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Developmental cascade models linking contextual risks, parenting, and internalizing symptoms: A 17-year longitudinal study from early childhood to emerging adulthood

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Abstract

Although internalizing problems are the most common forms of psychological distress among adolescents and young adults, they have precursors in multiple risk domains established during childhood. This study examined cascading risk pathways leading to depression and anxiety symptoms in emerging adulthood by integrating broad contextual (i.e., multiple contextual risks), parental (i.e., negative parenting), and child (i.e., internalizing behaviors) characteristics in early and middle childhood. We also compared common and differential pathways to depression and anxiety symptoms depending on the conceptualization of symptom outcomes (traditional symptom dimension vs. bifactor dimensional model). Participants were 235 children (109 girls) and their families. Data were collected at 3, 6, 10, and 19 years of child age, using multiple informants and contexts. Results from a symptom dimension approach indicated mediation pathways from early childhood risk factors to depression and anxiety symptoms in emerging adulthood, suggesting common and distinct risk processes between the two disorders. Results from a bifactor modeling approach indicated several indirect pathways leading to a general internalizing latent factor, but not to symptom-specific (i.e., depression, anxiety) latent factors. Our findings highlighted comparative analytic approaches to examining transactional processes associated with later internalizing symptoms and shed light on issues of early identification and prevention.

Keywords: bifactor model; contextual risk; developmental cascades; internalizing symptoms; parenting

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Internalizing problems, characterized by a variety of over-inhibited or internally focused symptoms including depression and anxiety, are the most common forms of psychological distress that adolescents and young adults experience (Petersen et al., 2018). Moreover, depression and anxiety symptoms can lead to other impairments, including poor academic achievement, troubled interpersonal relationships, and further development of mental health issues even among individuals who are below diagnostic thresholds (Hunt & Eisenberg, 2010; Stone et al., 2016). Therefore, understanding developmental pathways in early childhood that are associated with emerging adult internalizing problems is an important research issue that has strong implications for prevention.

While symptoms of depression and anxiety are highly correlated and co-occurring, prior research has indicated that the two disorders are distinct constructs. For example, several models, such as tripartite model (Clark & Watson, 1991) and models emphasizing the hierarchical structure of internalizing psychopathology (Brodbeck et al., 2011; Simms et al.,confirmatory factor analysis 2008) posited components that account for the unique symptoms of depression and anxiety in addition to shared components of the two disorders. Specifically, those studies found negative affectivity or general distress as shared components of depression and anxiety, whereas low positive affectivity and hopelessness were more specific to depression and physiological hyperarousal was more specific to anxiety. In addition, there is evidence for both common and different antecedents and developmental risk processes associated with depression and anxiety (Hopkins et al., 2013; Karevold et al., 2009). For example, Hopkins et al. (2013) found parental depression symptoms and parenting behaviors as common risks for both depression and anxiety, whereas children's effortful control was more specifically related to anxiety symptoms. Given that depression and anxiety are related but distinct constructs, studying both disorders in the same longitudinal sample could help distinguish general versus specific developmental precursors of depression and anxiety. Therefore, our main aim was to examine common and differential childhood risk pathways associated with depression and anxiety symptoms in emerging adulthood, highlighting multiple domains of risk and developmental cascading effects from early childhood.

Our study was grounded in the developmental psychopathology perspective, which highlights developmental pathways as well as interacting risk and protective factors when explaining the development and maintenance of psychopathology (Rutter & Sroufe, 2000). This framework provides several benefits for understanding developmental precursors of emerging adults' internalizing problems. First, even though depression and anxiety symptoms have a rapid spike in adolescence, risk factors in

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multiple domains already have been established during childhood and adolescence (Achenbach et al., 1991; Hopkins et al., 2013). For example, prior research has emphasized the role of child vulnerabilities and environmental factors such as parenting as early precursors of later internalizing problems (Ashford et al., 2008; Côté et al., 2009). Consistent with the concept of developmental cascades (Cox et al., 2010), these risk factors may set the stage for subsequent development of depression and anxiety symptoms. Additionally, long-term longitudinal studies may provide information about how those risk factors contribute to child adjustment outcomes across different developmental periods and help identify the optimal timing of interventions. Our study was also guided by ecological–transactional models of development. This approach conceptualizes the developmental process of psychopathology as a dynamic interplay between broader contextual features of the environment (e.g., sociodemographic status, negative life events, parent psychopathology, etc.), the caregiving system, and individual child vulnerabilities that all influence one another and make reciprocal contributions to child developmental outcomes (Cicchetti & Lynch, 1993; Sameroff, 2009). Considering multiple risk and protective factors that occur at different levels provides a clearer picture of the complex nature of symptom development. Combining developmental cascade and transactional perspectives would also help explicate developmental processes underlying internalizing problems by encompassing distal and proximal risk factors across multiple developmental periods.

Despite those advantages, few studies have integrated information about broad contextual features of the environment, quality of the caregiving system, and individual child vulnerabilities across lengthy periods of development when examining early risk pathways to internalizing problems. Moreover, while symptoms of anxiety and depression are highly co-occurring (Hankin et al., 2016), there has been a dearth of research distinguishing between depression and anxiety in explanatory models, which may hinder researchers from understanding whether antecedent risk processes are common for both depression and anxiety, or vary between each disorder (Hopkins et al., 2013). Therefore, using longitudinal data spanning ages 3–19 years, we examined common and differential pathways leading to depression and anxiety symptoms in emerging adulthood. Specifically, we focused on three constructs that have been established as key risk factors for later depression and anxiety symptoms: multiple contextual risks that families experience in their daily lives, suboptimal parenting quality, and children's prior internalizing symptoms.

Multiple contextual risks as distal antecedents

Several models of psychopathology, such as the bioecological model (Bronfenbrenner & Morris, 2007) the family stress model (Conger et al., 2002), and the transactional model (Cicchetti & Lynch, 1993; Sameroff, 1975) posit that multiple risk and protective factors occurring at different levels affect the development of a particular disorder. From this multilevel developmental systems perspective, contextual risks that families experience in their daily lives may serve as distal risk factors that directly or indirectly (e.g., through proximal factors such as parenting) contribute to adjustment outcomes. Common contextual risks include sociodemographic variables such as low parental education, managing a single parent household, and teenage parenthood as well as psychosocial factors such as negative life events, family conflict, and parental psychopathology (see Evans et al., 2013).

Rather than highlighting a single risk factor, we focused on exposure to multiple contextual risks as a distal factor potentially related to the development of later depression and anxiety symptoms. First, exposure to multiple risks might be a stronger predictor of child adjustment than exposure to single risks, considering that development occurs in multiple contexts and children often face constellations of risk rather than single, isolated adverse experiences (Evans et al., 2013; Gach et al., 2018). Second, although a more moderate level of one specific risk by itself may have negligible influence on later adjustment compared to the exposure to severe deprivation, it may matter a lot when accompanied by other risk factors or when it occurs within the context of other environmental risks (Evans et al., 2013). We focused on children's exposure to multiple contextual risks during the early childhood period, as prior research indicates that cumulative risk during early childhood is a stronger and more consistent predictor of long-term behavior problems than cumulative risks during middle childhood (Appleyard et al., 2005; Gutman et al., 2019).

Longitudinal studies have shown that children's exposure to early cumulative risk is associated with increased internalizing and externalizing problems in later years (Gach et al., 2018; Trentacosta et al., 2008). However, we still have limited understanding of whether and how cumulative risk during early childhood directly or indirectly contributes to longer-term internalizing problems spanning into adolescence and emerging adulthood. As a notable exception, one recent study showed that extreme institutional deprivation during early childhood was predictive of adult depression and anxiety symptoms (Golm et al., 2020). However, to our knowledge no study has examined associations between early childhood adversities and emerging adult depression and anxiety among lower-risk populations. Given that internalizing problems are one of the most prevalent forms of psychological distress in community-based adolescent and young adult populations (Kessler et al., 2005), studying such associations in lower-risk samples would provide valuable information on how accumulation of single, and seemingly less stressful, risk factors may predict later emotional disturbances with other environmental risks and through cascading pathways.

Negative parenting as a proximal risk factor in ecological systems

Parenting behaviors have long been recognized as critical predictors of children's internalizing problems. Negative parenting behaviors such as lack of warmth and frequent use of physical punishment have been associated with higher levels of anxiety and depression among children and adolescents (Côté et al., 2009; Yap et al., 2014). In addition to parenting behaviors, parents' attitudes, cognitions, and self-efficacy about parenting have been found to predict children's negative adjustment outcomes in later years (Callender et al., 2012; Hopkins et al., 2013; Sanders & Woolley, 2005).

