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Regular Article

Childhood adversity and adolescent mental health: Examining cumulative and specificity effects across contexts and developmental timing

Felicia A. Hardi[1,2], Melissa K. Peckins[3], Colter Mitchell[4,5], Vonnie McLoyd[1], Jeanne Brooks-Gunn[6,7], Luke W. Hyde[1,4] and Christopher S. Monk[1,4,8,9]

[1]Department of Psychology, University of Michigan, Ann Arbor, MI, USA, [2]Yale University, New Haven, CT, USA, [3]St. John's University, New York, NY, USA, [4]Survey Research Center of the Institute for Social Research, University of Michigan, Ann Arbor, MI, USA, [5]Population Studies Center of the Institute for Social Research, University of Michigan, Ann Arbor, MI, USA, [6]Teachers College, Columbia University, New York, NY, USA, [7]College of Physicians and Surgeons, Columbia University, New York, NY, USA, [8]Neuroscience Graduate Program, University of Michigan, Ann Arbor, MI, USA and [9]Department of Psychiatry, University of Michigan, Ann Arbor, MI, USA

Abstract

Associations between adversity and youth psychopathology likely vary based on the *types* and *timing* of experiences. Major theories suggest that the impact of childhood adversity may either be *cumulative* in type (the more types of adversity, the worse outcomes) or in timing (the longer exposure, the worse outcomes) or, alternatively, *specific* concerning the type (e.g., parenting, home, neighborhood) or the timing of adversity (e.g., specific developmental periods). In a longitudinal sample from the Future of Families and Wellbeing Study ($N = 4{,}210$), we evaluated these competing hypotheses using a data-driven structured life-course modeling approach using risk factors examined at child age 1 (infancy), 3 (toddlerhood), 5 (early childhood), and 9 (middle childhood). Results showed that exposures to more types of adversity for longer durations (i.e., cumulative in both type and timing) best predicted youth psychopathology. Adversities that occurred at age 9 were better predictors of youth psychopathology as compared to those experienced earlier, except for neglect, which was predictive of internalizing symptoms when experienced at age 3. Throughout childhood (across ages 1–9), aside from the accumulation of all adversities, parental stress and low collective efficacy were the strongest predictors of internalizing symptoms, whereas psychological aggression was predictive of externalizing symptoms.

Keywords: Adversity-specific; childhood adversity; cumulative; sensitivity periods; youth psychopathology

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Introduction

Extensive research has established that adverse experiences during development are associated with a host of poor outcomes, including risk for psychopathology (Cicchetti, 2016). Given the high prevalence of adversity during childhood (Kessler et al., 1997), considerable efforts have been made to understand how childhood adversities can serve as antecedents of psychopathology. Over several decades, many studies have focused on disentangling both the additive and distinct contributions of different types of adversities experienced at various developmental periods. Much of this work has focused on internalizing and externalizing symptoms during adolescence, as this is a period when many of the most significant symptoms of mental disorders emerge (Kessler et al., 2005). By identifying salient risk factors for youth mental health, this research aims to advance our understanding of the nature of the associations between childhood adversity and youth psychopathology, which can, in turn, inform intervention and prevention efforts.

Corresponding author: Christopher S. Monk; Email: csmonk@umich.edu

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The accumulation of different types of risk factors

Research on the cumulative risk model of adversity, which posits that mental health risks increase with the number of adversities that children experience (Figure 1), began with early seminal work such as the Isle of Wight (Rutter et al., 1979) and the Rochester Longitudinal Study (Sameroff & Seifer, 1995). These studies found that exposure to a *greater number* of environmental and family factors (e.g., marital problems, maternal stress and psychopathology, instability) was associated with greater psychopathology in adolescents. Guided by the ecological systems models (Bronfenbrenner & Morris, 2007), subsequent research expanded this work to include a broader array of risk factors across multiple contexts (e.g., parenting, family, neighborhood). These work provide evidence that a wide range of experiences such as harsh parenting, family violence, maternal stress, parental psychopathology, and financial hardship (e.g., Appleyard et al., 2005; Atkinson et al., 2015; Buehler & Gerard, 2013; Trentacosta et al., 2008) can have a combined effect on children's development (see review: Evans et al., 2013).

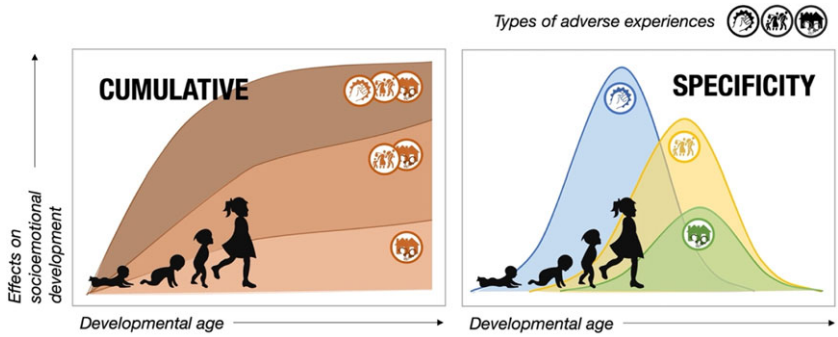

Figure 1. Theoretical illustration of the cumulative and specificity models of adversity. The cumulative model (left) suggests that the effects of childhood adversity on socioemotional development would increase with increasing number of adversity and longer exposures to various adverse experiences, up to a specific level whereby more exposures may not yield any additional effect. On the other hand, in the specificity model (right), each adverse experience is hypothesized to have a distinct effect on socioemotional development. The magnitude of influence (i.e., the height of the curves) of a specific adversity may vary across the different types of adverse experiences and when during development they occur (i.e., where they lie on the *x*-axis). These theoretical models provide a basis for much research on the effect of multiple types of adversity on youth mental health.

The accumulation of risk across multiple developmental periods

Taking a cumulative approach to understanding the effects of childhood adversity can also be extended to the consideration of the timing at which adverse events occur (Figure 1). For instance, many early studies on child maltreatment found that prolonged exposures to adversity (i.e., exposures to risks across multiple developmental periods) predicted greater youth psychopathology (Cicchetti & Toth, 1995; Egeland & Sroufe, 1981). Similar findings have been observed in more recent studies. For example, increased behavioral problems were more frequently observed in children who experienced maltreatment across multiple developmental periods, rather than specific time points (Dunn et al., 2018; Jaffee & Maikovich-Fong, 2011; Thompson et al., 2014; Thornberry et al., 2001). These findings collectively demonstrate that the effects of adversity on mental health may be proportional to the duration of exposure.

Specificity effect of childhood adversity across different types of events

Though understanding the cumulative effect of adversity across experiences and developmental periods is important, cumulative risk research struggles to elucidate the precise mechanisms by which adversity can lead to psychopathology because the risk factors themselves are examined collectively. Thus, in parallel, a host of research has focused on understanding the effect of specific adversities on specific psychopathology outcomes. For example, parenting practices have been found to be a critical factor for youth socioemotional development (Eisenberg et al., 1998; Morris et al., 2017; Rutherford et al., 2015). Both harsh parenting, characterized by coercion and aggression, and neglectful parenting, characterized by minimal attention and supervision, are robust risk factors for internalizing and externalizing outcomes (Chang et al., 2003; Hoffman-Plotkin & Twentyman, 1984; Shaw & Bell, 1993; Trickett & McBride-Chang, 1995). These parenting adversities increase the risk of internalizing symptoms by imposing stress and unpredictability, which disrupts the biological stress response (Loman & Gunnar, 2010; Repetti et al., 2002), while increasing the risk of externalizing behaviors by modeling aggression and interfering with the development of emotion regulation (Shaw & Bell, 1993).

Similarly, other adversities within the home environment can also present risks for later psychopathology by undermining socioemotional development. For instance, witnessing intimate partner violence exposes children to aggression and violence and models maladaptive conflict resolution and poor emotion regulation (Artz et al., 2014; Carpenter & Stacks, 2009). Parental psychological issues, such as maternal depression and parental stress, challenge parents' ability to be warm and responsive, which undermines the development of secure parent–child relationships (Cummings & Davies, 1994; Goodman et al., 2011; McQuillan & Bates, 2017). Frequent residential changes and material hardship have also been identified as risk factors for youth behavioral problems (Belsky et al., 2011; Doom et al., 2016; Gershoff et al., 2007), potentially because they increase distress for parents and introduce repeated ecological changes that children must adapt to. Thus, multiple adversities within the home can disrupt important child (e.g., development of emotion regulation) and dyadic processes (e.g., establishing a supportive parent–child relationship), which in turn can increase the risk for psychopathology.

