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# Adversity exposure and obesogenic food consumption in young children: The transgenerational role of emotion dysregulation

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## Summary

**Background:** Childhood adversity is linked with unhealthy eating behaviours and obesity, but the mechanisms underlying this association are unclear, specifically the transgenerational behavioural precursors that develop in early childhood.

**Objective:** To determine whether adversity predicts change in obesogenic food consumption through child emotion dysregulation, and whether caregiver emotion dysregulation modifies this association.

**Methods:** Participants included 190 low-income caregiver-child dyads (mean child age = 4.31 years [SD = 0.85]). Cumulative lifetime adversity exposure was assessed via study-created measure. The Difficulties with Emotion Regulation Scale and Emotion Regulation Checklist assessed caregiver and child emotion dysregulation, respectively. Children's obesogenic food consumption was assessed at two time points 6 months apart using a caregiver-report measure: the Children's Eating Habits Questionnaire. Moderated mediation models were tested using autoregressive structural equation modelling.

**Results:** Cumulative lifetime adversity was associated with child emotion dysregulation only when caregiver emotion dysregulation was high. Child emotion dysregulation in turn was associated with greater obesogenic food consumption 6 months later.

**Conclusions:** Among young children with caregivers high in emotion dysregulation, cumulative lifetime adversity was linked to an increase in obesogenic food consumption through child emotion dysregulation.

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AUTHOR CONTRIBUTIONS

Yo Jackson conceptualized and designed the parent study. Lindsay Huffhines conceived study hypotheses, carried out statistical analysis and drafted the manuscript. Stephanie Gusler contributed to writing the manuscript. Yo Jackson provided critical review of the manuscript. All authors reviewed and approved the final manuscript as submitted.

CONFLICT OF INTEREST

No conflict of interest was declared.

**Keywords**

adverse childhood experiences; eating; emotion regulation; intergenerational transmission; maltreatment; obesity

## 1 | INTRODUCTION

Childhood adversity, including exposure to violence, household dysfunction, and housing and food instability is associated with unhealthy eating behaviours in childhood and the development of obesity across the lifespan.[1-3] Some evidence suggests that this association begins in early childhood.[4-6] For instance, in studies of preschool-age children, exposure to adversity is associated with eating in the absence of hunger and emotional overeating, and may also be linked to greater consumption of obesogenic foods (eg, fast food, sugar-sweetened beverages, sweets, salty snacks).[7-9] Identifying mechanisms underlying the association between adversity and obesogenic food consumption in young children and intervening on this pathway as early as possible is crucial given that unhealthy eating behaviours contribute to childhood obesity.[10] Childhood obesity is known to persist into adulthood and precipitate life-long health problems.[11]

Emotion dysregulation is defined as difficulty modifying emotions and behaviour in response to environmental demands and to the same extent as peers in similar situations. Emotion dysregulation includes maladaptive emotion valence, intensity and/or duration that causes functional interference or behaviour problems beyond age expectations.[12,13] Individuals who are dysregulated typically exhibit emotional extremes or emotions that are inappropriate for the situation/context.[12,13] In separate literatures, emotion dysregulation is associated with adversity exposure *and* unhealthy eating behaviour. Thus, emotion dysregulation may be an understudied yet key mechanism linking adversity to obesogenic food consumption.

Numerous studies over several decades have shown emotion dysregulation in children exposed to adversity.[14,15] A recent meta-analysis drawing on 58 papers and encompassing more than 11 900 children found a medium to large effect size (0.42) indicating that children exposed to adversity were significantly more likely to display emotion dysregulation compared to non-exposed children.[16] This finding is evident in every age group, including preschool-age children. For example, in a recent study of predominantly Hispanic preschoolers, children's exposure to cumulative lifetime adversity was positively associated with teacher-rated emotion dysregulation.[17] Empirical evidence suggests that the cumulative nature of adverse events ‘piling up’ is a more potent predictor of maladaptive functioning than a single adverse event, or the dichotomous measurement of adversity as either present or absent.[18] With the exception of the aforementioned study, most research fails to employ a comprehensive measure of lifetime adversity, making it challenging to understand the downstream effects of aggregate risk.[18]

In addition to its association with adversity, emotion dysregulation is also linked with obesogenic food consumption. A recent review found that children with emotion dysregulation have been shown to select and overeat highly palatable and emotionally

rewarding junk food.[19] Another review showed that when children had less emotion dysregulation, they selected healthier foods.[20] For example, in one study, children with loss of control around eating had higher parent-reported emotion dysregulation than those who did not have loss of control around eating; parent-reported emotion dysregulation was also associated with greater observed energy intake after accounting for body mass.[21]