Prior literature has suggested that parenting may also serve as an important proximal factor to the child, that mediates the relationship between early contextual risks and later internalizing problems (Kim & Brody, 2005; Trentacosta et al., 2008). In other words, the factors more distal to the child (e.g., contextual risks) may be associated with child outcomes through their effects on more proximal factors such as parenting in early childhood (Hopkins et al., 2013). For example, Trentacosta et al. (2008) found that nurturant and involved parenting mediated the longitudinal relationship between cumulative environmental risk and later internalizing problems. For several reasons, we focused on negative dimensions of parenting at age 6 as a proximal factor linking early

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multiple risk exposure and later internalizing problems. First, children and their caregivers have frequent interactions during the early school age period and therefore associations between environmental risk and child adjustment are largely accounted for by parents' attitudes and behaviors (Morris et al., 2007). Moreover, age 6 is a critical time for adjustment as children enter primary school, which involves more structured environments and a widening range of social interactions (Blair, 2010). Therefore, parenting quality during this developmental period may be especially important for children's ability to successfully adjust to these developmental challenges by teaching important socialization and regulatory skills and providing emotional support.

While associations between parenting and internalizing problems have been well established, it is still unclear whether and to what extent parenting quality during this period has long-lasting associations with children's internalizing symptoms. For example, parent-child interactions during the early school period may play an immediate role in child internalizing symptoms which become exacerbated during adolescence and emerging adulthood. In addition, parenting during this period may make unique direct contributions to later depression and anxiety independent of concurrent internalizing problems. Explicating long-term direct and indirect associations between parenting and child internalizing symptoms could inform the timing of interventions designed to prevent depression and anxiety symptoms in early adulthood.

Earlier internalizing symptoms as markers of child vulnerability

Although depression and anxiety symptoms are more common during adolescence and adulthood, there is substantial evidence that the individual differences in internalizing symptoms start to develop in childhood and are relatively stable across development. For example, Ashford et al. (2008) found that parents' reports of child internalizing problems during the preschool years were the strongest predictors of internalizing problems at age 11 compared with other predictors such as low SES or family psychopathology. In addition, longitudinal studies have shown moderate to high continuity for internalizing problems from as early as toddlerhood through the school-age years, into adolescence, and adulthood (Keenan et al., 1998; McElroy et al., 2018; Newman et al., 1996). Similarly, person-centered studies have revealed a subgroup of children showing high and stable internalizing problems across developmental periods, suggesting continuities in internalizing symptoms (Ellis et al., 2017; Fanti & Henrich, 2010; Sterba et al., 2007). Prior studies also have shown significant cross-informant agreement on children's internalizing behaviors within and across time and contexts (Achenbach & Rescorla, 2016; Kerr et al., 2007). These findings indicate the rank-order stability of internalizing constructs throughout development taking into account bias due to common informants and methods, although it is still important to include multiple informants in behavioral ratings for valid and comprehensive understanding of child's behavior (De Los Reyes & Kazdin, 2005)

However, it is still unclear why and how stability in children's internalizing symptoms occurs over time. There are several potential explanations involving various risk mechanisms. For example, stability in internalizing symptoms may reflect children's vulnerability to internalizing psychopathology through genetic influences and/or temperament (e.g., negative affectivity) (Ellis et al., 2017; Karevold et al., 2009; Shore et al., 2018). On the other hand, these symptoms may be provoked by environmental risks such as suboptimal parenting or contextual adversity during early childhood, the formative period for cognitive and emotional development, then cascade across development (Côté et al., 2009; Karevold et al., 2009). Moreover, prior studies have revealed child-driven effects on the environment, such that children's behavioral problems themselves may elicit negative parenting, potentiating increased symptoms of internalizing problems (Gouze et al., 2017; Shewark et al., 2021; Yan & Ansari, 2016). Therefore, additional studies are warranted to explore possible mechanisms underlying the rank-order stability of internalizing problems by including repeated measures of internalizing problems at different developmental periods in conjunction with other contextual risk factors.

An alternative approach: bifactor model

One of the most common approaches to understanding internalizing psychopathology has involved using current DSM-defined symptom dimensions as outcome variables (e.g., depression symptoms, anxiety symptoms). However, more recent research has focused on latent dimensional models of psychopathology (Carragher et al., 2015; Hankin et al., 2016; Krueger & Eaton, 2015). One well-known approach is bifactor modeling, which allows symptoms to load on both a general factor and one specific factor (Caspi et al., 2014). This allows us to separate symptom-specific variances (e.g., depression and anxiety) from the variance of the general factor (internalizing). Therefore, bifactor modeling is optimal for studying shared and unique features of psychopathology and clarifying the etiology of highly comorbid disorders (Hankin et al., 2016). Prior literature from the bifactor model approach has evidenced general psychopathology (p-factor) and specific internalizing and externalizing factors (Caspi et al., 2014; Snyder et al., 2017), as well as general and specific components of depression and anxiety symptoms (Brodbeck et al., 2011; Simms et al., 2008).

Despite noteworthy studies on latent dimensional models of psychopathology using bifactor modeling, we have little understanding on how particular risk factors and mechanisms are linked to more general versus specific internalizing psychopathology (see Deutz et al., 2020; Hankin et al., 2017 for notable exceptions). Moreover, to our knowledge no study has examined pathways leading to depression and anxiety symptoms from both traditional symptom dimension and bifactor model approaches using the same sample. Comparing those two approaches would provide a more comprehensive picture of depression and anxiety symptomatology as both methods have unique strengths. For example, models using the traditional symptom level approach could help clarify developmental pathways leading to the actual manifestation of depression and anxiety symptoms. On the other hand, bifactor modeling could provide an opportunity to partition the common versus unique variances of internalizing symptoms, and therefore, help us better understand potentially distinctive pathways that are associated with the general internalizing factor versus narrow-band symptom clusters.

The current study

As reviewed, multiple contextual risks, adverse parenting, and earlier child internalizing symptoms have been established as key risk factors for later depression and anxiety symptoms. However, there have been relatively few studies exploring how childhood risk factors in multiple domains contribute to increased risk for depression and anxiety over a lengthy span of development,

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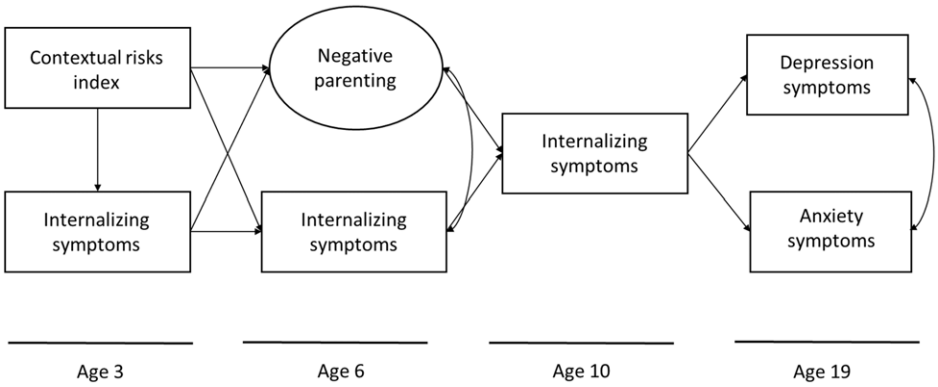

Figure 1. The conceptual model of developmental risk pathways leading to emerging adult depression and anxiety symptoms.

especially through emerging adulthood. In addition, we still have limited knowledge of common and differential developmental pathways to depression and anxiety, partly due to the lack of studies considering both symptoms simultaneously in the same model. Studying depression and anxiety in the same model may increase our understanding of risk pathways that either contribute to the development of both depression and anxiety symptoms or to specific subdimensions. Furthermore, this knowledge may help identify risk factors and specific developmental periods that need to be addressed for early prevention of later depression and anxiety.

Thus, the purpose of our study was to examine developmental pathways between ages 3 and 19 that are associated with depression and anxiety symptoms in emerging adulthood. Based on transactional and cascade models from the developmental psychopathology framework, we considered multiple domains of risk factors (distal contextual risks, familial characteristics, and child vulnerabilities) across multiple time points. When considering internalizing symptoms from multiple time points across a lengthy span of development, we used broader measures of internalizing symptoms through early and middle childhood and included depression and anxiety symptoms separately in emerging adulthood (conceptual model is shown in Figure 1). This is guided by prior literature suggesting that depression and anxiety emerge from a single construct of internalizing symptoms in early childhood and may become differentiated as children age after mid-adolescence (Patrick et al., 2010; Price et al., 2013), although there are mixed findings regarding when the differentiation starts (e.g., McElroy et al., 2018). Knowledge of developmental risk pathways from early childhood through emerging adulthood may potentially shed light on the early identification and prevention of internalizing problems by disrupting cascading pathways associated with later depression and anxiety.

In addition, following the suggestion of an external reviewer, in a secondary analysis we specified the conceptual model from the bifactor approach. Specifically, multiple domains of risk factors were linked to an age 19 general internalizing latent factor and symptom-specific latent factors, instead of using the DSM-oriented depression and anxiety symptoms. By including a bifactor model, we would like to compare how the indirect pathways leading to depression and anxiety symptoms would look similar/different depending on the conceptualization of symptom outcomes (i.e., traditional symptom levels vs. general and specific factors of internalizing symptoms).