In addition to family-level processes, neighborhood risk factors, such as low community support and high neighborhood violence, increase the risk of internalizing symptoms by exposing children to unpredictable threats and violent crimes (Sampson et al., 1997) and the risk of externalizing symptoms by providing opportunities for negative peer influence (Cantillon, 2006). Collectively, prior studies have demonstrated how different types of adverse events can have a specific influence on children's socioemotional development through multiple pathways and processes, and this knowledge has been key to informing approaches to prevent or treat youth psychopathology (Dodge, 2020).

Changes in children's environments across different developmental periods

The risks of each type of adversity for mental health may also differ depending on *when* during development these experiences occur. Childhood is marked by vast changes in the child's social and biological contexts within a short period of time. While scholars use different terms to describe various developmental periods, in the present study and subsequent discussion, we refer to data collected at age 1 as occurring during infancy, age 3 as toddlerhood, age 5 as early childhood, and age 9 as middle childhood, consistent with previous categorizations of this sample (James et al., 2021).

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During infancy, children are focused on learning basic processes and establishing secure attachments with their caregivers (Greenberg et al., 1990). From toddlerhood through the early preschool years, there are rapid changes in language, motor skills, and emotion processing. During this period, children begin to move independently and explore with increasing autonomy, while also learning to manage their "big" emotions and desire to maintain closeness with their caregivers. This can create significant challenges for parents as they navigate setting new boundaries, all while ensuring their children's safety (Gardner & Shaw, 2009). Another shift occurs when children begin to transition into formal schooling at around age 5. During this transition to elementary school, children begin spending more time out of the home, in school and neighborhood settings (Hofferth & Sandberg, 2001; Leventhal & Brooks-Gunn, 2000). As children approach the end of elementary school (e.g., around age 9), they prepare for another major contextual change of transitioning into middle school. This period also marks the onset of major biological changes, including puberty (in the present sample, 89% of girls and 81% of boys had begun pubertal development at age 9) and rapid development of brain regions critical to emotion and self-control (Blakemore, 2012). Socially, parents are more likely to allow children to explore their neighborhoods more independently (Smetana et al., 2006), and there is an increasing focus on peer groups compared to earlier ages, expanding the focus of social development beyond caregivers (Rubin et al., 2011).

These shifts across developmental periods have prompted research into how different types of experiences contribute to early risk for psychopathology, depending on the childhood period (i.e., infancy, toddlerhood, early childhood, middle childhood) during which the exposure occurs. Early in childhood, emotional development highly relies on parental influence; thus, disruptions in parent–child relationships during this period can be particularly impactful (Zeanah et al., 1997). For instance, childhood maltreatment (Cummings & Davies, 1994; Goodman et al., 2011; Hankin, 2005; Lee & Hankin, 2009), parental stressors (Crnic & Greenberg, 1990), and conflicts (Emery, 1982) at early ages can compromise parent–child bonds, foster insecure attachment in children, and produce cascading risks across childhood that could subsequently lead to internalizing and externalizing problems. As children mature from middle childhood into adolescence and begin to spend more time with their peers and engage socially outside of the home, it is hypothesized that neighborhood-level adversities can play a larger role for older children (Kleinepier & van Ham, 2018).

At the same time, other evidence suggests that these factors may have similar influences across different developmental periods. For instance, although it is commonly believed that children's environments expand beyond the home as they get older, neighborhood factors can be just as critical in early childhood. Affluent neighborhoods, for instance, offer young children access to resources and academic enrichment that promote school readiness (Anderson et al., 2019). Similarly, disruptions in caregiving can undermine children's emotional development during later childhood just as much as during earlier childhood. Children continue to rely on parents for security well beyond the early childhood period while learning to balance their competing desires for independence (Collins et al., 1995). Environmental instability, such as frequent residential changes or material hardship (e.g., eviction or food insecurity), is proposed to have detrimental effects regardless of whether it occurs in early or late childhood. For example, residential moves in early childhood are particularly impactful on children's development due to changes within the family context (Anderson et al., 2014; Gillespie, 2013; Heinlein & Shinn, 2000). In contrast, moves later in childhood can disrupt children's routines, especially when they involve changes in multiple environments, such as switching schools or social circles (Anastasio & Leventhal, 2023; Swanson & Schneider, 1999).

Sensitive periods of development

Beyond the idea that specific types of adversities may be more or less salient at different developmental periods, variations in the effects of childhood adversity over time could also be due to changes in the child's sensitivity to their environment (i.e., sensitive periods), rather than just changes within the child's environment. Sensitive periods refer to times when environmental influences exert the greatest impact on development, a concept rooted in early neurobiological research using animal models. For instance, Hubel and Wiesel demonstrated that depriving kittens of visual stimuli shortly after birth impairs their long-term visual system, whereas similar deprivation later in development leaves their sensory ability intact (Hubel & Wiesel, 1970). These findings motivated human researchers to better understand whether there are specific sensitive periods during which adversity has a particularly profound impact.

Consistent with this notion, some evidence suggests that early childhood may represent a sensitive period for adversity. For example, the seminal Bucharest Early Intervention Project revealed that removing children from institutional care became markedly less beneficial after the age of 2 (McLaughlin et al., 2015), underscoring the critical role of early caregiving for long-term socioemotional development. Similarly, in a study of maltreated children, childhood maltreatment, particularly before age 5, was found to predict the greatest risk for depression (Dunn et al., 2013; Kaplow & Widom, 2007). From a neurodevelopmental perspective, early childhood is a period of rapid development for critical neural structures involved in affective processing (Tottenham & Sheridan, 2010). In one study, for instance, maternal support during preschool (ages 3–6), but not school-aged (4–12), was associated with higher increases in hippocampal volume (Luby et al., 2016). Similarly, in a subsample of the current sample, harsh parenting at age 3 (but not later ages) was related to differences in amygdala reactivity (Gard et al., 2021). Taken together, these studies provide support that adversity experienced during early childhood may be particularly important for children's socioemotional development.

Though theory and these individual studies suggest that early childhood may be a sensitive period for adversity, other studies have questioned this notion. For instance, in one systematic review, Brett and colleagues found limited evidence supporting specific sensitive periods related to child maltreatment, noting that heightened responses to maltreatment have been associated with both early and late exposures (Brett et al., 2015). Contrary to the evidence on early childhood sensitive periods, studies have also found that maltreatment experienced later in childhood was more strongly associated with youth psychopathology than maltreatment experienced in early childhood (Dunn et al., 2023, 2018; Thornberry et al., 2010). Notably, in one study, Dunn and colleagues examined sensitive periods of child maltreatment in the same cohort as the present study. They found that harsh parenting was most strongly associated with parent report of youth internalizing and externalizing symptoms when experienced at age 9 for girls and age 5 for boys (Dunn et al., 2023). These findings suggest that heightened vulnerability to adversity may occur later

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in childhood. However, more research is needed to determine whether these findings extend to different types of adverse experiences and if similar results can be obtained using youth-reported measures of psychopathology.

Theory-informed, data-driven approaches

These various strands of research emphasizing the cumulative or potentially specific type and timing effects of adversity highlight the need to better understand the nature of the associations between adversity and youth psychopathology. While previous studies have examined the differential associations of adversity and psychopathology, as well as their cumulative effects (e.g., Shaw et al., 1998), few studies have directly compared these theoretical models within a single framework. For instance, studies have either explored the cumulative effects of multiple types of adverse experiences or the effects of adversity across multiple developmental time points or, in separate analyses, explored the associations of a few adverse experiences at specific time points with mental disorders in children and adolescents. However, to determine if a cumulative risk index could account for the majority of individual differences in internalizing and externalizing outcomes, a direct comparison of these models is needed. This comparison can help to disentangle whether the influence of childhood adversity primarily manifests as cumulative effects or whether certain developmental time points or experiences are especially detrimental.

One novel way to address these competing models is by leveraging a quantitative method called the Structured Life-Course Modelling Approach (SLCMA) (Smith et al., 2016, 2015; Smith et al., 2022). SLCMA is a theory-informed and data-driven method that leverages the least angle variable selection regression approach to identify the life-course hypothesis (e.g., cumulative, sensitive period) that could be best supported by the available data. In previous investigations, this method was used to examine the developmental impact of maternal depression (Lacey et al., 2023) and child maltreatment (Crawford et al., 2022; Dunn et al., 2023, 2018, 2019) on mental health. These studies directly compared the cumulative effect of repeated exposures to these adversities (across multiple time points) with their impact at specific developmental ages (i.e., early or late childhood sensitive periods). However, this approach has not been used broadly to assess the effect of an array of adversities across a range of developmental periods.

Moreover, further research is needed to compare these models in a large, well-sampled representative cohort. One of the many challenges of studying questions related to the nature of the associations between adversity and mental health is identifying a suitable sample for testing these disparate models. To accomplish this goal, a longitudinal cohort is needed that includes data on a wide range of risks, with continuous distributions of adversity and psychopathology, and a strong sampling frame for generalizability, particularly a cohort with increased representation of individuals exposed to elevated risks (i.e., a population with greater exposure).