Only one study to date has tested a pathway including adversity, self-regulatory ability and an obesity-related outcome in the same model. Findings demonstrated that in a sample of 9-year-olds, adversity was associated with larger gains in adiposity over time, and that these gains were largely explained by difficulty delaying gratification (ie, ability to wait for candy).[22] The current study seeks to build upon this initial evidence by testing a broader conceptualization of self-regulatory ability, namely emotion dysregulation, which may be an especially potent predictor for children exposed to adversity. Despite piecemeal support for emotion dysregulation as an intermediary mechanism between adversity and obesogenic food consumption in young children, a major gap remains in that the adversity and obesity literatures have yet to be brought together to more definitively test this potential cascade of risk.

Importantly, caregivers have a major influence on child emotion dysregulation throughout the preschool years, so much so that their own emotion dysregulation should be considered in any study of child emotion dysregulation.[23,24] Specifically, the tripartite model emphasizes caregivers' expression, modulation, modelling and socialization of emotion as major contributors to children's tendency towards emotion dysregulation.[25] Children who observe their caregivers having primarily heightened expressions of negativity and dysregulation are more likely to develop constrained emotional understanding and increased emotion dysregulation themselves.[26] Thus, children who are exposed to early adversity *and* experience caregiver emotion dysregulation may be at greatest risk for emotion dysregulation themselves. However, it unknown how these transgenerational pathways affect obesogenic food consumption.

The purpose of the current study was to examine the possible indirect pathway between cumulative lifetime adversity exposure and obesogenic food consumption in young children through the mechanism of child emotion dysregulation. Caregiver emotion dysregulation was also tested as a moderator of the adversity/child emotion dysregulation association. Guided by the tripartite model, it was hypothesized that caregiver emotion dysregulation would moderate the relation between adversity and child emotion dysregulation such that greater cumulative lifetime adversity would be associated with higher child emotion dysregulation for children whose caregivers were high in emotion dysregulation. In turn, child emotion dysregulation would be associated with increased obesogenic food consumption over time.

## 2 | METHODS

### 2.1 | Participants

Participants were 190 caregiver-child dyads who completed the first and second time points of a larger longitudinal project, the Preschoolers' Adjustment and Intergenerational Risk

(PAIR) project. The second time point of data collection took place 6 months after the first time point. Those who completed two time points did not differ from those in the larger study who completed one time point on measures of cumulative lifetime adversity ($t_{426.32}$ = .21; $P$ = .47), child emotion dysregulation ($t_{408.79}$ = .15; $P$ = .16), caregiver emotion dysregulation ($t_{422.06}$ = .19; $P$ = .17) or baseline obesogenic food consumption ($t_{402.23}$ = .30; $P$ = .25).

At time point 1, 56 children were 3-years-old, 64 children were 4-years-old, and 70 children were 5-years-old ($M$ = 4.31 years, SD = 0.85). Approximately 48% of children were female. The racial/ethnic background of the children was 74% African American or Black, 12% Multiracial, 9% Caucasian or White, 5% Other and 1% Hispanic. The average BMIz was 0.85 (80th percentile) for the total sample. Girls had an average BMIz of 0.73 (77th percentile), while boys had an average BMIz of 0.96 (83rd percentile).

Caregivers ranged in age from 21 years to 70 years, with a mean of 31 years (SD = 7.52). Approximately 96% of caregivers were female, and 51% were in a single caregiver household. Most caregivers were biological mothers (91%), with 4% biological fathers, 3% biological grandmothers and 2% adoptive mothers/legal guardians. The racial/ethnic background of the caregivers was 81% African American or Black, 4% Multiracial, 10% Caucasian or White, 5% Other and 2% Hispanic. Caregivers' BMI scores ranged from 16.12 to 64.43 for the whole sample, with a mean of 33.78 (SD = 9.34). The educational background of caregivers included 3% who completed some grade school, 15% who completed some high school, 15% who graduated high school or obtained a GED, 15% who graduated trade school or community college, 28% who completed some college, 6% who graduated with a four-year degree, 2% who attended graduate school and 6% missing. Most families had a yearly household income of $10 000 or less (44%), and 18% of families reported a yearly income of $10 001 to $20 000, 17% reported $20 001 to $30 000, 8% reported $30 001 to $40 000, 8% reported $40 001 to $50 000 and 5% reported a yearly income of $50 001 or higher.