Specific hypotheses were as follows:

1. Higher levels of multiple contextual risk at age 3 will be linked to increased depression and anxiety symptoms at age 19 via negative parenting at age 6 and higher levels of internalizing symptoms at ages 6 and 10.
2. Internalizing symptoms will show rank-order stability across development, such that higher levels of internalizing symptoms at age 3 will be linked to increased depression and anxiety symptoms at age 19 via higher levels of internalizing symptoms at ages 6 and 10 years. In addition, we tested whether long-term stability in internalizing symptoms was mediated by negative parenting at age 6.
3. In exploratory analyses, we examined whether there are common or unique developmental pathways to anxiety and depression symptoms at age 19. We tested the same developmental pathways from both traditional symptom dimension and bifactor model approaches.

Method

Participants

Participants were part of a larger longitudinal project on the development of children's behavioral problems. Children were 3 years ($M = 3.44$, $SD = 0.16$), 6 years ($M = 5.77$, $SD = 0.31$), 10 years ($M = 10.42$, $SD = 0.63$), and 19 years ($M = 19.09$, $SD = 1.09$) of age at four waves of data collection. Of 245 children who participated initially, 230 participants (109 females) whose parents reported internalizing symptoms and multiple contextual risks at age 3 were included in our study. Most families (95%) were recruited using local and regional newspaper ads, fliers at daycare centers and preschools, with others referred by teachers and pediatricians. Due to the initial design of the study, children were recruited to represent the full range of Externalizing and Internalizing Problems on the CBCL (CBCL/2-3; Achenbach, 1992), with overrepresentation of moderate to high levels of externalizing problems ($T > 60 = 44.5\%$) (see Olson et al., 2005 for more details).

Children's ethnic backgrounds were primarily non-Hispanic Euro-American (86%), followed by Biracial (8.7%) African American (3.4%), Hispanic American (0.4%), and Asian American (0.4%). The majority of children were from two-parent households (i.e., married 89.6%; living with a partner, 3.5%), with smaller numbers of other family constellations (i.e., single, 4.3%;

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separated or divorced, 2.6%). Annual family income at recruitment ranged from $10,000 to over $100,000 per year with a median between $60,000 and $70,000. Of the initial sample of 230 families at age 3, 221 (96%), 203 (88%), and 147 (64%) participated at 6, 10, and 19 years, respectively.

Missing data and attrition

Emerging adults who provided data at W4 were more likely to be raised in two-parent households ($M = .06$, $SD = .23$) compared to those who dropped out ($M = .26$, $SD = .44$) ($t(145.04) = 4.28$. $p < .005$). Also, families of emerging adults who participated in W4 data collection had higher incomes at W1 compared to those who did not ($M = 9.71$, $SD = 2.83$, vs. $M = 8.81$, $SD = 3.22$, t $(189.19) = -2.21$, $p = .03$). No other missing data or attrition-related differences were found. Little's missing completely at random (MCAR) test (Little, 1988) was not significant, which suggests that the pattern of missingness does not significantly deviate from a missing completely at random pattern (χ^2 $(256) = 248.51$, $p = .62$). To handle the missing data, a FIML approach was used for the sample with W1 contextual risks and internalizing symptoms ratings ($N = 230$) (Schafer & Graham, 2002).

Measures

Multiple contextual risks (at age 3)

A multiple contextual risk index was created at age 3 years, which represented risk factors that families may experience across multiple domains. Nine risk factors were included: teenage parent, low education, single parent, transition of family structure during last 12 months, economic burden during last 12 months, maternal depression, negative life events, marital aggression, and maternal perceived social isolation. Dichotomous risk factors (i.e., teenage parent, low education, single parent, transition to family structure, economic burden, maternal depression) were scored as 0 = not present, 1 = present. In order to avoid artificial dichotomization of continuous variables (e.g., Evans et al., 2013), continuous risk factor scores (negative life events, marital aggression, maternal perceived social isolation) were converted into proportion scores. Specifically, following the approach of Lengua et al. (2020), each score was divided by the total possible score so that each risk ranged from 0 to 1, and thus, were on a similar scale as the dichotomous variables. The total multiple family risk score was the sum of all component factors.

Per mothers' reports at the time of the study, 3.4% were teenage parents (<20 years) when the child was born. Low education was indicated by mothers' not graduating from high school (0.4% of participants at age 3 years). Single parent status was defined by being never married, currently widowed, separated, or divorced. 15% of families was classified as having single parent structure. In addition to single parent status, family structure transition during the last 12 months of data collection was included as additional risk factors. At the time of data collection, 7% mothers indicated that they were recently divorced, reconciled or separated during last 12 months. Risk of economic burden was indicated by mothers reporting either that the family was deeply in debt or when income decreased substantially (i.e., more than 20%) during the last 12 months of data collection (17% of participants). Mothers also reported their depressive symptoms on the BSI (Derogatis & Melisaratos, 1983) depression subscale. Mothers who received T scores of 63 or higher were classified as having clinically significant depressive symptoms (6% of mothers).

Negative life events were calculated using maternal reports on an adapted version of the LEQ (Coddington, 1972; Garmezy et al., 1984). Of 22 events, 9 items were identified as negative events and included in the study. Examples of events included death of immediate family members or close friends, legal problems, member of family lost job, and alcohol or drug problems. Mothers reported whether the events occurred in the last 12 months. We used the number of events occurred as total score of negative life events ($M = .54$, $SD = .85$, range 0–4). The total score was then converted into a proportion of the possible 9 events. Internal reliability was low ($\alpha = .34$) in this study, suggesting that these life events are relatively independent. This is consistent with prior studies of discrete stressful life events, as the experience of one negative life event is not expected to increase the likelihood of another event (Hoffmann & Su, 1998; Mitchell & Ronzio, 2011).

Mothers reported marital aggression over the last 12 months of the W1 data collection using the Conflicts and Problem-Solving Scale (Kerig, 1996), a reliable and valid measure of marital adjustment. Mothers indicated the frequency with which their partner and themselves experienced severe (e.g., strike, kick, bite partner) and moderate (e.g., throw objects, slam doors, break things) levels of physical aggression, as well as verbal (e.g., yelling, accusing, insulting) and emotional aggression (e.g., name calling, cursing, insulting). Mothers indicated whether each item was present on a scale of 0 (never) to 3 (often). The responses were combined into a total marital aggression index, which was then converted into a proportion score ($\alpha = .95$ in this study).

Parents also completed the Abidin (1983) PSI. The Social isolation Subscale was used to measure parents' perceived social isolation. It consists of six items about one's perceived levels of social connectedness and support, ranging from 1 (strongly agree) to 5 (strongly disagree). Sample items includes "When I run into a problem taking care of my children, I have a lot of people to whom I can talk to get help or advice". Higher scores on the subscale indicate higher levels of perceived social isolation. The total score from the subscale was converted into a proportion score ($\alpha = .76$ in this study).

The contextual risk index score does not provide a clear indication of the number of risks in the sample since we made proportion scores for continuous variables. However, for descriptive purposes, the mean and the median of the multiple contextual risk score was 1.07 ($SD = .92$) and .63, respectively, with minimum score of .20 and maximum of 5.72.

Negative parenting (at Age 6)

Negative appraisals of child behavior. Mothers rated 11 items measuring parents' perceptions of their child's level of responsiveness and reciprocal affection towards them. The items were rated on 5-point scales, ranging from strongly agree (1) to strongly disagree (5). Perceptions of child unresponsiveness were measured using six items from the Unresponsiveness Subscale in the Maternal Perceptions Questionnaire (MPQ; Olson et al., 1982). The questionnaire has been shown to have high internal consistency and predictive validity for long-term child adjustment outcomes (Olson et al., 1982, 2000). Sample items included: "My child seems to prefer spending time by himself/herself rather than with me," and "I wish my child were more affectionate to me." Perceptions of children's lack of affection were measured using five items from the Child Reinforces Parent Subscale in the PSI (Abidin, 1983). The questionnaire has high test–retest reliability and has been effective in differentiating abusive and non-abusive parents (Éthier et al., 1995; Abidin, 1983). Sample items included:

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"Sometimes I feel my child doesn't like me and doesn't want to be close to me," and "When I do things for my child, I get the feeling that my efforts are not appreciated very much." ($\alpha = .67$ in this study)

Self-perceived caregiving inefficacy. To measure parent's self-reported caregiving stress when children were 6 years of age, we used the Sense of Competence subscale of the PSI (Abidin, 1983) that has been found to have good test–retest reliability, convergent validity, and criterion-related validity (Feindler et al., 2003). Parents rated items on 5-point Likert scales, ranging from strongly agree (1) to strongly disagree (5). Sample items included "Child likes to play with me", "As a parent, I feel that I am very good" ($\alpha = .53$ in this study).