A final consideration is the potential "shape" of the association between cumulative adversity over time and type. Originally, two types of cumulative risk models were proposed. Rutter and colleagues found that exposure to more risk factors, up to a certain number, incrementally increased problem behaviors (Rutter et al., 1979). These findings are consistent with the landmark epidemiological study on adverse childhood experiences (Felitti et al., 1998), which suggests that a specific threshold of risk exposures can have the greatest consequences on health. This quadratic cumulative model contrasts with other research that demonstrates a linear pattern, proposing a continuous escalation of maladaptive outcomes with exposure to a greater number of risk factors (Sameroff, 1998). Thus, investigations into the potentially cumulative effects of adversity also need to test whether models are linear or show ceiling/quadradic effects.

Present investigation

The present study aims to test the cumulative and specificity effects of adversity across a range of childhood periods and contexts to prospectively predict youth internalizing and externalizing problems using a novel theory-informed data-driven approach in a diverse, longitudinal, population-based sample spanning 15 years. We focused on risk factors with previously established and robust links to internalizing and externalizing problems using measures of parenting practices, home environment, and neighborhood environment. In terms of hypotheses, we first anticipated a stronger support for a cumulative risk model across both timing and types, indicating that risk exposures over an extended period, as well as across a greater number of risks, would have the largest impact on youth internalizing and externalizing problems. Additionally, we hypothesized that there would be differential associations relating to timing (sensitive periods) and adversity type (adversities that pose meaningful influence beyond cumulative effect). We posited that adverse experiences occurring in the earlier years of development (ages 1 and/or 3), which are critical for establishing the building blocks of emotion processing, would exert the most substantial influence on mental health compared to adversity experienced at later periods in childhood. Concerning adversity-specific associations, we hypothesized that adversities related to caregiving and the home environment would have a greater influence during early childhood but that neighborhood-related adversities would have a greater influence during later childhood.

Methods

Participants

This study used data from the Future of Families and Child Wellbeing Study (FFCWS), a population-based birth-cohort sample of 4,898 children born in large cities with populations over 200,000 in the United States, with a 3:1 oversampling ratio for nonmarital births (Reichman et al., 2001). Children were followed throughout childhood and adolescence, and data were collected at multiple time points (child's birth, age 1, 3, 5, 9, and 15). In the current investigation, children who were reported to not live with mother at least half of the time during any waves ($n = 290$) and those with data collected at fewer than two time points across included waves in any predictors of interest (i.e., adversity measures) were excluded ($n = 398$), yielding a final sample of $N = 4{,}210$ (47% females, 49% Black, 25% Hispanic, 18% White, 8% other/multiracial; $22,500 median household income at child's birth) for analysis. The included sample ($N = 4{,}210$) does not differ from the full sample ($N = 4{,}898$) across key demographic variables (Supplemental Table 1).

Measures

Childhood adversity

Ten variables were examined as indicators of childhood adversity experienced across 4 waves within the child's first 9 years (collected when the child was 1, 3, 5, and 9 years of age). These variables were examined in previous investigations (Gard et al., 2021; Goetschius,

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Hein, McLanahan, et al., 2020; Goetschius, Hein, Mitchell, et al., 2020; Hardi et al., 2022, 2023; Hein et al., 2020; Peckins et al., 2020) and were selected to represent parenting, home, and neighborhood factors that contribute to youth mental health. Included predictors in the present study were measures of parenting, including (a) physical aggression, (b) psychological aggression, and (c) child neglect; parent or family factors: (d) intimate partner violence, (e) maternal depression, and (f) parental stress; indicators of instability and disadvantage within the home: (g) residential moves and (h) household material hardship; and aspects of the neighborhood: (i) low collective efficacy, and (j) community violence.

Parenting behaviors were measured using parent responses on the Parent–Child Conflict Tactics Scale (Straus et al., 1998) when the child was 3, 5, and 9 years of age. In accordance with recommendations for the scale (Straus et al., 1998), physical aggression, psychological aggression, and neglect were treated as separate constructs. Physical aggression was measured by five questions from the physical assault subscale capturing the frequency that the primary caregiver reported having engaged in behaviors such as "spanked [child] on the bottom with their bare hand" or "hit [child] on the bottom with something like a belt, hairbrush, a stick or some other hard object" in the past year. Psychological aggression was measured using five questions capturing the past year frequency that the caregiver reported having engaged in behaviors such as "shouted, yelled, or screamed at" or "swore or cursed at" the child. Neglect was measured by five questions capturing past year frequency that the caregiver reported having engaged in behaviors such as "had to leave their child home alone, even when they thought some adult should be with him/her" or "was not able to make sure their child got to a doctor or hospital when he/she needed it." Responses to each question were coded using a dichotomous variable (0 = *did not happen*, 1 = *has happened one or more times*) (Straus et al., 1998), and scores at each wave were summed to compute the developmental timing-specific measures of physical aggression (age 3 $\alpha = .49$; age 5 $\alpha = .52$; age 9 $\alpha = .62$), psychological aggression (age 3 $\alpha = .53$; age 5 $\alpha = .53$; age 9 $\alpha = .63$), and neglect (age 3 $\alpha = .51$; age 5 $\alpha = .50$; age 9 $\alpha = .58$).

Intimate partner violence (IPV) was measured using the mother's report on five questions about relationship quality such as "how often does father slap or kick you?" or "how often does father try to isolate you from friends/family?" when the child was 1, 3, 5, and 9 years of age, consistent with prior research (Hunt et al., 2017). Responses were coded as 0 = *never*, 1 = *sometimes*, 3 = *often*, where higher scores indicate a greater prevalence of IPV. In cases where the mother was no longer with the biological father of the child, the mother reported information about her current partner instead. IPV was computed at each developmental age by summing all items at each wave (age 1 $\alpha = .63$; age 3 $\alpha = .64$; age 5 $\alpha = .66$; age 9 $\alpha = .69$).

Two variables, maternal depression and parental stress, were examined as separate factors to represent parental mental health. Maternal depression was measured using the mother's self-report on the Composite International Diagnostic Interview – Short Form (CIDI-SF) (Kessler et al., 1998) when the child was 1, 3, 5, and 9 years of age. The CIDI-SF, consistent with the Diagnostic and Statistical Manual of Mental Disorders – Fourth Edition (Bell, 1994), included questions measuring whether the mother had feelings of depressed mood or anhedonia (loss of pleasure or interest in activities that they usually found enjoyable) in the past year that lasted 2 weeks or more. If so, they were asked more detailed questions about losing interest, tiredness, changes in weight, sleep, concentration, worthlessness, and any suicidal ideation. Maternal depression was dichotomously coded (1 = *yes*, 0 = *no*), and diagnostic criteria were met if the mother endorsed a depressed mood or anhedonia lasting at least half of the day nearly every day, along with two or more additional symptoms.

Parental stress was measured using four questions adapted from the Child Development Supplement of the Panel of Study of Income Dynamics (Hofferth et al., 1997) such as "I often feel tired, worn out, or exhausted from raising a family" and "I feel trapped by my responsibilities as a parent." These questions measured parenting stress triggered by changes in employment, income, or other factors and were taken from the Parent Stress Inventory (Abidin et al., 2006). The scale was administered when the child was 1, 3, 5, and 9 years of age, and the mother's responses to the questions were coded on a 4-point scale (0 = *strongly disagree*, 1 = *somewhat disagree*, 2 = *somewhat agree*, 3 = *strongly agree*), where higher scores indicate greater reported parental stress. Parental stress at each developmental age was computed by summing all items at each wave (age 1 $\alpha = .62$; age 3 $\alpha = .64$; age 5 $\alpha = .67$; age 9 $\alpha = .67$).

Frequent residential moves or household instability have been related to difficulties in adjustments for children (Adam, 2004; Leventhal & Newman, 2010). When the child was 1, 3, 5, and 9 years of age, mothers or primary caregivers were asked whether the family had moved since the last data collection, and if yes, how many times. Residential instability at each developmental age was measured by the frequency of moves between each wave and was coded as 0 if the family had not moved since the last wave.

Material hardship was measured by eight-item binary questions (1 = *yes*, 0 = *no*) when the child was 1, 3, 5, and 9 years of age using parent reports on whether the family experienced housing, utility, food, medical, and financial hardship in the past year. Items were selected from the 1996 Survey of Income and Program Participation; the 1997 and 1999 New York City Social Indicators Survey, and the 1999 Study of Work, Welfare, and Family Well-Being of Iowa families (Bauman, 1999; Mayer & Jencks, 1989). Scores were summed to represent material hardship at each age (age 1 $\alpha = .61$; age 3 $\alpha = .62$; age 5 $\alpha = .62$; age 9 $\alpha = .61$).

