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Exploring the Association Between Trauma, Instability, and Youth Cardiometabolic Health Outcomes Over Three Years

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Abstract

Purpose: Childhood adversity plays a fundamental role in predicting youth cardiometabolic health. Our understanding of how adverse experiences in childhood should best be conceptualized remains elusive, based on one-dimensional measures of adversity. The present study fills a major gap in existing research by examining two distinct forms of threat and instability-related exposures that may impact cardiometabolic risk (CMR) in adolescence.

Methods: We explore two specific subtypes of adversity: trauma (e.g., badly hurt, victim of crime, loss of close person) and instability (e.g., moving, change of schools, change in household structure) as differential influences that can accumulate to impact early childhood onset of CMR (body mass index, high-density lipoprotein (HDL), low-density lipoprotein, diastolic and systolic blood pressure, triglycerides, C-reactive protein, insulin sensitivity). Secondary data were drawn from a randomized control behavioral trial of youth recruited during sixth grade from urban Cleveland (Ohio) schools beginning in 2012–2014 (n = 360) and followed for 3 years. Participants reported on 12 adverse experiences, six trauma- and six instability-specific. Multiple regression assessed effects of prospective and accumulative indices of trauma and instability with 3-year trajectories of eight objective CMR markers.

Results: Instability was associated with increased body mass index, decreased high-density lipoprotein, and increased C-reactive protein slopes. Trauma was associated with trends in triglyceride levels but not with any other CMR outcomes.

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Discussion: Experiences with instability distinctly impacted adolescent CMR. Future research is needed to examine factors that can enhance stability for families in marginalized communities.

Keywords

Adverse childhood experiences; Trauma; Instability; Low-income; Adolescence; Cardiometabolic risk; Obesity; Diabetes

Research suggests adverse childhood experiences occurring across the first 10e18 years of life can have negative effects on cardiovascular health in late-stage adolescence and young adulthood [1,2]. Recent theoretical innovations propose an integration of dimensional models of risk to include assessment of forms of threat and deprivation that occur in contexts with varying levels of harshness and unpredictability [3]. Although existing research has established the association between early life adversity and later health risks in both children and adults [1,4–8], far less is known about the impact of unpredictability or instability on the development of cardiometabolic risk (CMR) during adolescence. This is important because instability is often less visible than trauma and not as easy to identify and address through individualized interventions. The present study aims to fill several major gaps in existing literature by examining two distinct forms of threat and instability-related exposures in underserved communities that may impact CMR in adolescence.

Childhood CMR may be the consequence of disrupted development after exposure to adverse circumstances. A prolonged heightened stress response after trauma exposure can cause changes to the immune, metabolic, neuroendocrine, behavioral, and psychosocial systems that can impact CMR [9] or hinder social, cognitive, and emotional functioning that contributes to health behaviors that increase CMR [8,10]. Recent theories on the neurological mechanisms of adversity and child mental health have begun to distinguish different aspects of adversity, such as threat (i.e., trauma) versus deprivation [11,12] or harmful, threatening experiences versus the absence of needed resources. Our study builds upon an integrated model that accounts for the lack of predictability or instability in access to needed resources, in addition to focusing on trauma as two underlying dimensions [3].

The trauma dimension is consistent with traditional definitions of traumatic or adverse childhood experiences and includes unexpected events that harm, victimize (i.e., arrested by police, crime victim), or threaten children (i.e., sudden loss of parent or close relationship). Instability refers to the lack of, or unstable and inconsistent access to, necessary basic physical and social resources such as unstable housing (e.g., multiple relocations, uncertainty of housing stability), inconsistent access to food, and irregular access to basic utilities (e.g., electricity, water) [13]. Fear of community violence is associated with lower social cohesion, which reduces trust, exacerbates concerns over physical safety, and can place limitations on activity outside the home and increase CMR risks [14,15].