Unresponsiveness. Unresponsive parenting was rated by mothers using the PDI (Power, 1993). On 6-point Likert scales, mothers rated 11 items from the Nurturance and Responsiveness subscales, which were averaged and standardized to form a total score measuring Unresponsiveness. The 6-point scale ranged from "not at all descriptive of me" (1) to "highly descriptive of me" (6). This subscale was theoretically related to maternal warmth (Eisenberg et al., 2005). Items were reverse-coded to represent negative dimension of parenting. Sample items included "My child and I have warm intimate moments together" (nurturance); and "I encourage my child to express his/her opinion" (responsiveness) ($\alpha = .76$ in this study).

Physical punishment. During a home interview at W2, mothers answered two questions of how frequently (1) they and (2) their partner used physical discipline (e.g., spank, shake, grab) towards their child during the last three months (Dodge et al., 1994). Mothers reported answers on 5-point scales to yield a total score of physical punishment. Responses included: never (0), once/month (1), once/week (2), daily (3), and several times daily (4). Dodge and colleagues (1994) found high test–retest reliability (.80) and strong validity in relation to other measures of harsh parenting. Due to the low frequency of physical punishment, we created a rank-order scale based on the frequency with which the mother reported that her child received physical punishment from either parent (Kerr et al., 2004). For example, the lowest score indicated no physical punishment from either parent. The next lowest score was assigned to children who received physical punishment once per month by one parent but were not physically punished by the other. Children who experienced physical discipline several times daily from both parents received the highest score. Following this procedure, a total score of physical punishment was yielded. To validate mothers' reports of physical punishment, we calculated its correlation with fathers' report ($r = .43$, $p < .001$, $n = 135$).

Child internalizing symptoms (at Ages 3, 6, and 10)

Mothers and fathers completed the CBCL for Ages 2–3 (Achenbach, 1992) at 3 years, and the CBCL for Ages 6–18 (Achenbach & Rescorla, 2001) at 6 and 10 years. The broadband Internalizing Problems scale was used in our study. Since different versions of CBCL have different items to reflect symptoms at different developmental stages, *T*-scores were used in all three waves. To obtain a multi-informant index of children's symptoms, we averaged across maternal and paternal measures when available ($\alpha = .85$, 84, and .90, at ages 3, 6, and 10, respectively). Both CBCL 2–3 and CBCL 6–18 have been found to have high test–retest reliability and solid content and concurrent validity (Achenbach & Rescorla, 2001; Achenbach, 1992).

Depression and anxiety symptoms in emerging adulthood (at Age 19)

At W4, young adults self-reported their levels of anxiety and depressive symptoms using the YSR Scale (Achenbach, 1991). DSM-oriented scales of Depression and Anxiety were used in our study, that have been shown sound validity in terms of associations with DSM-IV and other standardized rating scales of psychopathology (Achenbach et al., 2003). For consistency with measures of prior internalizing symptoms, we also used *T*-scores to represent depression and anxiety symptoms in emerging adults. 7.8% and 6.6% of participants satisfied scores to be considered as clinical ($T \geq 70$) and subclinical ($T \geq 65$) range of depression, respectively. Similarly, 5.6% and 9.1% of emerging adults endorsed clinical and subclinical ranges of anxiety, respectively. Both depressive ($\alpha = .83$) and anxiety symptoms ($\alpha = .82$) had good internal consistency.

Concurrent contextual risks and responsive parenting (Age 19)

In order to take into account the variability of concurrent environmental risks, we planned to include age 19 contextual risk and parenting behaviors as covariates. For contextual risks, we used mothers' reports of a single-item question about life stresses during the past three years (i.e., "During the past 3 years, have there been any events in your family life that you consider especially stressful?"). To measure W4 parenting behaviors, mothers completed a questionnaire about how they would deal with their child's misbehaviors (Dodge et al., 1990; Lansford et al, 2006). Specifically, responsive parenting consisted of three items, including (1) tell child how or how not to behave, (2) talk and explain reasons, and (3) promise reward for good behavior. The frequency of each type of parenting behavior was rated on a 5-point scale (1 = never, 2 = less than once a month, 3 = about once a month, 4 = about once a week, 5 = about every day). Responsive parenting showed good internal consistency ($\alpha = .77$).

Data analysis plan

All statistical analyses were conducted using SPSS 21.0 and Mplus 8.0. After examining descriptive statistics and correlations among all variables, the main analyses proceeded in three steps. First, we conducted CFA to examine whether different parenting measures can be loaded into a latent factor. Next, SEM was conducted to test our conceptual model (Figure 1). To test developmental cascade models following the hypotheses 1–3, variables at earlier developmental periods were modeled as predictors of variables at next time point. Specifically, age 3 contextual risks and internalizing symptoms were modeled as predictors of negative parenting and internalizing symptoms at age 6. Subsequently, pathways from age 6 negative parenting and internalizing symptoms to age 10 internalizing symptoms, as well as pathways from age 10 internalizing symptoms to age 19 depressive and anxiety symptoms were included in the model. In addition, considering the potential bidirectional associations between parenting and child internalizing symptoms, we modeled the correlation between age 6 negative parenting and age 6 internalizing symptoms. Finally, given the high comorbidity between depression and anxiety symptoms, we allowed correlation between these two outcome variables. Gender was included as a covariate of all outcome variables in the model given gender differences in the prevalence of

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Table 1. Means and standard deviations of the key variables

Variables	*M*	*SD*	Range
W1 Contextual risks index	1.07	.92	.20–5.72
W2 Negative appraisals	1.81	.50	1–3.83
W2 Caregiving inefficacy	1.67	.44	1–3
W2 Unresponsiveness	.00	1.7	−2.02 to 6.86
W2 Physical punishment	4.13	5.6	0–32
W1 Internalizing symptoms	47.03	8.55	29–71
W2 Internalizing symptoms	46.79	7.88	33–73
W3 Internalizing symptoms	50.16	9.63	33–77
W4 Depression symptoms	57.62	8.03	50–86
W4 Anxiety symptoms	56.58	7.43	50–83

internalizing disorders and research evidence showing gender-differentiated pathways of internalizing symptoms (Grant & Weissman, 2007; Gutman & McMaster, 2020). Family SES was not included as a covariate, as the families in our study were predominantly middle-class sample. As the last step, bias-corrected bootstrap analyses were conducted using BCBOOTSTRAP command (bootstrap = 10,000) in Mplus to examine mediation pathways in our model (MacKinnon et al., 2010; Shrout & Bolger, 2002). Even though we were interested in complex cascading pathways consistent with a transactional framework, we also expected that single domains of risk at ages 3 and 6 might have direct associations with age 19 depression and anxiety symptoms. For example, negative parenting quality at age 6 may have a direct association with higher levels of depression and anxiety symptoms at age 19 while controlling for indirect pathways and prior internalizing symptoms. Therefore, measures of contextual risk and parenting quality were modeled as predictors of age 19 depression and anxiety symptoms to capture the potential long-lasting associations. Finally, we tested the same conceptual model using the bifactor model approach.

Model parameters were estimated via full information maximum likelihood estimation with robust standard errors robust to nonnormality and handles missing data well (Huber, 1967; White, 1982; Yuan & Bentler, 2000). To establish model fit, multiple fit statistics are interpreted as outlined by Kline (2004). Specifically, we used scaled Chi-square statistic, CFI, TLI, SRMR, and RMSEA. For the excellent fit, the scaled Chi-square statistic should be non-significant, CFI and TLI should exceed 0.95, SRMR should be less than 0.08, and RMSEA should be less than 0.05. Standardized beta coefficients and 95% CIs were reported for all analyses.

Results

Preliminary analyses

Means and *SD* of the key variables are represented in Table 1. Intercorrelations among the study variables are reported in Table 2. At the bivariate level, the age 3 contextual risk index was significantly correlated with child internalizing symptoms at ages 3 and 6. Generally, measures of negative parenting at age 6 were significantly associated with one another and significantly correlated with prior internalizing symptoms. Those measures had some associations with concurrent and subsequent internalizing symptoms as well, particularly for caregiving efficacy. In addition, internalizing symptoms in different developmental periods were significantly associated with one another. Depression and anxiety symptoms at age 19 showed high levels of inter-correlation. However, age 19 contextual risk and responsive parenting were not significantly correlated with concurrent depression and anxiety symptoms or with earlier environmental risks. Therefore, we did not include those measures in our final SEM model as covariates in order to preserve a better model fit.

Moreover, cross-informant (mother, father, child) correlations of internalizing symptoms within and across ages are presented in Table 3. In general, 5 out of 18 lagged cross-informant correlations were significant. Specifically, mothers' and fathers' reports of internalizing symptoms were all significantly correlated within ages, and largely correlated across ages (4 out of 6 correlations were significant). Depression and anxiety symptoms reported by their adult children at age 19 were not significantly correlated with prior internalizing symptoms reported by mothers and fathers, except for the positive correlation between child-reported anxiety symptoms at age 19 and mother-reported internalizing symptoms at age 10 (1 out of 12 correlations were significant).