Low collective efficacy was measured using questions capturing cohesion and social control within the neighborhood when the child was 3, 5, and 9 years of age. Neighborhood cohesion was measured using four questions taken from the Social Cohesion and Trust Scale (Sampson et al., 1997; Sampson, 1997) such as "people around here are willing to help their neighbors" and "this is a close-knit neighborhood". Responses were coded as 3 = *strongly disagree*, 2 = *disagree*, 1 = *agree*, 0 = *strongly agree*, where higher scores indicate a greater low neighborhood cohesion. Neighborhood social control was measured using four questions taken from the Informal Social Control Scale (Sampson et al., 1997; Sampson, 1997) such as "likely neighbors intervene if children skipping school and hanging on street?" and "how likely neighbors intervene if fight broke out in front of the house?" Responses were coded as 3 = *very unlikely*, 2 = *not very unlikely*, 1 = *somewhat*, 0 = *very likely*, where higher scores indicate a greater low neighborhood informal social control. Low collective efficacy at each developmental age was measured by summing all items of neighborhood cohesion and social control at each wave (age 3 $\alpha = .84$; age 5 $\alpha = .87$; age 9 $\alpha = .87$).

Exposure to community violence was measured using three questions administered to the primary caregiver when the child was 3, 5, and 9 years of age about neighborhood conditions such as "in the past year, how often did you see person get hit, slapped, punched?" and "in the past year, how often did you see person attacked with weapon?" Responses were coded on a 4-point scale (0 = *never*, 1 = *once*, 2 = *2–3 times*, 3 = *4–10 times*, 4 = *more than*

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10 times). Exposure to community violence at each developmental age was computed by summing all items at each wave (age 3 α = .74; age 5 α = .76; age 9 α = .77).

Adolescent mental health outcomes

Internalizing and externalizing problems at age 15 were modeled separately as latent factors using item-level data of all available youth-report measures of internalizing and externalizing symptoms. Confirmatory factor analysis was conducted using MPlus v8.8 (Muthén & Muthén, 2017) with a WLSMV estimator.

Adolescent internalizing symptoms were measured using a latent factor score of youth responses to anxiety and depressive scales collected at age 15: six items from the Brief Symptom Inventory 18 (BSI-18) (Derogatis & Kathryn, 2000) and five items from the Center for Epidemiologic Studies Depression Scale (CES-D) (Radloff, 1977) (α = .84). The BSI-18 questions were coded on a 4-point scale (0 = *strongly disagree*, 1 = *somewhat disagree*, 2 = *somewhat agree*, 3 = *strongly agree*), where higher scores indicate greater self-reported anxiety. The CES-D questions were coded on a 4-point scale (0 = *strongly disagree*, 1 = *somewhat disagree*, 2 = *somewhat agree*, 3 = *strongly agree*), where higher scores indicate greater self-reported depressive symptoms. Each item was loaded onto a latent factor of overall internalizing symptoms. Model fit indices indicated good model fit (CFI = .970, TLI = .962, RMSEA = .073, SRMR = .034) (Hu & Bentler, 1999). Internalizing latent factor score was then extracted.

Adolescent externalizing behaviors were measured using a latent factor of youth responses to delinquency and substance use questions collected at age 15, 13 items from the Delinquency scale adopted from the National Longitudinal Study of Adolescent Health (Add Health) (Harris, 2013), and 5 items on youth substance use (α = .81). Youth-reported delinquency was measured by 13 questions, and responses were coded on a 3-point scale (0 = *never*, 1 = *sometimes*, 2 = *often*), where higher scores indicate greater delinquent behavior. Substance use was measured using five binary questions (0 = *no*, 1 = *yes*) capturing alcohol use (more than two drinks without parents), tobacco, and other illicit substances (marijuana, illegal or prescription drugs). Each question was loaded onto a latent factor of overall externalizing behavior. Model fit indices indicated good model fit (CFI = .961, TLI = .955, RMSEA = .038, SRMR = .091) (Hu & Bentler, 1999). Externalizing latent factor score was then extracted.

Covariates

The following covariates were included in subsequent sensitivity analysis models: ethnoracial identity, sex at birth, parental marital status, birth city, child temperament (shyness, emotionality), and pubertal development. Youth self-reported ethnoracial identity (Black/African American only, non-Hispanic; white only, non-Hispanic; Hispanic/Latino; multiracial, non-Hispanic; other only, non-Hispanic) at age 15. Dummy-coded variables of these ethnoracial identity categories were included to account for experiences of race-related adversity such as structural racism that were not measured in the present investigation and could influence mental health (Bailey et al., 2017) (multiracial and other groups were combined in analysis). Sex was parent-reported at the child's birth (0 = female, 1 = male) and was included to account for any sex differences in adolescent mental health (Rutter et al., 2003). Parental marital status was parent-reported at child age 1 (0 = unmarried, 1 = married) and was included to account for the FFCWS sampling strategy (Reichman et al., 2001). The birth city was measured by dummy-coded variables representing 20 study sites (see Supplemental Table 1 for a full list of study locations) and was included to account for geographical differences in sampling. Child temperament was measured at age 1 using the sum scores of parent responses to questions about the child's shyness and emotionality (1 = "*not characteristic or typical of your child*" and 5 = "*very characteristic or typical of your child*") and was included to account for early indicators of psychopathology (Nigg, 2006). Questions on temperament were adapted from the Emotionality, Activity, and Sociability Temperament Survey of Children (Buss & Plomin, 2013) and were only administered at age 1 in the present sample. Shyness was defined as the tendency for the child to be socially inhibited (e.g., "tends to be shy"), and higher scores represent a greater level of shyness. Emotionality was defined as the tendency for the child to become easily distressed (e.g., "reacts intensely when upset"), and higher scores represent a greater level of emotionality. Pubertal development was measured using parent-reported responses on the Pubertal Development Scale (Petersen et al., 1988) at age 9, and was included to account for differences in pubertal development. These questions measured the extent to which specific physical changes (e.g., facial hair, growth spurt, breast development) have occurred (1 = *no*, 2 = *yes, barely*, 3 = *yes, definitely*, 4 = *development complete*). Scores for females and males were computed separately as there were questions that pertained only to females (e.g., menarche).

Analytic strategy

Structured Life-Course Modeling Approach (SLCMA)

This study used the Structured Life-Course Modeling Approach (SLCMA) (Smith et al., 2016) to test the cumulative and specificity hypotheses. SLCMA was implemented using R v4.2.1 and is a variable selection method that selects predictors, among a set of competing predictors, that would best explain the variation in a measured outcome. SLCMA uses the least angle regression variable selection technique, a type of least absolute shrinkage and selection operator regression, which employs a regularization penalty to regressors with low predictability, and is an approach that provides greater statistical power that is robust to multicollinearity (Efron et al., 2004; Smith et al., 2016, 2022). This method is particularly suited to compare theoretical models (e.g., cumulative vs. sensitive periods) as it performs a data-driven model selection on a set of competing models that were identified a priori. SLCMA has been applied to study the effects of longitudinal exposure to adversity on psychopathology (Dunn et al., 2018), DNA methylation (Dunn et al., 2019), and cognitive performance (Nweze et al., 2023) among others.

In this study, two models of SLCMA were implemented (Table 1). The first tested the cumulative and specificity hypotheses by developmental *timing* by modeling adversity scores at each wave (ages 1, 3, 5, 9) and their cumulative score (sum of each childhood adversity across time) as separate predictors of internalizing and externalizing problems at age 15. These models were tested separately for each type of adversity; thus 10 models were examined for each behavioral outcome (internalizing and externalizing symptoms). Physical aggression, psychological aggression, neglect, residential moves, low collective efficacy, and community violence were not measured at age 1; thus, only data at ages 3, 5, and 9, as well as their cumulative scores, were included as predictors in those timing-specific SLCMA models. To account for multiple comparisons, statistical significance in the post-inference model selection was Bonferroni

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Table 1. Types of SLCMA models tested in the present investigation