Participants were all living in an urban Midwest city and recruited either from community organizations, serving families living below the poverty line (90% of the sample), or from the state's Department of Social Services (DSS; 10% of the sample). For complete recruitment methods and study procedures, see Griffith et al.[27]

Exclusion criteria for participation in the project included the caregiver or child being non-fluent in English, taking cardiac medications, taking anticoagulants, taking diuretics, having a pacemaker, taking medications for seizures, or having a known diagnosis of an intellectual or developmental disability.

### 2.2 | Procedure

The first participants to complete the study (n = 103) filled out questionnaires using paper and pencil, with research assistants reading questions aloud to caregivers if they indicated difficulties with reading or comprehension. The Audio Computer Assisted Self Interview (ACASI) software, which is on laptop computers, was then introduced and used by all subsequent participants (n = 87 for the current study). Through the ACASI software each

question and answer choice were presented visually and read-aloud to caregivers. Those who completed the measures using the ACASI did not differ from those who completed the measures using paper forms on cumulative lifetime adversity ($t_{406.85}$ = .30; $P$ = .82), child emotion dysregulation ($t_{415.94}$ = .45; $P$ = .91), caregiver emotion dysregulation ($t_{415.02}$ = .27, $P$ = .55), baseline obesogenic food consumption ($t_{412.82}$ = .35, $P$ = .40) or follow-up obesogenic food consumption ($t_{412.10}$ = .41, $P$ = .37). All participants were compensated with a gift card for their time and participation. All project recruitment and procedure methods were approved by the University of Kansas Institutional Review Board and DSS.

## 2.3 | Measures

**2.3.1 | Demographics—**At time points 1 and 2, caregivers provided demographic information including child and caregiver age, sex and race/ethnicity; caregiver education level; single caregiver household status; and family income.

**2.3.2 | Cumulative lifetime adversity exposure—**A measure of children's adverse experiences was created for the purposes of the larger study (ie, the Childhood Experiences Measure), by accumulating a comprehensive list of adverse life events that have been used in previously validated measures and synthesizing the items for complete coverage of possible events and to eliminate overlap of items. The majority of the items in the current measure were collected from the Preschool Age Psychiatric Assessment (PAPA),[28] Life Events Checklist (LEC),[29] Trauma History Questionnaire (THQ),[30] Juvenile Victimization Questionnaire (JVQ)[31] and Child and Adolescent Needs and Strengths (CANS).[32] The items include several kinds of adversities, including those associated with violence exposure, natural disaster, maltreatment, difficulty based on current income, household dysfunction, family or community involvement with the law, death or separation from close others and physical or psychological difficulties. It is the most comprehensive list of possible event items relevant for preschool to school-age children in existence.

Aside from the items included, the design of the measure was influenced by previous measures, as well as several review articles and consortiums on adversity exposure assessment (eg, National Child Traumatic Stress Network; www.nctsn.org) that described various limitations of the currently available adversity measurement tools.[33] To address these limitations, the measure assessed for both direct and indirect exposure to adversity, chronic and acute events, and the time frame of events from first to last exposure. The current study used data from this measure collected at time point 1.

Caregivers reported on their child's experience of 50 adverse life events (eg, caregivers divorcing, caregiver incarceration, reduction in standards of living, caregiver substance abuse, witnessing violence, accidental injuries, removal from the home due to abuse or neglect). Adversity exposure was operationalized as the sum of all possible events (ie, cumulative lifetime adversity exposure); it was calculated by dichotomizing each item as 1 = endorsed as having occurred to the child and 0 = not endorsed, and summing all events such that scores could range from 0 to 50.

In this sample, number of cumulative lifetime adverse events children were exposed to ranges from 0 to 23, with a mean of 7.01 events (SD = 5.52); 2.10% of the sample

experienced 0 adverse events, 37.30% experienced 1 to 5 adverse events, 24.50% experienced 6 to 10 adverse events, 27.90% experienced 11 to 15 adverse events, 6.20% experienced 16 to 20 adverse events and 2.00% experienced 21 to 24 adverse events. At some point in their childhood, 6.80% of children had been removed from their home due to abuse or neglect. Of those who had been removed from their home due to maltreatment, all but one resided with their biological mother or father at the time of the study.