CFA: negative parenting

Prior to computing the SEM model, we conducted a CFA to examine whether four different variables related to parenting attitudes and behaviors (i.e., maternal reports of negative appraisals about child behavior, self-perceived parenting inefficacy, unresponsiveness, and physical punishment) could be combined into a latent construct of negative parenting. Figure 2 shows the results of the CFA on negative parenting. Results indicated a single-factor latent construct of negative parenting, in which all factor loadings were statistically significant. Given the good fit of the CFA model and high correlations among parenting measures, we decided to use the latent construct of negative parenting in further analyses.

Structural equation model from an individual symptom dimension approach

Figure 3 shows the results of the structural equation model to examine developmental pathways leading to depression and anxiety symptoms from early childhood to emerging adulthood. Note that only significant pathways are shown in Figure 3 and gender was included as a covariate. The final model had excellent model fit, with non-significant chi-square statistic, high CFI and TLI, and good RMSEA and SRMR.

In general, risk factors in the prior wave were associated with factors measured in subsequent waves. For example, higher levels of contextual risk at age 3 were associated with more negative parenting quality at age 6 and higher internalizing symptoms at ages 3 and 6. Similarly, internalizing symptoms at age 3 were positively associated with negative parenting at age 6.

Supporting the rank-order stability of internalizing problems, internalizing symptoms in the previous wave significantly predicted symptoms in the next wave. Specifically, higher levels of internalizing symptoms at age 3 were significantly associated with higher levels of internalizing symptoms at age 6, which in turn were associated with higher internalizing symptoms at age 10. Internalizing symptoms at age 10 were positively associated with anxiety symptoms at age 19, but not with depression symptoms. Internalizing symptoms at age 3 were directly associated with internalizing symptoms at age 10, taking into account for the

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Table 2. Bivariate correlations among key variables

Variable		1	2	3	4	5	6	7	8	9	10	11	12
1	Child gender	–											
2	W1 Contextual risks index	.19**	–										
3	W2 Negative appraisals	−.11	.13	–									
4	W2 Caregiving inefficacy	−.10	.16*	.37**	–								
5	W2 Unresponsiveness	−.09	.09	.44**	−.37**	–							
6	W2 Physical punishment	−.13	.05	.27**	.23**	.34**	–						
7	W1 Internalizing symptoms	.06	.17**	.13*	.19**	.10	−.03	–					
8	W2 Internalizing symptoms	−.10	.18**	.20**	.22**	.08	.15*	.35**	–				
9	W3 Internalizing symptoms	−.11	.14	.25**	.20**	.11	.09	.33**	.51**	–			
10	W4 Depression symptoms	.04	−.06	.13	.25**	.20*	.05	.03	.06	.16	–		
11	W4 Anxiety symptoms	.13	−.13	.14	.15	.11	.06	.06	.11	.26**	.64**	–	
12	W4 Stressful life events	.02	−.04	−.09	.09	−.08	.24**	−.13	.00	−.01	.08	.13	–
13	W4 Responsive parenting	.16	.04	.15	.03	.10	.04	−.11	−.05	.01	.01	.12	.00

Note. *$p < .05$, **$p < .01$; Child gender: Boy = 0, Girl = 1; W1 = age 3, W2 = age 6, W3 = age 10, W4 = age 19.

Table 3. Cross-informant correlations of internalizing symptoms within and across ages

Variable		1	2	3	4	5	6	7
1	W1 Internalizing symptoms, mother report	–						
2	W1 Internalizing symptoms, father report	.38**	–					
3	W2 Internalizing symptoms, mother report	.40**	.15	–				
4	W2 Internalizing symptoms, father report	.14	.36**	.33**	–			
5	W3 Internalizing symptoms, mother report	.34**	.23**	.54**	.25**	–		
6	W3 Internalizing symptoms, father report	.09	.25*	.39**	.39**	.53**	–	
7	W4 Depressive symptoms, child report	.05	−.11	.07	.05	.14	.10	–
8	W4 Anxiety symptoms, child report	.02	.05	.14	.07	.22*	.24	.64**

Note. *$p < .05$, **$p < .01$; W1 = age 3, W2 = age 6, W3 = age 10, W4 = age 19.

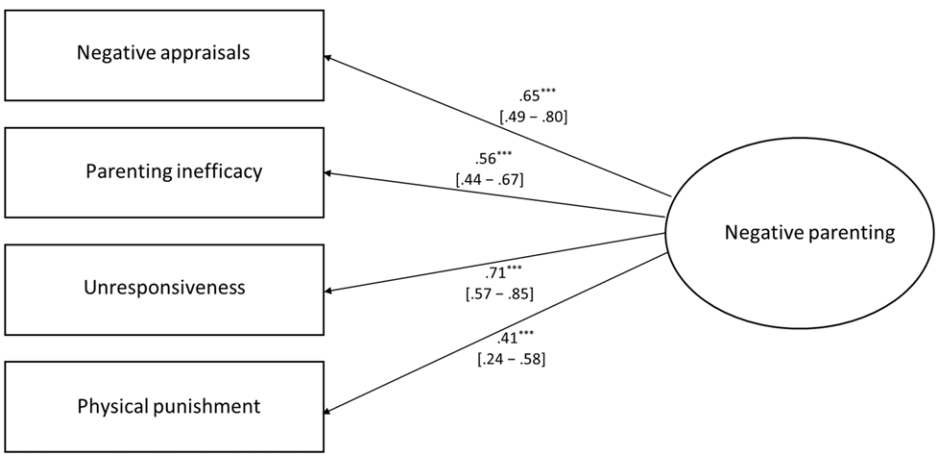

Figure 2. CFA on negative parenting at age 6. *Note*. Standardized beta scores and 95% CI are reported; ***$p < .001$; The model had a good fit: $\chi 2$ (2) = .62, $p = .73$, RMSEA = .00, CFI = .1, TLI = .1, SRMR = .01.

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Table 4. Indirect effects on emerging adult depression and anxiety symptoms from an individual symptom dimension approach

	W4 Depressive symptoms			*W4 Anxiety-symptoms*		
Indirect pathways	Unstand. Estimate	*SE*	95% CI	Unstand. Estimate	*SE*	95% CI
W1 Contextual risks index → W2 Negative parenting →	**0.512**	**0.3**	**.096–1.288**	**0.31**	**0.22**	**.029–.896**
W1 Contextual risks index → W2 Negative parenting → W3 Internalizing symptoms →	0.04	0.04	−.006 to .210	0.059	0.06	−.003 to .249
W1 Contextual risk index → W2 Internalizing symptoms →	0.011	0.16	−.294 to .393	0.063	0.14	−.181 to .403
W1 Contextual risk index → W2 Internalizing symptoms → W3 Internalizing symptoms →	0.101	0.08	−.005 to .334	**0.15**	**0.09**	**.039–.423**
W1 Internalizing symptoms → W2 Negative parenting →	**0.05**	**0.03**	**.008–.130**	**0.03**	**0.02**	**.002–.097**
W1 Internalizing symptoms → W2 Negative parenting → W3 Internalizing symptoms →	0.004	0.01	−.001 to .022	0.006	0.01	.000–.026
W1 Internalizing symptoms → W2 Internalizing symptoms → W3 Internalizing symptoms →	0.022	0.02	−.003 to .061	**0.032**	**0.02**	**.009–.075**

Note. Unstandardized estimates and Bias-corrected 95% CI are reported; Bold numbers indicate statistically significant pathways; W1 = age 3, W2 = age 6, W3 = age 10, W4 = age 19.

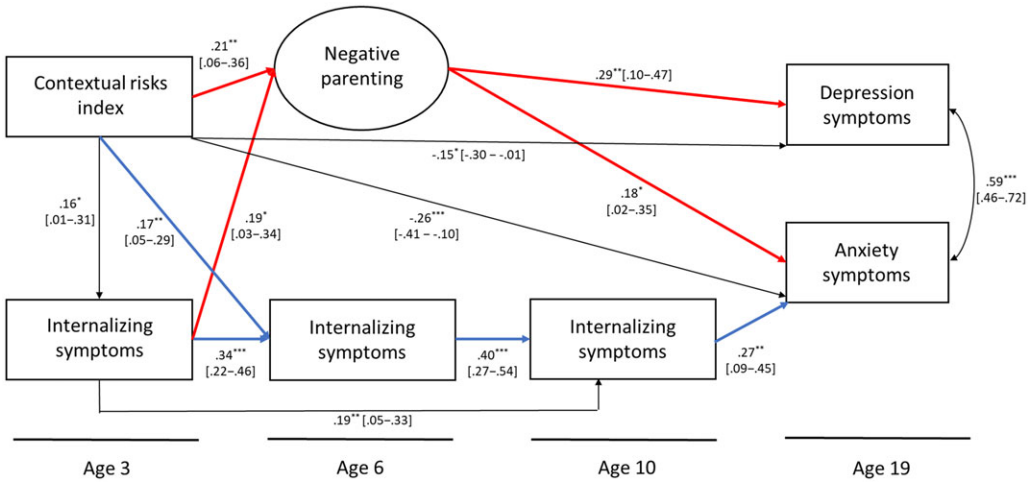

Figure 3. The empirical results of the SEM from an individual symptom dimension approach. *Note*. Standardized beta scores and 95% CI are reported; Only significant pathways are presented in the figure; *$p < .05$, **$p < .01$, ***$p < .001$; Significant mediation paths are indicated by weighted lines; The model had a good fit: $\chi 2$ (23) = 24.03, $p = .40$, CFI = .997, TLI = .993, RMSEA = .014, SRMR = .033; Child gender was included as a covariate.

variability of internalizing symptoms at age 6 to age 10. In this model, depression and anxiety symptoms at age 19 showed significant positive correlations, reflecting that they are highly related but separate constructs.