SLCMA model	Youth outcomes	Adversity	Predictors	Covariates
Timing-specific	Internalizing/externalizing symptoms	Physical aggression	Age 3; age 5; age 9; cumulative score across ages 3, 5, 9	Sex at birth, ethnoracial identity, birth city, parental marital status, shyness, emotionality, pubertal development
	Internalizing/externalizing symptoms	Psychological aggression	Age 3; age 5; age 9; cumulative score across ages 3, 5, 9	
	Internalizing/externalizing symptoms	Neglect	Age 3; age 5; age 9; cumulative score across ages 3, 5, 9	
	Internalizing/externalizing symptoms	Intimate partner violence	Age 1; age 3; age 5; age 9; cumulative score across ages 1, 3, 5, 9	
	Internalizing/externalizing symptoms	Maternal depression	Age 1; age 3; age 5; age 9; cumulative score across ages 1, 3, 5, 9	
	Internalizing/externalizing symptoms	Parental stress	Age 1; age 3; age 5; age 9; cumulative score across ages 1, 3, 5, 9	
	Internalizing/externalizing symptoms	Residential moves	Age 1; age 3; age 5; age 9; cumulative score across ages 1, 3, 5, 9	
	Internalizing/externalizing symptoms	Material hardship	Age 1; age 3; age 5; age 9; cumulative score across ages 1, 3, 5, 9	
	Internalizing/externalizing symptoms	Low collective efficacy	Age 3; age 5; age 9; cumulative score across ages 3, 5, 9	
	Internalizing/externalizing symptoms	Community violence	Age 3; age 5; age 9; cumulative score across ages 3, 5, 9	
Type-specific	Internalizing/externalizing symptoms	All	Average scores across all time points of physical aggression; psychological aggression; neglect; intimate partner violence; maternal depression; parental stress; residential moves; material hardship; low collective efficacy; community violence; cumulative score of all adversities examined	

Note. No data were collected at age 1 for physical aggression, psychological aggression, neglect, residential moves, low collective efficacy, and community violence.

corrected for ten models. The second SLCMA model tested for cumulative and specificity hypotheses by *types* by modeling all adversity scores across childhood (average scores of each childhood adversity across ages 1, 3, 5, 9) and their cumulative score (sum of all examined childhood adversity) as separate predictors of internalizing and externalizing problems (two separate models) at age 15. All models were adjusted for covariates to control for potential confounding factors.

Steps outlined in previous publications were followed to determine the best-fitting hypothesis (Dunn et al., 2018; Smith et al., 2016, 2022). First, bivariate correlations of all variables were examined to ensure that there were no highly correlated/collinear variables ($r > .80$) that would limit model selection. Missing data were handled following recommendations on missing data and multiple imputations (Woods et al., 2021) using the R *MICE* package (Buuren & Groothuis-Oudshoorn, 2011) (20 imputations with 20 iterations). Then, in the first stage of SLCMA, each resulting elbow plot was examined to select the variables that best explain the outcome in the SLCMA model. In the second stage, selective inference methods (Efron et al., 2004) were implemented to determine pooled estimates of effect sizes and confidence intervals for the variables that were selected in the first stage. Selective inference outperforms other inference methods in minimizing bias in effect sizes, confidence intervals, and *p*-value estimates by adjusting for family-wise error rates based on the number of variables selected (Smith et al., 2022; Zhu et al., 2021).

Results

Models testing timing-specific effects of childhood adversity

Levels and prevalence of each adversity at each developmental age are shown in Table 2.

At each developmental time point, there were weak to moderate correlations among each childhood adversity exposure ($rs = .19$ to .59) (Supplemental Table 2). Parental stress had the highest correlations and stability over time (average $r = .50$), while neglect had the lowest correlations across the different ages (average $r = .21$). Material hardship was the most correlated with other adversities (average $r = .22$), while parental stress was the least associated with all other adversities (average $r = .13$). In terms of associations with internalizing and externalizing symptoms, psychological and physically aggressive parenting, number of residential moves, material hardship, and low collective efficacy were all consistently related to externalizing symptoms (and sometimes related to internalizing symptoms), whereas neglect, maternal depression, intimate partner violence, and low collective efficacy were relatively equally related to both internalizing and externalizing symptoms. Zero-order correlations of all predictors and outcomes at each time point are shown in Supplemental Table 2.

Across all ten timing-specific SLCMA models predicting internalizing symptoms, cumulative was selected 9 times (53%) followed by age 9 (6 times; 35%), age 5 (1 time; 6%), and age 3 (1 time; 6%) in the first stage of model selection (Figure 2). These findings suggest that a cumulative score across all the

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Table 2. Mean and standard deviation of adversity at each time point

Adversity	Age 1 *M* (*SD*)	Age 3 *M* (*SD*)	Age 5 *M* (*SD*)	Age 9 *M* (*SD*)
Physical aggression	*NA*	1.71 (1.15)	1.69 (1.22)	1.73 (1.38)
Psychological aggression	*NA*	1.89 (0.96)	2.08 (1.01)	2.29 (1.25)
Neglect	*NA*	0.14 (0.48)	0.14 (0.45)	0.34 (0.73)
Intimate partner violence	0.37 (0.97)	0.29 (0.87)	0.27 (0.82)	0.19 (0.72)
Maternal depression	0.15 (0.36)	0.20 (0.40)	0.16 (0.37)	0.16 (0.37)
Parental stress	4.69 (2.68)	4.97 (2.67)	4.70 (2.71)	4.12 (2.74)
Residential moves	0.61 (0.85)	0.69 (0.95)	0.70 (0.91)	1.06 (1.27)
Material hardship	0.87 (1.23)	0.87 (1.26)	0.90 (1.27)	1.10 (1.36)
Low collective efficacy	*NA*	9.61 (6.37)	7.84 (5.91)	7.69 (5.92)
Community violence	*NA*	0.95 (1.75)	0.88 (1.74)	0.63 (1.48)

Note. No data were collected at age 1 for physical aggression, psychological aggression, neglect, residential moves, low collective efficacy, and community violence.

developmental time points examined explained the most variance in youth internalizing symptoms for almost all SLCMA adversity models, followed by age 9 exposure, and age 5 or 3.

Effect sizes and confidence intervals were then estimated in the second stage of SLCMA based on the number of variables indicated by each elbow plot (Figure 3a). Results from post-selection inference suggested that the cumulative score significantly predicted internalizing symptoms in three adversity models: maternal depression, material hardship, and low collective efficacy (Table 3). Additionally, parental stress at age 9 and neglect at age 3 also predicted internalizing symptoms (Table 3). These findings suggest that prolonged exposures to maternal depression, material hardship, and low collective efficacy most strongly predicted internalizing symptoms. Additionally, exposure to parental stress at age 9 and early exposure to neglect (at age 3) most strongly predicted internalizing symptoms relative to any other age.

Across the 10 timing-specific SLCMA models examined to predict externalizing behaviors, cumulative was selected 10 times (56%) followed by age 9 (6 times; 33%) and age 5 (2 times; 11%) (Figure 2). Similar to the SLCMA models predicting internalizing symptoms, these findings suggest that a cumulative score across all the developmental time points best explained youth externalizing behaviors for all SLCMA adversity models examined, followed by age 9 exposures and age 5. After determining the number of selected variables using respective elbow plots (Figure 3b), post-selection inference showed that cumulative effect best predicted externalizing behaviors in six timing-specific SLCMA models: physical aggression, psychological aggression, neglect, parental stress, residential moves, material hardship, and exposure to community violence (Table 4). Additionally, maternal depression at age 9 and intimate partner violence at age 5 also significantly predicted externalizing behaviors (Table 4). These findings suggest that the prolonged childhood exposures to most adversities examined in the present study best predicted youth externalizing symptoms. However, externalizing symptoms were also predicted by maternal depression at age 9 and intimate partner violence at age 5 relative to all other ages examined.

Models testing type-specific effects of childhood adversity across childhood

There were weak to moderate correlations among childhood adversity exposures across childhood ($rs = .05$ to $.64$) (Table 5). Physical and psychological aggression were most correlated at $r = .64$, while intimate partner violence was least correlated with physical aggression at $r = .05$.

In the type-specific SLCMA model predicting internalizing symptoms, three variables – cumulative, parental stress, and low collective efficacy – were selected in the first stage of SLCMA (Figure 3c), whereby the cumulative score of all adversities across childhood explained the greatest proportion of variance in the model ($r^2 = 1.18\%$) followed by parental stress ($r^2 = 0.29\%$) and low collective efficacy ($r^2 = 0.66\%$) (Table 2). Post-selection inference found that the cumulative effect, parental stress, and low collective efficacy significantly predicted adolescent internalizing symptoms (Table 6). These findings suggest that a cumulative score that captures exposure to all the adversities across ages 1–9 explained the most variance in youth internalizing symptoms. Additionally, parental stress and low collective efficacy explained additional variance in youth internalizing symptoms.

In the type-specific SLCMA model predicting externalizing behaviors, three variables – cumulative ($r^2 = 1.16\%$), psychological aggression ($r^2 = 2.48\%$), and material hardship ($r^2 = 0.50\%$) were selected in the first stage of SLCMA (Figure 3c). Post-selection inference found that the cumulative effect of all types of adversity as well as psychological aggression significantly predicted youth externalizing behaviors (Table 6). These findings suggest that a cumulative score that captures exposure to all the adversities across ages 1–9 explained the most variance in youth externalizing symptoms. Additionally, psychological aggression explained additional variance in externalizing symptoms.