**2.3.3 | Caregivers' emotion dysregulation—**The Difficulties with Emotion Regulation Scale (DERS)[34] administered at time point 1 was used as a self-report measure of caregivers' emotion dysregulation. The DERS consists of 36 items, each of which is rated on a five-point Likert scale from 1 ‘Almost Never’ to 5 ‘Almost Always’. The DERS measures emotional awareness (eg, ‘I pay attention to how I feel’), emotional clarity (eg, ‘I am clear about my feelings’), nonacceptance of emotions (eg, ‘When I'm upset, I feel like I am weak’), regulation strategies (eg, ‘When I'm upset, it takes me a long time to feel better’), impulse control (eg, ‘When I'm upset, I lose control over my behaviors’) and goal-oriented behavior (eg, ‘When I'm upset, I have difficulty concentrating’). Items indicative of positive emotion regulation (eg, ‘I pay attention to how I feel’ and ‘I am clear about my feelings’) were reverse scored and then each item was compiled to create a total score, with higher scores indicating greater emotion dysregulation. This total DERS score has evidenced high internal consistency ($\alpha$ = .93) in previous research,[34] and shows adequate consistency in the current study ($\alpha$ = .79). Within the non-clinical norm sample in which the DERS was initially tested, the total DERS mean score was 77.99. In the current sample, 71.02% of caregivers fell above this score ($M$ = 85.00), suggesting that the current sample may have more emotion dysregulation than a typical sample.[34]

**2.3.4 | Children's emotion dysregulation—**Children's emotion dysregulation was measured at time point 1 using the caregiver-report Emotion Regulation Checklist (ERC).[35,36] The Lability/Negativity subscale was used in the current study given its high reliability in previous samples ($\alpha$ = .96 in a school-age sample[35] and $\alpha$ = .85 in a preschool-age sample[26]) and its construct validity for emotion dysregulation.[35,36] The Lability/Negativity subscale consists of 15 items, for which caregivers' rate their child's behaviours on a scale from 1 ‘Almost Always’ to 4 ‘Never’. The Lability/Negativity subscale captures caregivers' perceptions of their child's dysregulation (eg, ‘Is prone to angry outbursts/tantrums easily’, ‘transitions well from one activity to another; doesn't become angry, anxious, distressed, or overly excited when moving from one activity to another’, ‘can recover quickly from upset or distress [for example, doesn't pout or remain anxious or sad after emotionally upsetting events]’, ‘exhibits wide mood swings [child's emotional state difficult to anticipate because he/she moves quickly from a positive to a negative mood]’). Items phrased in terms of positive ER abilities were reverse coded and a sum score across the 15 items was used to capture dysregulation, with high scores indicating more dysregulation. For the current study, the internal consistency for the Lability/Negativity subscale of the ERC was good ($\alpha$ = .82). The ERC has evidenced validity in discriminating between groups of children who have experienced maltreatment and those who have not and between those who display emotion dysregulation during brief observations from those who do not.[35,36] Further, among samples of preschoolers who are enrolled in Head Start or whose families are receiving community

supports, the mean Lability/Negativity score has ranged from 28.93 to 32.37,[37] which is comparable to the mean score in the current sample ($M$ = 29.00).

**2.3.5 | Obesogenic food consumption—**The current study used the Children's Eating Habits Questionnaire[38] at time points 1 and 2 to assess overall pattern of obesogenic food consumption. Caregivers rated how often in the past 7 days their child had consumed sugar-sweetened beverages, fast food, sweets and salty snacks on a scale of 1 = 'none' to 7= '4 or more times per day'. Higher scores on each of these items reflect more frequent weekly consumption of these foods. The scores for each food category were added together to create an obesogenic food consumption index, with higher scores indicating an unhealthier diet. Jackson and Vaughn found that this index demonstrated adequate internal consistency in a sample of young children ($\alpha$ = .70).[9] Internal consistency in the current sample was good ($\alpha$ = .75).

**2.3.6 | Child BMIz and caregiver BMI—**Children's and caregivers' heights and weights were collected using the SECA 213 portable stadiometer and SECA 813 scale at time points 1 and 2. Participants were instructed to remove their shoes and outwear (eg, jackets or coats). Height was measured to the nearest millimetre, and weight was measured in kilograms. Heights and weights were measured to two decimal places. Measurements for both height and weight were obtained three times and then averaged. Child body mass index z-scores (BMIz) were obtained using the Pediatric Z-score Calculator (available at https://zscore.research.chop.edu/), wherein weight in kilograms is divided by height in meters squared, and is converted to z-scores based on children's age and gender. Caregiver BMI was obtained using the Adult BMI Calculation (available at https://www.cdc.gov/healthyweight/assessing/bmi/adult_bmi/english_bmi_calculator/ bmi_calculator.html).

