenotypes that mediate the pathogenic influences of early adversity, investigators can establish modifiable psychological and biological influences.
processes that could be targeted in early intervention efforts to deflect the developmental course of childhood psychopathology.

Several additional study limitations should be acknowledged. First, our coverage of psychopathology was limited. Additional research involving a wider range of disorders (e.g. eating disorders and bipolar disorders) is needed to explore the architecture of psychopathology (Kotov et al., 2017). Second, higher-order and bifactor models did not fit acceptably to our diagnostic correlations. This could be related to our high-risk sampling procedure; the bifactor model, for instance, has fit adequately in several epidemiological datasets (see Lahey et al., 2016). Indeed, the residual correlation between Internalizing and Externalizing factors in our structural model was .50, suggesting appreciable shared variation across these dimensions even after adjusting for early adversity. We advise that future research should evaluate these various factor structures simultaneously to compare the associations of early social stress with subfactors (e.g. Fear), spectra (e.g. Internalizing), and super-spectra (e.g. p-factor) from hierarchical structural models of psychopathology (Kotov et al., 2017). Third, like virtually all social science research, we were unable to disentangle possible gene–environment correlation effects from a true, causal association between maladaptive family functioning and offspring mental disorder. It is possible that parents’ genetic risk for mental disorder, which is passed down to the offspring, is correlated with propensity to foster dysfunctional environments, creating a spurious association between family environment and offspring risk for psychopathology (see Scarr & McCartney, 1983). We advise cautious interpretation of our results until replications in genetically informative samples (e.g. twins) are available. Finally, maladaptive family functioning data were available from only one source (i.e. parent). Incorporating multiple informants and multiple measurement domains (e.g. legal records, children, and teachers) in future research will help avoid bias in the assessment of adversity.

Conclusion
We found that adverse family environments early in child development prospectively predict higher-order liability dimensions for psychopathology, but not specific disorder entities. Thus, effects of family dysfunction (and possibly other social stressors) may be multifinal because they operate through broad, transdiagnostic pathways. Targeting tumultuous family environments in early childhood might, therefore, prevent susceptibility to diverse mental disorders.

Supporting information
Additional Supporting Information may be found in the online version of this article:
Table S1. Tetrachoric Correlations among Offspring Lifetime Mental Disorder Diagnoses.

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Key points
- Social stressors in early childhood portend a wide array of psychiatric problems later in development.
- In a high-risk community sample, we found that such multifinality is attributable to childhood stressors activating transdiagnostic, as opposed to disorder-specific, liabilities to psychopathology.
- Instead of continuing to investigate early stressors in relation to single disorders – or small clusters of related disorders – researchers would benefit from examining transdiagnostic outcomes that cut across traditional disorder boundaries.
- Likewise, assessment and early intervention for psychiatric disorders might be streamlined by targeting transdiagnostic factors, such as Internalizing and Externalizing dimensions.

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Notes

1. To explore this model misfit further, we estimated the bifactor model while omitting either the Externalizing or Internalizing specific factors. The model that removed Externalizing fit acceptably, \( \chi^2(37) = 58.58, p < .05; \) CFI = 0.96; TLI = 0.94; RMSEA = .03; WRMR = 0.86, BIC = 5,447.58, as did the model without Internalizing, \( \chi^2(40) = 59.82, p < .05; \) CFI = 0.97; TLI = 0.95; RMSEA = .03; WRMR = 0.86, BIC = 5,429.90. However, neither was superior to the hypothesized oblique two-factor model, which outperformed the bifactor models on BIC and was roughly equivalent according to other fit indices, \( \chi^2(43) = 64.43, p < .05; \) CFI = 0.96; TLI = 0.95; RMSEA = .03; WRMR = 1.01. Family dysfunction had positive, moderate effects on both Internalizing (\( b = 0.13, SE = 0.03, p < .001, \beta = 0.20 \)) and Externalizing (\( b = 0.11, SE = 0.03, p < .001, \beta = 0.18 \)) traits while adjusting for sex. Thus, the same pattern of effects was observed as in the primary analysis, although effect sizes were reduced roughly 33%, probably attenuated due to measurement error. No diagnosis-specific effects of family dysfunction on internalizing or externalizing diagnoses were detected in this analysis.

References


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