Discussion

Using a structured theory-informed and data-driven approach, this study compared different life-course hypotheses (cumulative, timing specificity), as well as the accumulation and unique contributions of different types of adverse experiences (cumulative, adversity-specificity) on adolescent internalizing and externalizing symptoms. We examined these questions in a prospective, diverse, population-based sample of youth from whom data were collected at multiple time points spanning 15 years. Consistent with our hypotheses, results suggested that internalizing and externalizing symptoms in youth were best explained by the cumulative effect of adversity across multiple types of adversity and over time. However, our results also demonstrated some adversity- and timing-specific effects through the distinct contributions of certain types of adversity at certain developmental periods. In particular, in addition to cumulative effects, most adversities experienced at age 9 better explained youth internalizing and externalizing symptoms than any other time points (age 1, 3, or 5). There were a few notable exceptions. The association between neglectful parenting and youth internalizing symptoms was better explained by exposures earlier in development (age 3), and the association between intimate partner violence and youth externalizing behaviors was better explained by experience at age 5. Moreover, in addition to cumulative effects across the types of adversities, parental stress and low collective efficacy predicted internalizing symptoms, whereas parental psychological aggression predicted externalizing behaviors in youth. Importantly, our analyses adjusted for pubertal development and early child

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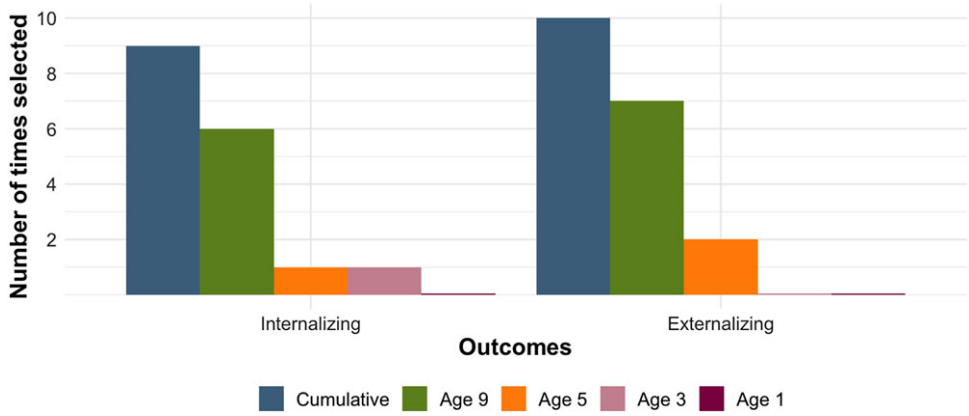

Figure 2. Summary of model selection across all SLCMA models. Cumulative was selected 9 times (53%) in predicting youth internalizing symptoms and 10 times (56%) for externalizing symptoms. Age 9 exposure was selected six times (35%) for internalizing symptoms and six times (33%) for externalizing symptoms. Age 5 exposure was selected one time (6%) for internalizing symptoms and two times (11%) for externalizing symptoms. Finally, age 3 was selected one time (6%) for internalizing symptoms. Selected variables were determined based on the location of the elbow plots in separate timing-specific SLCMA models during the first stage of SLCMA (Figure 3).

temperament risk. Thus, the contributions of these separate adversities, as well as their cumulative effects on youth internalizing and externalizing problems, were over and above early childhood risk for psychopathology or differences in pubertal development. Collectively, these results suggest that, although childhood adversity is most likely to contribute prospectively to youth internalizing and externalizing symptoms in a cumulative fashion, there are specific developmental periods and types of adversity that have unique effects above the broad cumulative impact of adversity.

These findings support the cumulative risk model as particularly powerful in explaining the associations between childhood adversity and youth internalizing and externalizing symptoms. These findings align with other research suggesting that the number of different types of childhood adversity as well as across multiple developmental periods may be most detrimental to children's mental health compared to the distinct contributions of any specific type of adversity at any developmental point (Evans et al., 2013). The cumulative model of adversity converges with the principle of the allostatic load model (McEwen, 1998), which describes the process through which stressful experiences can increase the "wear and tear" of the body, to the detriment of health (Evans, 2003; McEwen & Stellar, 1993). The term "allostasis" refers to the neurobiological response to stressors in order to regain homeostasis or stability. When stress is experienced acutely, this response is adaptive and necessary for survival; however, repeated efforts to maintain homeostasis in response to the cumulative burden of stressful life events can lead to the detrimental effects of "allostatic load". These cumulative findings can also reflect a model of adversity in which the risks for maladaptive behaviors increase with more exposures and longer durations of adversity but require a specific threshold to be met for maladaptive behaviors to manifest. Consequently, the specific type or timing of adversity may be less critical than the total number of adversities or the duration that they are experienced – a key finding to consider in models and studies of psychopathology, which often attempt to isolate a singular adversity at a particular time point and relate it to a singular outcome. Though this result may seem obvious in some ways – that the more adversity a child experiences, the greater the risk for psychopathology – it is important to consider for translational efforts. If our interventions focus only on one factor, we may have less success at promoting positive health for youth who are experiencing high cumulative risk. Thus, interventions that address risks across multiple domains (e.g., multisystemic therapy; Henggeler, 1999) or those that can be tailored to specific needs and risk profiles of youth or families (e.g., Gill & Shaw, 2020) may be more effective in settings with high exposure to adversity.

Though the SLCMA models examined in the present study consistently favored cumulative effects, there were also timing-specific and type-specific associations that warrant attention. In particular, adverse experiences later in childhood (age 9) were more likely to be selected in the first stage of SLCMA to explain both youth internalizing and externalizing symptoms compared to adversities earlier in childhood (ages 1, 3, or 5). This pattern was evident across most factors examined (parenting, home, neighborhood), highlighting the importance of later childhood experiences for youth mental health. Age 9 is also a time when numerous social and biological changes are beginning. Emerging evidence on pubertal stress recalibration (Gunnar et al., 2019) indicates that this period leading up to puberty may hold greater significance for the development of stress response mechanisms than previously believed. Thus, the present findings suggest that adversity experienced during this developmental period is particularly critical for youth mental health. Additionally, it is also possible that developmental adaptation may be more difficult to accomplish when children have already established patterns and routines in their existing environment (Chetty et al., 2016). Thus, environmental changes due to adverse events occurring during later childhood could present more challenges than currently understood. More research is needed to test these hypotheses and examine the differential mechanisms underlying adversity later in development.

The importance of experiences later in childhood and the lack of specificity in the developmental timing across contexts (e.g., home vs. neighborhood) in the present study are contrary to other studies that have established the significance of early childhood experiences. However, our pattern of results may diverge from previous studies for several reasons. First, the present sample contains a large representation of low-income families, which contrasts with some of the previous literature that were focused on more advantaged communities or clinical samples. It could be that timing effects are modulated by the total overall exposure to

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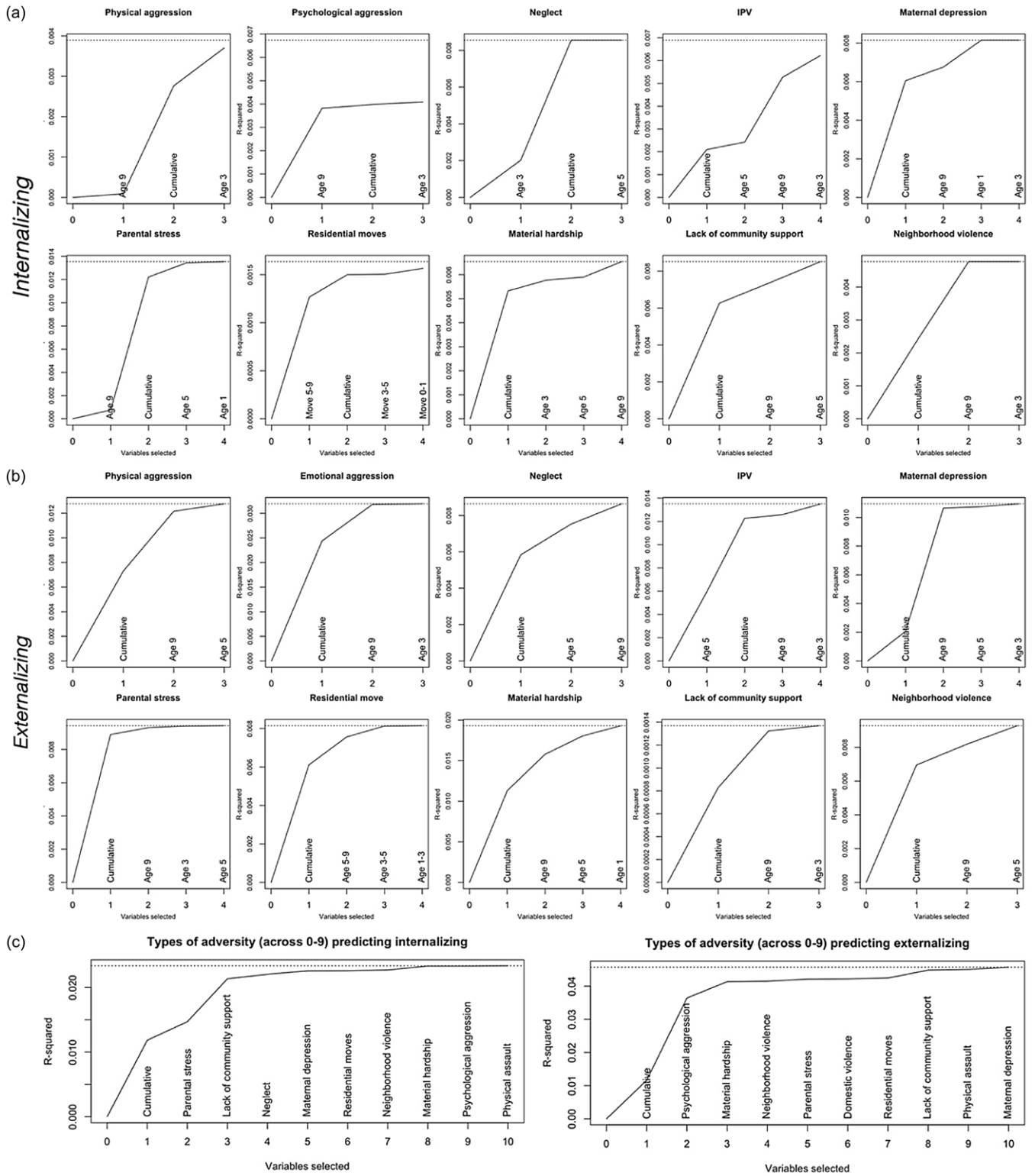