## 2.4 | Statistical analysis

Descriptive statistics and correlations among study variables were first examined using SPSS software, version 25. All variables included in the analyses met appropriate standards for skewness and kurtosis. Rates of missingness in the dataset were not associated with any of the primary study variables or covariates. There were no significant differences in primary study variables and covariates between families that were recruited from community organizations vs DSS. To maximize sample size, full-information maximum likelihood estimation (FIML) was used to accommodate missing data (2.02% of values). The FIML method yields the most accurate results compared to other methods (eg, mean replacement, listwise deletion) by minimizing bias in regression and SE estimates for all types of missing data (ie, MCAR, MAR, NMAR) when the amount of missing data does not exceed 20%.[39]

Only one potential covariate (child age) was related to emotion dysregulation, and thus was included in final models. Child BMIz and caregiver BMI were also included in final models given their relevance to the outcome of interest. However, because other covariates (child sex, child race and ethnicity, caregiver age, caregiver sex, caregiver race and ethnicity, caregiver education level, single caregiver household status) failed to predict child emotion dysregulation or health behaviour in any of the models, we maximized parsimony by

excluding these variables from the final models. Furthermore, the pattern of results remained identical with and without inclusion of these other variables in analytic models.

The primary hypothesis was tested using autoregressive structural equation modelling (SEM) in Mplus 7.0.[40] Cumulative lifetime adversity exposure, caregiver emotion dysregulation, their interaction, and covariates were specified as predictors of child emotion dysregulation, which in turn predicted change in obesogenic food consumption 6 months later. The autoregressive path between obesogenic food consumption at baseline and follow up was included. Model fit was assessed using standard criteria, including the root mean square error of approximation (RMSEA; values <.08 indicate acceptable fit), comparative fit index (CFI; values > .90 indicate acceptable fit), Tucker-Lewis index (TLI; values >.90 indicate acceptable fit) and the standardized root mean square residual (SRMR; values <.08 indicate acceptable fit).[41]

To reduce multicollinearity, the continuous predictor (cumulative lifetime adversity) and moderator (caregiver emotion dysregulation) were mean-centred, then multiplied to create the interaction term.[42] The significant interaction term was probed using procedures specified by Aiken and colleagues, and simple slopes were calculated at low (−1 SD) and high (+1 SD) levels of the moderator variable.[42] To test for hypothesized moderated indirect effects, models were assessed using 1000 bootstrap replicates to obtain bias-corrected bootstrap confidence intervals for the indirect effects.[43]

## 3 | RESULTS

### 3.1 | Descriptive analyses

Table 1 provides means, standard deviations and intercorrelations for the variables used in the primary analysis. Although cumulative lifetime adversity exposure and caregiver emotion dysregulation were significantly correlated in our sample ($r = .19$), the association was modest, indicating sufficient variability to examine potential interactions between the two constructs.

### 3.2 | Primary analysis

#### 3.2.1 | Indirect effect of cumulative lifetime adversity exposure on obesogenic food consumption via child emotion dysregulation, with caregiver emotion dysregulation as a moderator—

The model tested included child emotion dysregulation as the intermediary mechanism between early childhood cumulative lifetime adversity exposure and obesogenic food consumption, with caregiver emotion dysregulation moderating the pathway between adversity exposure and child emotion dysregulation. Child age, child BMIz and caregiver BMI were included as covariates. Correlations were specified among exogenous predictors. The model provided good fit to the data, $\chi^2(80) = 110.16$, $P < .001$, RMSEA = .05, CFI = .97, TLI = .98, SRMR = .05. As Figure 1 illustrates, the interaction between adversity exposure and caregiver emotion dysregulation was associated with child emotion dysregulation ($\beta = .48$, SE = .09, $P = .001$). Child emotion dysregulation, in turn, predicted greater obesogenic food consumption from baseline to 6-month follow up ($\beta = .32$, SE = .08, $P = .001$). This model accounted for a significant amount of the variance

in obesogenic food consumption at 6-month follow up ($R^2$ = .19, $P$ = .001), while constraining the autoregressive path to zero.

As depicted in Figure 2, simple slopes demonstrated that adversity exposure was positively associated with child emotion dysregulation when caregiver emotion dysregulation was high (b = .24, SE = .11, $P$ = .01) but not low ($b$ = −.10, SE = .10, $P$ = .22). Results of bias-corrected bootstrapping tests indicated that the indirect path involving the interaction between adversity exposure and caregiver emotion dysregulation, and obesogenic food consumption was significantly different from zero (95% CI [.05, 3.17]). Specifically, higher levels of child emotion dysregulation served as an intermediary mechanism between adversity exposure and obesogenic food consumption only when caregiver emotion dysregulation was high (95% CI [.12, 2.33]), not low (95% CI [−0.41, .02].