Initially, we modeled bivariate pathways from earlier environmental risk factors to age 19 depression and anxiety symptoms. In our model, more negative parenting quality at age 6 predicted higher levels of depression and anxiety symptoms at age 19, controlling for prior internalizing symptoms. The contextual risk index at age 3 was negatively associated with depression symptoms and anxiety symptoms at age 19. Gender was included as a covariate in this model and significantly associated with several risk factors. Specifically, parents showed more negative parenting towards boys than girls ($\beta = -.22$, $p < 01$, CI = −.36, −.07). Compared with boys, girls showed lower internalizing symptoms at age 6 ($\beta = -.16$, $p < .05$, CI = −.28, −.03) and higher levels of anxiety symptoms at age 19 ($\beta = .24$, $p < .01$, CI = .09, .39). There were no other significant gender differences.

Mediation pathways

Finally, indirect effects were examined to explore cascading developmental pathways associated with depression and anxiety symptoms in emerging adults. Results are reported in Table 4. We found indirect paths from the contextual risk index at age 3 to depression and anxiety symptoms at age 19. Specifically, higher levels of contextual risks at age 3 were associated with depressive symptoms at age 19, mediated by more negative parenting quality at age 6. This pathway supports partial mediation, given that all other indirect paths from age 3 contextual risks to age 19 depressive symptoms were non-significant, except for the direct path. In addition, higher levels of contextual risk at age 3 predicted increased anxiety symptoms at age 19, mediated by higher levels of internalizing symptoms at ages 6 and 10. In support of partial mediation, parenting at age 6 also mediated the relationship between the age 3 contextual risk index and age 19 anxiety symptoms.

Moreover, indirect paths from internalizing symptoms at age 3 to depression and anxiety symptoms at age 19 were observed.

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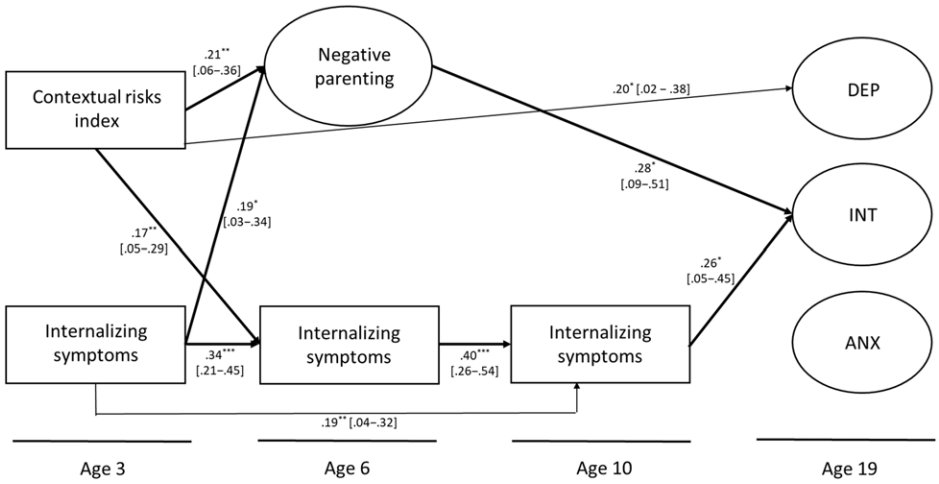

Figure 4. The empirical results of the SEM from a bifactor model approach. *Note*. Standardized beta scores and 95% CI are reported; Only significant pathways are presented in the figure; *$p < .05$, **$p < .01$, ***$p < .001$; Significant mediation paths are indicated by weighted lines; The model had a good fit: $\chi 2\ (29) = 38.521$, $p = .11$, CFI = .966, TLI = .923, RMSEA = .038, SRMR = .043; Child gender was included as a covariate.

Specifically, higher levels of internalizing symptoms at age 3 were associated with more negative parenting at age 6, which predicted higher levels of depression symptoms at age 19. This supports full mediation, given that all other indirect paths and direct path were not significant. On the other hand, in support of partial mediation, internalizing symptoms at age 3 had a significant indirect effect on anxiety symptoms at age 19 via internalizing symptoms at ages 6 and 10, and via negative parenting at age 6.

Structural equation model from a bifactor model approach

Finally, in secondary analyses, we examined the same structural equation model using a bifactor model approach. Specifically, we modeled outcome variables at age 19 (i.e., depression and anxiety symptoms) with a bifactor model to partition the variance into common (general internalizing symptoms) versus depression- and anxiety-specific symptoms. To examine the structure of internalizing psychopathology at age 19, we conducted a CFA using 20 items from the YSR DSM-oriented scales of Depression and Anxiety. The results indicated good fit and confirmed both general internalizing latent factor and symptom-specific (i.e., depression and anxiety specific) latent factors (please see Supplemental material for more information on CFA for internalizing bifactor model at age 19). Second, using the factor scores resulted from CFA as outcome variables at age 19, we conducted SEM to examine developmental pathways leading to depression and anxiety. As indicated in Figure 4, the final model had good fit, with non-significant chi-square statistic, acceptable CFI and TLI, and good RMSEA and SRMR. Note that only significant pathways are shown in Figure 4 and gender was included as a covariate.

As with the previous model, risk factors at age 3 were associated with factors measured in subsequent waves. Specifically, more contextual risk at age 3 was associated with higher levels of negative parenting quality and child internalizing symptoms at age 6. In addition, age 3 internalizing symptoms were positively associated with age 6 negative parenting. Internalizing symptoms in the previous wave significantly predicted symptoms in the next wave, supporting the rank-order stability of internalizing problems. Specifically, higher levels of internalizing symptoms at age 3 were associated with higher levels of internalizing symptoms at age 6, which in turn were associated with higher internalizing symptoms age 10. Internalizing symptoms at age 10 were positively associated with general internalizing latent factor at age 19. In addition, age 3 internalizing symptoms were directly associated with age 10 internalizing symptoms, controlling for the variability of internalizing symptoms at age 6.

Even though we were interested in cascading pathways, we also expected that there may be direct associations between early childhood risk factors and age 19 outcome latent variables (controlling for prior internalizing symptoms in early and middle childhood, as with the previous model). Specifically, higher levels of contextual risk at age 3 were associated with higher scores on the depression-specific latent factor, but not with the anxiety-specific or general internalizing latent factors. In addition, more negative parenting at age 6 directly predicted higher scores on the general internalizing latent factor but did not have direct associations with symptom-specific latent factors. Child gender was included as a covariate in this model and significantly associated with several risk factors and outcome variables. For example, parents showed more negative parenting towards boys than girls in early childhood. Compared with boys, girls showed higher factor scores on general internalizing and anxiety-specific latent factors, but not on the depression-specific latent factor.