Figure 3. Elbow plots of timing-specific and type-specific SLCMA models testing the associations of the childhood adversity and adolescent mental health problems. A full list of variables included in each SLCMA model is outlined in Table 1. (a) Elbow plots of timing-specific SLCMA models predicting youth internalizing symptoms. Of all 10 models, cumulative was selected in nine timing-specific SLCMA models: physical aggression, neglect, intimate partner violence, maternal depression, parental stress, residential moves, material hardship, lack of community support, and neighborhood violence. Age 9 was selected in six timing-specific SLCMA models: physical aggression, psychological aggression, intimate partner violence, parental stress, residential moves, and neighborhood violence. Age 5 was selected for one timing-specific SLCMA model: intimate partner violence. Age 3 was selected for one timing-specific SLCMA model: neglect. (b) Elbow plots of timing-specific SLCMA models predicting externalizing symptoms. Of all 10 timing-specific SLCMA models, cumulative was selected in all models. Age 9 was selected in six timing-specific SLCMA models: physical aggression, psychological aggression, maternal depression, residential moves, material hardship, and lack of community support. Age 5 was selected in two timing-specific SLCMA models: neglect and intimate partner violence. (c) Elbow plots of the type-specific SLCMA models predicting internalizing (left) and externalizing (right) symptoms. Cumulative, parental stress, and lack of community support were selected in the first stage of the type-specific SLCMA model in predicting internalizing symptoms. Post-selection inference showed that all three variables significantly predicted internalizing symptoms. Cumulative, psychological aggression, and material hardship were selected in the first stage of type-specific SLCMA models. Post-selection inference showed that both cumulative and psychological aggression significantly predicted externalizing behaviors.

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Table 3. Models testing cumulative versus specificity by developmental timing to predict internalizing symptoms

		Covariates-adjusted models		
Adversity model	Variable(s) selected	r^2 change (%)	Coefficient [CI]	P_{bonf}
Physical aggression	Age 9	0.009	0.021 [−0.079, 0.106]	.100
	Cumulative	0.267	0.010 [−0.040, 0.048]	.100
Psychological aggression	Age 9	0.382	0.053 [0.028, 0.075]	.002
Neglect	Age 3	0.202	0.108 [0.031, 0.182]	.037
	Cumulative	0.653	0.034 [0.000, 0.063]	.241
Intimate partner violence	Cumulative	0.210	0.006 [−0.053, 0.035]	.100
	Age 5	0.032	0.045 [−0.049, 0.143]	.100
	Age 9	0.284	0.047 [−0.044, 0.132]	.100
Maternal depression	Cumulative	0.605	0.074 [0.048, 0.101]	<.001
Parental stress	Age 9	0.076	0.021 [0.003, 0.036]	.117
	Cumulative	1.146	0.006 [0.000, 0.011]	.188
Residential moves	Age 5–9	0.127	0.023 [−0.021, 0.198]	.888
	Cumulative	0.023	0.003 [−0.115, 0.018]	1.00
Material hardship	Cumulative	0.533	0.019 [0.011, 0.026]	<.001
Low collective efficacy	Cumulative	0.627	0.006 [0.004, 0.008]	<.001
Community violence	Cumulative	0.243	0.012 [0.000, 0.025]	.225
	Age 9	0.234	0.016 [−0.027, 0.042]	1.00

Note. Coefficient and *p*-values were extracted only for variables that were selected in the first stage of SLCMA (determined by r^2 values in corresponding elbow plots). The following predictors were included in each adversity model in the first-stage model specification: adversity at age 1 (for intimate partner violence, maternal depression, parental stress, residential moves, material hardship), age 3, age 5, age 9, cumulative score across all ages, and all covariates (sex at birth, ethnoracial identity, birth city, parental marital status, shyness, emotionality, pubertal development). Alpha values shown were Bonferroni corrected for 10 comparisons.

Table 4. Models testing cumulative versus specificity by developmental timing to predict externalizing symptoms

		Covariates-adjusted models		
Adversity model	Variable(s) selected	r^2 change (%)	Coefficient [CI]	P_{bonf}
Physical aggression	Cumulative	0.730	0.021 [0.007, 0.044]	.035
	Age 9	0.486	0.022 [−0.035, 0.052]	1.00
Psychological aggression	Cumulative	2.439	0.043 [0.028, 0.063]	<.001
	Age 9	0.743	0.028 [−0.015, 0.060]	.894
Neglect	Cumulative	0.583	0.047 [0.020, 0.100]	.008
	Age 5	0.170	0.045 [−0.158, 0.106]	1.00
Intimate partner violence	Age 5	0.595	0.070 [0.030, 0.114]	.006
	Cumulative	0.632	0.015 [−0.005, 0.028]	.661
Maternal depression	Cumulative	0.207	0.047 [0.015, 0.078]	.160
	Age 9	0.858	0.107 [0.010, 0.190]	.028
Parental stress	Cumulative	0.889	0.009 [0.006, 0.012]	<.001
Residential moves	Cumulative	0.611	0.021 [0.008, 0.053]	.019
	Age 5–9	0.146	0.014 [−0.077, 0.040]	1.00
Material hardship	Cumulative	1.130	0.021 [0.012, 0.036]	<.001
	Age 9	0.448	0.026 [−0.030, 0.051]	1.00
Low collective efficacy	Cumulative	0.083	0.002 [−0.003, 0.010]	1.00
	Age 9	0.049	0.002 [−0.022, 0.009]	1.00
Community violence	Cumulative	0.696	0.020 [0.013, 0.026]	<.001

Note. Coefficient and *p*-values were extracted only for variables that were selected in the first stage of SLCMA (determined by r^2 values in corresponding elbow plots). The following predictors were included in each adversity model in the first-stage model specification: adversity at age 1 (for intimate partner violence, maternal depression, parental stress, residential moves, material hardship), age 3, age 5, age 9, cumulative score across all ages, and all covariates (sex at birth, ethnoracial identity, birth city, parental marital status, shyness, emotionality, pubertal development). Alpha values shown were Bonferroni corrected for 10 comparisons.

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Table 5. Zero-order correlations of adversity variables (average across 1, 3, 5, 9 years old)

Variable	*M*	*SD*	1	2	3	4	5	6	7	8	9
1. Physical aggression	1.72	1.03									
2. Psychological aggression	2.09	0.87	.64**								
3. Neglect	0.21	0.41	.22**	.27**							
4. Intimate partner violence	0.29	0.63	.05*	.10**	.14**						
5. Maternal depression	0.17	0.26	.13**	.22**	.19**	.19**					
6. Parental stress	4.65	2.18	.18**	.23**	.24**	.15**	.26**				
7. Residential moves	0.76	0.68	.12**	.14**	.09**	.06**	.20**	.08**			
8. Material hardship	0.92	0.98	.23**	.23**	.21**	.18**	.37**	.20**	.38**		
9. Low collective efficacy	4.25	2.47	.13**	.16**	.14**	.14**	.14**	.18**	.13**	.17**	
10. Community violence	0.82	1.31	.14**	.18**	.13**	.07**	.15**	.13**	.08**	.17**	.23**

Note. M and SD are used to represent mean and standard deviation, respectively. Zero-order correlations of adversity variables at each developmental wave are in Supplemental Table 2. Internal consistency indices for each measure are in Supplemental Table 3. * indicates $p < .05$. ** indicates $p < .01$.