## 4 | DISCUSSION

In a sample of low-income preschool-age children, greater cumulative lifetime adversity exposure was associated with higher emotion dysregulation, but only when caregiver emotion dysregulation was high. In turn, child emotion dysregulation was associated with an increase in obesogenic food consumption 6 months later. These findings support the hypothesis that child emotion dysregulation is likely a key mechanism by which early adversity increases unhealthy eating behaviours that place children at risk for obesity, specifically for children whose caregivers also have high levels of emotion dysregulation. In summary, child *and* caregiver emotion dysregulation may be viable targets for interventions aimed at reducing obesity risk in young children exposed to significant early adversity.

Given the robust association between adversity in childhood and obesity in adulthood,[1-6] examination of the preliminary mechanisms underlying this relation is warranted yet understudied in very young children. The current results contribute to the growing but limited prior work by demonstrating an indirect association between early adversity and obesogenic food consumption through child emotion dysregulation, among children with emotionally dysregulated caregivers. The observed association between greater cumulative lifetime adversity exposure and higher obesogenic food consumption is consistent with previous research showing that cumulative lifetime adversity score was linked to obesogenic food consumption in both crosssectional and longitudinal analyses, even when accounting for multiple covariates.[9] Similarly, in a sample of low-income children, early stress exposure predicted increases in child eating behaviours associated with obesity.[7] The current findings add to the extant literature on adversity and eating behaviour by providing novel evidence that this association may come about through child emotion dysregulation. Moreover, these findings provide evidence that general emotion dysregulation, not just eating-specific regulation, is linked to obesogenic food consumption.

Regarding the moderating role of caregiver emotion dysregulation, these results extend past work. For example, in a sample of preschool-age children and their parents, parent and child effortful control, a construct similar to emotion regulation, was positively correlated with each other, and both negatively correlated with children's consumption of candy.[8] Although this study did not examine causal associations, its findings suggest parent emotion

dysregulation as a critical factor influencing children's eating behaviour. In addition, Miller and colleagues found that proximal parenting factors (eg, depression and parenting style) were more strongly associated with child emotional overeating than more distal factors such as violence exposure.[7] This is somewhat in line with the present finding that adversity exposure was associated with emotion dysregulation only among children who lived with a caregiver who was emotionally dysregulated, thus implicating caregiver factors as crucial to obesity development.[44]

Given the major influence of caregivers' own regulation abilities on ER development in the preschool years, it is possible that a caregivers' emotion dysregulation may cause the child to become dysregulated themselves, with chronic emotion dysregulation in the caregiver begetting chronic emotion dysregulation in the child.[24] A caregivers' emotion dysregulation may also add to a child's emotion dysregulation that has stemmed from adversity exposure and other environmental stressors.[24] The tripartite model posits that a caregiver with emotion dysregulation may be unable to model positive emotion regulation strategies for their child or provide opportunities to practice strategies and fail to scaffold the child appropriately when they become dysregulated.[25] Any of these scenarios, or their combination, may make it more likely for the child to consume high-fat, high-sugar foods in an attempt to manage their dysregulation. It is also possible that emotion dysregulation prevents the caregiver from intervening with the child's eating behaviour. For instance, a dysregulated caregiver may be unable to respond in a helpful way when their child is consuming or over-consuming unhealthy foods, or may allow unhealthy eating because it soothes the child and causes the caregiver less distress.[45]

Dysregulated caregivers may also create more obesogenic food environments. For example, greater maternal executive function difficulties (ie, problems with higher order cognitive processes that allow individuals to control their thoughts, emotions, and actions) have been associated with less use of recommended food-related parenting practices and less healthful home food environment characteristics such as providing frequent family meals at home, implementing consistent mealtime schedules and structure, and avoiding using food to regulate children's emotions.[46] Dysregulated children may be particularly likely to have difficulty in a home environment such as this.

Finally, these hypotheses do not dismiss the possibility of children's emotion dysregulation also influencing emotion dysregulation in their caregiver, as the caregiver-child relationship is bidirectional.[47] For example, prior research has found that child factors such as temperament influence caregivers' emotional reactions to their children.[48] Further, in one study child emotion dysregulation was associated with parenting stress, and stability of parenting stress over time was influenced by the interaction of child emotion dysregulation and externalizing problems.[49] The present findings likely reflect complexity in households marked with significant adversity, suggesting the importance of both caregiver and child contributions to eating behaviours. Future studies should examine the possible reciprocal relationship between caregiver and child emotion dysregulation, and its impact on obesogenic food consumption.