Mediation pathways

As reported in Table 5, we found indirect pathways from risk factors at age 3 to the general internalizing latent factor at age 19. First, higher levels of contextual risk at age 3 were associated with the general internalizing latent factor at age 19, mediated by more negative parenting at age 6. In support of partial mediation, internalizing symptoms at age 6 and 10 also mediated the association between contextual risks at age 3 and the general internalizing factor at age 19. Moreover, results indicated indirect paths from age 3 internalizing symptoms to the age 19 general internalizing latent factor. Specifically, higher levels of internalizing symptoms at age 3 were associated with more negative parenting at age 6, which in turn predicted higher factor scores on general internalizing latent factor. In support of partial mediation, the association between age 3 internalizing symptoms and age 19 general internalizing latent factor was mediated by internalizing symptoms at

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Table 5. Indirect effects on emerging adult depression and anxiety symptoms from a bifactor model approach

	W4 Internalizing general			*W4 Depression-specific*			*W4 Anxiety-specific*		
Indirect pathways	Unstand. Estimate	*SE*	95% CI	Unstand. Estimate	*SE*	95% CI	Unstand. Estimate	*SE*	95% CI
W1 Contextual risks index → W2 Negative parenting →	**0.055**	**0.03**	**.012–.148**	0.018	0.02	−.017 to .078	0.003	0.02	−.034 to .047
W1 Contextual risks index → W2 Negative parenting → W3 Internalizing symptoms →	0.007	0.01	.000–.027	−0.002	0	−.018 to .002	0.001	0	−.002 to .014
W1 Contextual risk index → W2 Internalizing symptoms →	0.001	0.02	−.031 to .036	0.006	0.02	−.019 to .047	0.003	0.02	−.031 to .038
W1 Contextual risk index → W2 Internalizing symptoms → W3 Internalizing symptoms →	**0.017**	**0.01**	**.004–.047**	−0.004	0.01	−.023 to .006	0.003	0.01	−.008 to .022
W1 Internalizing symptoms → W2 Negative parenting →	**0.005**	**0**	**.001–.015**	0.002	0	−.002 to .007	0	0	−.003 to .005
W1 Internalizing symptoms → W2 Negative parenting → W3 Internalizing symptoms →	0.001	0	.000–.003	0	0	−.002 to .000	0	0	.000–.002
W1 Internalizing symptoms → W2 Internalizing symptoms → W3 Internalizing symptoms →	**0.004**	**0**	**.001–.008**	−0.001	0	−.004 to .002	0.001	0	−.002 to .004

Note. Unstandardized estimates and Bias-corrected 95% CI are reported; Bold numbers indicate statistically significant pathways; W1 = age 3, W2 = age 6, W3 = age 10, W4 = age 19.

ages 6 and 10. There were no significant indirect pathways from risk factors at age 3 to symptom-specific depression and anxiety latent factors at age 19.

Discussion

Multiple domains of risk factors (e.g., contextual, familial, and child individual vulnerabilities) have been associated with internalizing problems in emerging adulthood. However, there have been few longitudinal studies of internalizing psychopathology that cover a lengthy period of time, from early childhood to emerging adulthood. Moreover, we have limited knowledge of common and differential developmental pathways to depression and anxiety symptoms. Therefore, guided by ecological-transactional models of development and developmental psychopathology framework, we examined early common and differential risk pathways associated with depression and anxiety symptoms in emerging adulthood. Strengths of our study included the longitudinal design with multiple waves from early childhood to emerging adulthood, risk factors across multiple domains, and the inclusion of depression and anxiety symptoms in the same model. Overall, we identified multiple cascading risk processes that were associated with increased depression and anxiety symptoms in emerging adulthood. Specifically, we found common risk pathways to depression and anxiety symptoms, such that early childhood risk factors were associated with later internalizing symptoms mediated by negative parenting quality. The results also suggested differential risk pathways leading to depression and anxiety symptoms via different mediators.

Our first research goal was to examine cascading pathways from early childhood contextual risks to emerging adult depression and anxiety symptoms. Specifically, we hypothesized that children's exposure to multiple contextual risks in early childhood would be linked to age 19 depression and anxiety symptoms via negative parenting and prior internalizing symptoms. Partly consistent with this first hypothesis, we found significant mediational pathways from age 3 contextual risks to age 19 depression and anxiety symptoms, via common and different mediators. Specifically, higher levels of contextual risk at age 3 were associated with increased depression and anxiety symptoms at age 19, via more negative parenting at age 6. On the other hand, higher levels of prior internalizing symptoms at ages 6 and 10 served as unique mediators between higher contextual risks at age 3 and higher levels of anxiety but not depression symptoms at age 19. In support of partial mediation, there were direct associations between age 3 contextual risks and age 19 depressive and anxiety symptoms.

These findings are consistent with several theories (Bronfenbrenner & Morris, 2007; Conger et al., 2002; Evans et al., 2013) and research findings (Golm et al., 2020; Gutman et al., 2019; Trentacosta et al., 2008) that highlight the role of broader contextual stressors during early childhood as important risks for later internalizing symptoms. As an extension of prior literature, our findings suggest that early contextual risks may be predictive of long-term emotional problems into emerging adulthood as the start point of a cascade effect. In addition, the mediators in our study (i.e., negative parenting quality, prior internalizing symptoms) linking earlier contextual risks and later depression and anxiety symptoms are in line with prior studies that emphasized parenting and child characteristics as proximal factors contributing to the development of internalizing symptoms (Côté et al., 2009; Hopkins et al., 2013; Karevold et al., 2009; Kim & Brody, 2005). These indirect paths also provide support for ecological–transactional models of the development of internalizing symptoms (Cicchetti & Lynch, 1993; Sameroff, 1975). That is, how distal environmental risk factors are associated with more proximal factors in later years (i.e., more negative parenting, higher child internalizing symptoms) and in turn, contribute to increased risk for depression and anxiety symptoms in emerging adulthood. The direct association between early childhood contextual risks and emerging adult depression and anxiety symptoms could suggest the possibility of other mediators, such as children's suboptimal emotion regulation strategies (Lengua et al., 2015). Alternatively, this direct association may be explained by the persistence of environmental risks from early childhood throughout emerging adulthood. That is, children who experience more contextual stressors and/or negative parenting than others may face ongoing experiences of risks during later adolescence or emerging adulthood, which may have linked to more emotional

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distresses during emerging adulthood. Together, our findings underscore the relevance of multiple contextual risks for later emotional adjustment among community samples of children, who might be exposed to multiple but less severe environmental adversities during early childhood compared to high-risk populations.

The second aim of our study was to examine the rank-order stability of internalizing symptoms from early childhood through emerging adulthood. Partly supporting the hypothesis, we found significant indirect pathways starting from age 3 internalizing symptoms to age 19 depression and anxiety symptoms. Similar to the results for the first hypothesis, negative parenting at age 6 mediated the relationship between age 3 internalizing symptoms and age 19 depression and anxiety symptoms. These pathways provide hints for possible bidirectional associations between parents and children: not only do parents contribute to children's adjustment, the child's own vulnerability, such as early internalizing symptoms, may elicit less optimal parenting behaviors. Indeed, prior studies have supported child-driven effects, such that children's problem behaviors or specific temperament (e.g., higher levels of negative affectivity; low self-control) provoke suboptimal parenting, which in turn partially accounts for the development of later internalizing symptoms (Gouze et al., 2017; Shewark et al., 2021; Yan & Ansari, 2016). In support of partial mediation, we also found that internalizing symptoms at ages 6 and 10 mediated the relationship between age 3 internalizing symptoms and age 19 anxiety symptoms. That is, children who showed higher levels of internalizing symptoms in early childhood continue to exhibit similar problems throughout the middle childhood, and in turn, are likely to endorse anxiety symptoms in emerging adulthood. In line with prior studies, this pathway provides additional evidence for the developmental consistency of internalizing symptoms, especially anxiety symptoms (Ellis et al., 2017; McElroy et al., 2018), and shows that the individual differences of internalizing symptoms persist across long developmental periods – in this case, the period between early childhood and emerging adulthood.

Third, we aimed to understand whether there are common or unique developmental pathways to anxiety and depression symptoms at age 19 as an exploratory hypothesis. Our empirical model suggested both common and different pathways associated with emerging adult depression and anxiety symptoms, such that the risk factors during early childhood were associated with depression and anxiety through same and different mediators. Specifically, age 6 negative parenting quality served as a common mediator linking early childhood risk factors and both depressive and anxiety symptoms. On the other hand, prior internalizing symptoms (ages 6 and 10) uniquely mediated the associations between early childhood risk factors and emerging adult anxiety symptoms. The finding that the negative parenting quality serves as a common mediator for emerging adult depression and anxiety converges with a large body of research emphasizing the role of parenting on the development of internalizing symptoms (Kim & Brody, 2005; Trentacosta et al., 2008; Yap et al., 2014). Although our study highlights a broad measure of parenting quality as a common proximal risk factor, we note that prior studies have shown differential associations between specific types of parenting behaviors on depression and anxiety symptoms (Hopkins et al., 2013; Rapee et al., 2009). For example, overprotective parenting has been suggested as a strong predictor of children's later anxiety symptoms, whereas harsh or critical parenting has been related to depressive symptoms (Rapee et al., 2009). Therefore, extending the findings of our study, in future research it would be helpful to include specific types of parenting behaviors in a long-term longitudinal study. This would provide more sophisticated knowledge about common and differential cascading pathways associated with depression and anxiety symptoms (e.g., whether and how specific types of parenting differentially mediate links between early childhood risk factors and emerging adult depression and anxiety symptoms).