Table 6. Models testing cumulative versus specificity by adversity type to predict youth internalizing and externalizing

	Covariates-adjusted models		
Outcome: Internalizing Variable(s) selected	r^2 change (%)	Coefficient [CI]	*p*
Cumulative	1.18	0.025 [0.015, 0.034]	<.001
Parental stress	0.29	0.023 [0.007, 0.037]	.003
Low collective efficacy	0.66	0.018 [0.002, 0.030]	.015
	Covariates-adjusted models		
Outcome: Externalizing variable(s) selected	r^2 change (%)	Coefficient [CI]	*p*
Cumulative	1.16	0.024 [0.014, 0.036]	<.001
Psychological aggression	2.48	0.097 [0.065, 0.129]	<.001
Material hardship	0.50	0.038 [-0.012, 0.069]	.061

Note. Coefficient and *p*-values were extracted only for variables that were selected in the first stage of SLCMA (determined by r^2 values in corresponding elbow plots). In the first-stage model specification for testing cumulative versus specificity by adversity type, average scores across all time points of all adversity were included as predictors: physical aggression, psychological aggression, neglect, intimate partner violence, maternal depression, parental stress, residential moves, material hardship, low collective efficacy, community violence, cumulative score across all adversity, and all covariates (sex at birth, ethnoracial identity, birth city, parental marital status, shyness, emotionality, pubertal development).

adversity or the relative disadvantage of the context surrounding the exposures. Second, FFCWS data collection at age 9 coincides with the Great Recession (Garfinkel et al., 2016), suggesting that this period of instability may have contributed additional stressors beyond the measures we examined, with downstream implications for children as they mature into adolescence (Schneider et al., 2015). More research is needed to examine the influence of acute economic downturns and other stressors (e.g., the global pandemic) that produce a widespread adverse effect on families. Future studies are also needed to replicate these findings in other diverse samples to test the generalizability of the present results.

There were two notable exceptions where experiences earlier in childhood (ages 3 or 5) were favored over the cumulative effect or age 9 exposures. First, exposure to neglectful parenting at age 3 was more likely to predict internalizing symptoms than any other developmental periods or cumulative effect. These findings echo other studies on early sensitive periods associated with previously institutionalized children (e.g., McLaughlin et al., 2015) who experienced severe neglect. It is possible that neglect more directly represents deprivation of an expected environment compared to other types of adversity (e.g., violence or neighborhood factors) (Brett et al., 2015). Consequently, neglect can lead to biological changes and subsequently internalizing symptoms in more children. Second, intimate partner violence at age 5 better predicted externalizing symptoms than cumulative effect or violence during any other developmental periods. This significance of intimate partner violence is consistent with prior research (Moss et al., 2023) showing that children begin to develop a more nuanced understanding of conflict during this developmental period. Thus, witnessing violence at home may be more dysregulating for the child at age 5 than earlier (when they have limited contextual understanding) or later (when they have better emotion regulation skills or have developed other coping skills from school contexts).

When examining the relative contributions of specific adversities across childhood, in addition to cumulative effects, parental stress and low collective efficacy predicted internalizing symptoms above other types of adverse experiences. This is notable, particularly in light of the well-established evidence supporting the family stress model (Conger et al., 2002; Masarik & Conger, 2017). Within this framework, parents' psychological distress is recognized as a critical factor in explaining how socioeconomic disadvantage can result in children's maladaptive

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behaviors through increased caregivers' emotional distress, strained parental interpersonal relationships, and disrupted parenting practices (Masarik & Conger, 2017). A supportive neighborhood can buffer against within-home stress (Browning et al., 2016; Silk et al., 2004). Therefore, the present findings demonstrate that the presence of both parental stress and low collective efficacy can have a significant impact on youth internalizing symptoms over and above other adverse experiences.

The meaningful role of parent psychological aggression across childhood, in addition to the cumulative risk, in predicting adolescent externalizing behaviors aligns with an extensive body of research investigating risk factors for externalizing outcomes (Hinshaw & Lee, 2003). These findings could indicate genetic risks through personality traits associated with externalizing behaviors (e.g., impulsivity, interpersonal aggression) that are passed down from parents to children (Gard et al., 2019). Alternatively, harsh parenting could also predict later externalizing behaviors through environmental mechanisms (Burt et al., 2021). These associations likely reflect influences of both genetic and environmental processes and their interactions. For example, according to multiple developmental models of youth externalizing, early childhood difficult temperament interacts with parent psychological aggression via disrupted empathy development (Eisenberg, 2005; Kochanska, 1997) and the modeling of aggressive interactions (Granic & Patterson, 2006), leading to a cycle of coercive interactions over time that spill out into other settings (e.g., schools, peers; Patterson et al., 1989). This work highlights the importance of preventing harsh parenting as a key public health measure to reduce the risk of later externalizing behaviors (Dodge et al., 2009; Dodge, 2001).

There are several limitations to our study. First, we selected risk factors that the current literature indicates as particularly salient for both internalizing and externalizing symptoms. Although the present study examined 10 types of childhood adversity across 4 waves in the first 9 years of childhood, there are other risk factors that could have been included, but not measured in the present sample. Moreover, given that adverse experiences are often interconnected and overlapping, there could be interactions between these experiences that have important implications. Second, given the large-scale survey approach of FFCWS, most of the measures capturing adversities were brief and potentially less robust than other studies (e.g., many were represented only by a few items, leading to lower internal consistencies). This brief measurement may introduce errors and fail to capture each construct deeply. Moreover, although the maternal report can capture an important perspective on adversity exposure, especially when collected prospectively during childhood (Tajima et al., 2004), the childhood adversities examined here were measured only using parental responses, which may capture only one slice of the context in which a child is growing up in (De Los Reyes et al., 2019). Third, data collection in this cohort was focused primarily on childhood ages (i.e., 1–9) with outcomes measured in adolescence (age 15), and we had no data during the critical developmental period between 9 and 15 or the prenatal period. Furthermore, many timing-specific measures were examined using data from only one time point (e.g., no data were collected for neglect at age 1). More studies are thus needed to examine these questions at additional time points, including the in utero period, infancy, and pre-adolescence. Fourth, SLCMA presently is unable to accommodate time-varying covariates. Thus, we are unable to account for changes in factors that could vary across time. Fifth, although the current models provide a statistical method to tease apart developmental timing differences in adversity exposure, it is important to acknowledge that experiences during later development inherently include temporal information from earlier development. More research is thus needed to further distinguish the influence of exposures at age 9 from chronic experiences or cascading effects. Lastly, although this study utilized prospective, longitudinal data spanning 15 years, as with all longitudinal studies, causal relationships between these experiences and behavioral outcomes cannot be determined (Rutter, 2003). There are also numerous other biological and contextual factors, such as the interactions of genetic and environmental influences, that are critical in explaining the antecedents of psychopathology (Manuck & McCaffery, 2014).

Conclusion

In a well-sampled cohort of youth at an increased risk for exposure to adversity, this investigation provides empirical evidence supporting the longitudinal associations between childhood adversity and youth mental health outcomes, using a theory-informed and data-driven method. These findings underscore the profound impact of childhood adversity and carry direct policy implications.

First, considering the evidence that cumulative effects were the strongest in predicting adolescent mental health, it is unsurprising that interventions targeting only a single form of adversity might yield less consistent or substantial mental health improvements, especially for individuals living in poverty. For instance, Shelleby and Shaw (2014) found that while some parenting interventions led to significant improvements in child outcomes, children from lower socioeconomic backgrounds benefited less. This may be due to the exposure to a myriad of risks and increased barriers that many disadvantaged families face when trying to access care (Staudt, 2007). Therefore, interventions targeting multiple exposures or personalizing their approach based on the family's unique constellation of risks may prove to be more effective (Shaw et al., 2000; Shaw et al. 2006). Moreover, policies and interventions that target systemic structures (e.g., profound wealth inequality, concentrated poverty, structural racism) can be a more efficient and effective way to allocate resources (Braveman & Gottlieb, 2014), as they prevent youth from encountering an array of risks across an extended period of development.

Second, while research on early childhood has provided valuable insights into the etiology of mental disorders, more efforts are needed to understand the mechanisms of prepubertal experiences identified as key predictors for mental health in this sample. Evidence supporting a potentially sensitive period during late childhood underscores the importance of understanding the biological shifts leading up to puberty and how these changes could create opportunities for prevention and intervention.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/S0954579424001512.

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