### 4.1 | Limitations and strengths

These findings should be interpreted with consideration of several limitations. First, the outcome of interest—obesogenic food consumption—was obtained from a caregiver-reported measure and thus was not objectively assessed. In addition, this questionnaire measures only one dimension of a broad construct. Future studies should examine other aspects of eating behaviour aside from diet composition, such as food and satiety responsiveness, and utilize observational protocols. Second, caregivers reported on their children's adversity exposure and emotion dysregulation, as well as their own v, which may have introduced some bias into the results, such that dysregulated caregivers may have been more likely to report higher levels of dysregulation in their children. To strengthen this measurement, future studies should seek to replicate these results using multireporter, multi-method approaches, including examination of physiological measures of emotion dysregulation, such as vagal tone and respiratory sinus arrhythmia. The heavy reliance on caregiver-reported measures in this study somewhat limits the conclusions that can be drawn, and speaks to the importance of future studies investigating these pathways using non-caregiver-reported measures. Third, although the longitudinal nature of this study is a strength, associations should be examined over longer periods of time and into later childhood and adolescence to determine how early adversity and emotion dysregulation affect important health behaviours and obesity over the lifespan. The current study was not long enough to explore whether adversity and emotion dysregulation impacted changes in body mass index or other anthropometric indicators of obesity. Further, adversity exposure, caregiver emotion dysregulation and child emotion dysregulation were measured at the same time point, making it impossible to determine true causal associations, or bidirectional relationships, among these three variables. Future research, beginning in infancy and extending over a longer time-period, would be needed to examine causal associations between caregiver emotion dysregulation, child emotion dysregulation and adversity exposure.

These limitations are offset by the fact that this study is novel in its effort to examine a key predictor of unhealthy eating—emotion dysregulation—in both children and caregivers in an effort to determine its role in obesity development in young children exposed to adversity. The study established that general emotion dysregulation, not just regulation around eating behaviours, is related to obesogenic food consumption in early childhood. Given that cumulative lifetime adversity exposure may be a stronger predictor of increased health risk compared to dichotomously measuring the presence or absence of adversity,[18,50] this study employed a comprehensive measure of cumulative lifetime adversity exposure. In addition, the study examined obesogenic food consumption with a widely used, valid and reliable measure, allowing for a clearer understanding of how adversity and emotion dysregulation is associated with unhealthy eating.

### 4.2 | Conclusions

An emerging body of literature has shown links among adversity, emotion dysregulation, eating behaviours and obesity, which has led some scholars to advocate for trauma-informed obesity prevention.[9] The current findings support this argument, given results that early childhood adversity is associated with increased obesogenic food consumption through child

emotion dysregulation, but only for those whose caregivers have high emotion dysregulation. Both caregiver and child emotion dysregulation may be important mechanisms contributing to obesogenic food consumption and ultimately childhood obesity, thus interventions focused on improving transgenerational emotion dysregulation are needed to prevent obesity among high-risk children such as these.

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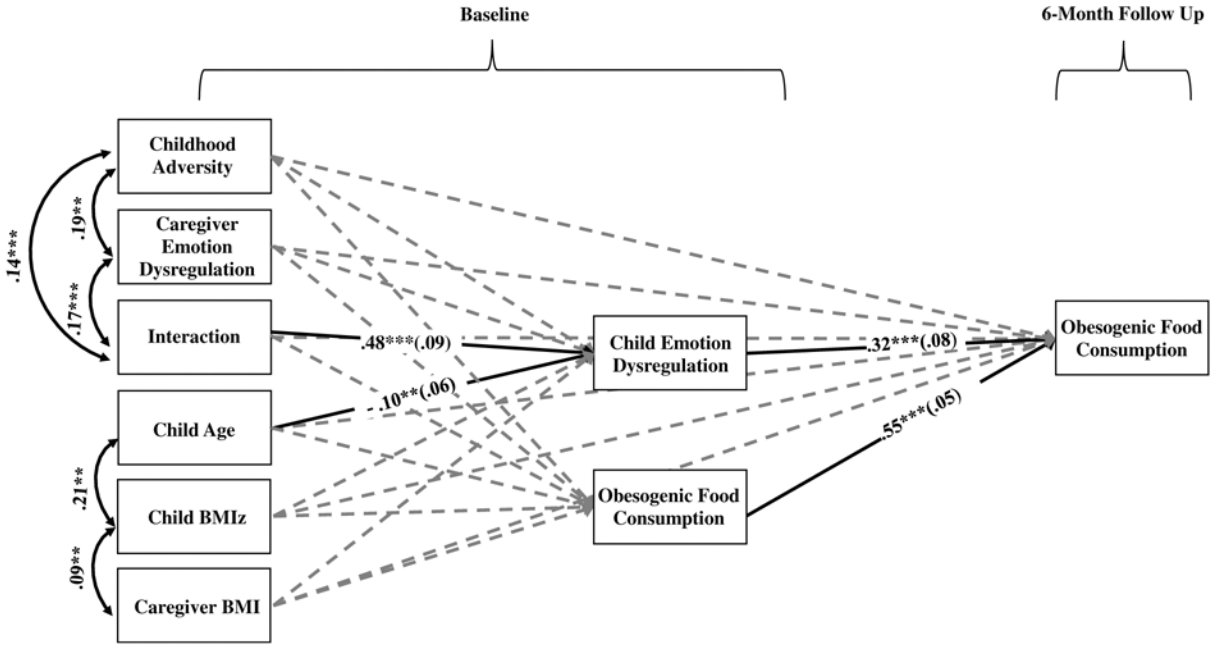