Our finding that the prior internalizing symptoms were mediators of anxiety, but not depression, may be explained by the principles of homotypic and heterotypic continuity in internalizing problems. That is, the development of anxiety symptoms may reflect the process of homotypic continuity, where internalizing problems during childhood predict the occurrences of same symptoms over time (Snyder et al., 2017). Indeed, prior studies have shown that anxiety disorders are one of the earliest occurring psychopathologies with consistent prevalence rates over development and moderate to strong homotypic continuity within anxiety disorders (Fitzgerald & Taylor, 2015; Rapee et al., 2009). On the other hand, depression symptoms might develop in a less homotypic way and follow heterotypic continuity (Lahey et al., 2014). This speculation is aligned with prior studies showing that the depression follows a discontinuous symptom trajectory whereas anxiety symptoms are more dependent on the pattern of preexisting symptoms (Cohen et al., 2018; Rutter, Kim-Cohen, et al., 2006). Moreover, researchers have found that other types of disorders, such as oppositional defiant disorders, predict subsequent depressive symptoms even after controlling for initial levels of depression (Lavigne et al., 2014). We cannot confirm that these findings reflect heterotypic continuity since we did not include other psychosocial impairments in our model, but it is plausible that early environmental factors or parenting may contribute to different psychological symptoms such as poor emotion regulation or externalizing problems, which in turn predict depression symptoms in a later time point.

Alternatively, the different mediators associated with later depression and anxiety symptoms might be attributable to measurement issues and the developmental timing of internalizing symptoms. For example, compared to depression, anxiety symptoms tend to emerge more frequently in earlier years (Sterba et al., 2007). Since we measured prior internalizing symptoms during childhood and preadolescence, it is possible that the internalizing symptoms might primarily reflect vulnerabilities to anxious responding and thus were significantly associated with later anxiety, not depression, symptoms. Given that depressive symptoms have a later onset (Kessler et al., 2005), future studies of internalizing symptoms that measure depression during the adolescent to adult transition may help us comprehensively understand whether and to what extent depression and anxiety symptoms show common or differential developmental pathways.

Finally, we examined the same conceptual model leading to depression and anxiety symptoms from a bifactor modeling approach, using factor scores of general internalizing and symptom-specific measures as outcome variables. This new model indicated several indirect pathways from age 3 risk factors to the age 19 general internalizing latent factor. Specifically, the contextual risk index at age 3 was associated with the general internalizing factor score at age 19 via negative parenting at age 6, and via internalizing symptoms at ages 6 and 10. This pattern of results is consistent with recent studies from empirical models of psychopathology indicating the association between environmental risks and internalizing factor (Deutz et al., 2020; Lynch et al., 2021; Wilson et al., 2015). As an extension, results from our study suggest that the accumulation of multiple types of contextual risks in early

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childhood may contribute to the common symptoms of internalizing disorders in emerging adulthood, either by shaping parenting quality or heightening the vulnerability for early internalizing symptoms. In addition, age 3 internalizing symptoms were associated with the age 19 general internalizing latent factor, partially mediated by age 6 negative parenting as well as ages 6 and 10 internalizing symptoms. The results support the rank-order stability of common internalizing symptoms, such that children presenting higher internalizing symptoms in early childhood are likely to manifest higher levels of symptoms related to both depression and anxiety through emerging adulthood.

On the other hand, there were no significant indirect effects from early risk factors to symptom-specific latent factors of depression and anxiety. In addition, negative parenting and prior internalizing symptoms did not significantly predict unique variance in the depression and anxiety symptoms. These results appear to be somewhat inconsistent with findings from the traditional symptom dimension approach, where early contextual risks and internalizing symptoms were predictive of emerging adult depression and anxiety symptoms via mediators. The different results in two models are not surprising given the partitioned variance of the outcomes in bifactor model. In other words, as all items measuring depression and anxiety symptoms were significantly loaded to the general internalizing latent factor with high factor loadings, significant indirect paths from symptom dimension approach turned out to be insignificant predictors of symptom-specific variances from bifactor model approach and instead predicted general variance in internalizing symptoms. The large variance of the general internalizing factor over the symptom-specific variances may indicate substantial overlap of depression and anxiety symptoms as well as their common developmental pathways (Simms et al., 2008). In addition, the result that indirect pathways were only predictive of the general variance of internalizing symptoms might suggest that early childhood risk factors heighten the risk of general internalizing psychopathology, rather than specific features of each disorder. It is possible that the accumulation of multiple risks in early years of life broadly confer the risk of general internalizing disorder, whereas temporally more proximal and developmentally specific risks contribute to the manifestation of specific syndromes (Hankin et al., 2016).

However, it is noteworthy that higher levels of contextual risk at age 3 were significantly associated only with higher levels of the depression-specific, and not anxiety-specific, latent variable. This finding emphasizes the unique effect of early contextual risk on later depressive symptomatology, suggesting the possibility that young adults who shows signs of depression might have been more affected by multiple negative experiences in early childhood, compared to those with anxiety symptoms. Alternatively, this may reflect the stability of contextual risks, rather than the long-lasting added effect of early risks. That is, the families with early multiple risks may continue to experience higher levels of adversity throughout the children's lifetimes, which serves as a concurrent risk factor for depressive symptoms in emerging adulthood. Therefore, future research would benefit by including measures of concurrent contextual risks, in addition to contextual risks in early childhood, to offer more sophisticated explanations on to what extent early versus consistent contextual risks contribute to the development of depressive symptoms. In sum, while the results from the bifactor model approach should be interpreted carefully as it is not our prespecified analysis, the findings in our study provide useful hints about early developmental pathways leading to the general factor of internalizing symptoms and specific features of depressive and anxiety disorders.

Our study had several limitations. First, although we successfully collected data from 64% of the sample in emerging adulthood, the modest size of the follow-up sample may have limited statistical power and reduced our capacity to detect some effects. Relatedly, given our relatively modest sample size, having complex models with several mediation paths may have resulted in overfitting, that potentially causes failures of fitting additional data. Replication efforts with independent and larger samples is needed. Second, reflecting the demography of our moderately sized university town, our participants were primarily from two-parent, middle-class Euro-American families. Therefore, our findings may not generalize to ethnically diverse families or those that are experiencing severe hardships or traumatic stressors. Despite several benefits of testing these risk factors in a community sample, our findings need to be replicated in other sociodemographic contexts to support the generalizability of the model in a broader range of family ecologies. Third, given that our results are based on correlational relationships between variables, we cannot assume causation. However, our study provides informative hints to developmental risk processes by encompassing multiple time points and controlling for prior internalizing symptoms. Fourth, even though we created a latent factor of negative parenting quality to incorporate various parenting attitudes and behaviors, we note that the internal consistency for some measures (i.e., negative appraisal of child behavior and self-perceived caregiving efficacy) were somewhat modest. Fifth, although we included multiple developmental periods from early childhood to emerging adulthood, there was a significant time gap in our data from ages 11 to 19 years. Given that internalizing symptoms, especially depressive symptoms, develop rapidly during this period, establishing more comprehensive longitudinal trajectories including adolescence period awaits future research. In addition, our study was not genetically informed. Although we found significant associations between parenting quality and child internalizing symptoms in multiple developmental periods, those observed associations may reflect different types of gene–environment correlations (e.g., evocative or passive rGE; Plomin et al., 1977; Rutter, Moffitt, et al., 2006). Genetically informed research such as adoption studies could help disentangle the role of genetic and environmental factors in the relationship between parenting and child internalizing symptoms. Moreover, while we were interested in common and differential risk processes within internalizing psychopathology, future studies would benefit from examining the risk processes associated with other outcomes, such as externalizing problems. Given that cumulative contextual risks and negative parenting quality are known to be predictive of future externalizing behaviors (Evans et al., 2013; Pinquart, 2017), such study would provide useful knowledge of whether those risk processes are transdiagnostic across psychopathology or unique precursors of internalizing outcomes. Finally, while exposure to multiple risks serves as a strong indicator of long-term adjustment (Evans et al., 2013), it is noteworthy that the contextual risk index in our study did not reveal the role of distinct environmental risks. For example, researchers have suggested that different dimensions of contextual risks, such as threat or deprivation, could be associated with later outcomes in distinct ways (McLaughlin & Sheridan, 2016; Zeanah & Sonuga-Barke, 2016). Similarly, while we weighted each risk factor equally in the contextual risk index, it is possible that some risks contributed more strongly to the index and later outcomes than others. In addition, including measures of concurrent or more proximal contextual risks (e.g., in adolescence) and negative parenting in the final model would help researchers

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disentangle complex relationships between early environmental risks and later adjustment outcomes, as to what extent early risks contribute to the development of internalizing symptoms after taking account for continuity in environmental stresses throughout the development. These are intriguing topics for further investigation.

In conclusion, our findings have several important implications. First, they provide a nuanced picture of developmental processes leading to internalizing symptoms from early childhood through emerging adulthood. Specifically, by integrating multilevel risk factors, including contextual, familial, and child characteristics, our results demonstrate the possibility of both common and differential transactional pathways to depression and anxiety. These findings show that even in a sample of primarily middle-class families, multiple environmental risks and parenting quality contribute to emotional problems across long expanses of development. Therefore, our study highlights the importance of early identification and prevention efforts that disrupt cascading pathways associated with emerging adult depression and anxiety.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/S0954579422001043

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