**FIGURE 1.**

Autoregressive path model testing indirect effect of childhood adversity on change in obesogenic food consumption through child emotion dysregulation, moderated by caregiver emotion dysregulation. Interaction refers to the interaction between child adversity and caregiver emotion dysregulation. Parameter estimates for structural paths are standardized path coefficients, with standard errors in parentheses. Dashed lines indicate non-significant pathways. For clarity, only significant path coefficients are depicted. Correlations were specified among exogenous predictors. For clarity, only significant correlations are depicted. $*P < .05$. $**P < .01$. $***P < .001$

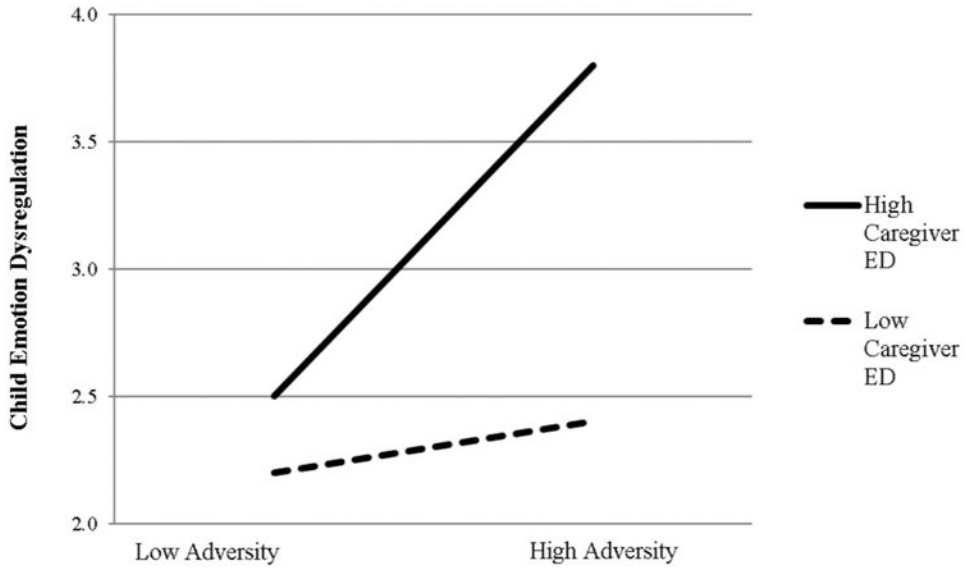


**FIGURE 2.**

Predicted values for child emotion dysregulation (ED) illustrating the interaction of level of childhood adversity and caregiver emotion dysregulation at values that are one standard deviation below and above their respective means

**TABLE 1**

Descriptive statistics and correlations among substantive variables of interest

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
|---|---|---|---|---|---|---|---|---|
| 1. Child adversity | - | | | | | | | |
| 2. Parent emotion dysregulation | .19** | - | | | | | | |
| 3. Child emotion dysregulation | .37*** | .11** | - | | | | | |
| 4. Obesogenic food consumption—baseline | .10* | .06* | .11* | - | | | | |
| 5. Obesogenic food consumption—6-month follow up | .32** | .31** | .41** | .58** | - | | | |
| *Covariates included in models* | | | | | | | | |
| 6. Child age | .05 | .05 | −.15* | .01 | .06 | - | | |
| 7. Child BMIz | .09 | .05 | .07 | .10* | .10* | .21** | - | |
| 8. Parent BMI | .03 | .05 | .05 | .11* | .13* | .01 | .09** | - |
| Mean | 7.01 | 85.00 | 29.00 | 17.00 | 18.50 | 4.31 | 0.85 | 33.78 |
| SD | 5.52 | 15.00 | 8.00 | 5.25 | 4.90 | 0.85 | 0.16 | 9.34 |

*$P < .05$.

**$P < .01$.

***$P < .001$.