Inconsistent inputs can destabilize the social or physical environment and heighten the perception of perceived threats [16,17]. Experiences with instability are typically less acute than traumatic experiences (e.g., that involve the loss of a loved one or being badly hurt or sick) but can result in major changes in regular routines, including health behaviors correlated with CMR. In two recent studies, childhood instability was found

to be a robust and independent predictor of adult health-related outcomes, including health-related quality of life, functional ability, and discounting and externalizing behaviors, while controlling for other measures of childhood adversity [16,18]. Perceptions of instability can stem from inconsistency in the degree of support a child receives from their primary caregiver or other adults or from variability in a child's daily schedule or routines [16]. For example, parental divorce can result in major changes to daily routines, structured and unstructured activities, and can dramatically shift the levels of support available to the family, contributing to increased feelings of uncertainty and instability [19]. Research on economic instability consistently shows accumulated economic stress experienced in early childhood is associated with higher body mass index (BMI) percentile growth [7] and less healthful eating patterns during adolescence that contribute to CMR [8]. Broader economic shifts leading to rapid increases in food, housing, and utility costs [20] absorb substantially larger proportions of income for households with limited resources, which can ultimately negatively affect health behaviors that impact CMR [21]. Further, high levels of economic hardship can increase parenting stress, which can result in poorer dietary outcomes [22] and shifts in protective health routines (e.g., physical activity, sleep) that impair growth patterns optimal for promoting cardiometabolic health [23]. Because families can experience instability that extends beyond separate income, food, or housing issues alone, a better understanding of the accumulation of instability on CMR, distinct from exposure to trauma, is needed.

Although exposure to traumatic or violent events may operate through similar mechanisms to impact child CMR [15], most studies of childhood adversity and CMR have combined indicators of both trauma and instability rather than treating them as potentially unique contributors to CMR. The present study aims to address this and several major gaps in related existing literature. The few prospective studies focusing on the impacts of adversity on CMR in adolescence have targeted later stages of adolescence [2,24,25] rather than the critical periods in early adolescence when preventative interventions may be most timely for reducing CMR [26]. Finally, few studies have focused specifically on high-risk youth, such as those who are overweight or obese and whose cardiometabolic health may be particularly susceptible to these environmental stressors. By including eight objective biomarker measures to capture CMR (BMI, high-density lipoprotein [HDL], low-density lipoprotein [LDL], diastolic and systolic blood pressure, triglycerides, C-reactive protein (CRP), and insulin sensitivity), we further address existing limitations of single-outcome or self-reported health measures used in much of the existing research [27,28].

In the present study, we hypothesize that instability, linked to early and sustained impairments in child development and functioning [29], may operate through the same developmental pathways as what is traditionally defined as traumatic, or adverse childhood experiences. Specifically, we aim to test whether trauma and instability during early adolescence are unique drivers associated with early onset risk for CMR prospectively and longitudinally over three years among a high-risk sample of overweight and obese youth. Study findings will provide the specificity needed to guide future research on testing meaningful protective factors on the paths from trauma and instability to adolescent cardiometabolic growth and development.

Method

Procedure and Sample

This prospective study design uses secondary data drawn from a three-year, randomized control behavioral trial [30] that included 360 youth-parent dyads recruited from urban, low-income Cleveland (Ohio) middle schools as youth entered sixth grade (2012e2014, mean age 11.6). Eligible youth had a BMI ≥ 85th percentile. Youth were excluded from the study if they had stage 1 hypertension with end organ damage, stage 2 hypertension, type 1 or two diabetes, sickle cell disease, were taking medications that could alter appetite or weight, or had a known medical condition that causes obesity (e.g., Prader-Willi syndrome). Data were collected at baseline (Year 0 [Y0]), and again annually for 3 years (year 1 [Y1], year 2 [Y2], and year 3 [Y3]) via interview using audio-assisted software in English and Spanish. Participant health outcome data were collected by certified, trained personnel at two Cleveland hospital system clinical research units, and children's schools. Participant retention in the study was 92%, 93.3%, and 91.9% in years 1, 2, and 3, respectively. The study was approved by the Case Western Reserve University human subjects institutional review board. All youth from the primary study intervention and control groups were included in the present study.

For context, all students were recruited from neighborhood-based elementary schools in the Cleveland Metropolitan School District. In 2022, the median household income in Cleveland was $33,678, with 31.4% of the population living below the poverty line. Among adults 25 and older, 82.6% had a high school diploma or GED, and 19.2% had a bachelor's degree or higher [31]. The city is racially diverse, with 47.4% African American, 34.0% non-Hispanic White, 12.2% Hispanic or Latino, 2.5% Asian and Pacific Islander, 0.5% Native American, and 7.1% from two or more races [31]. Cleveland, like many Midwest manufacturing cities, experienced significant urban flight, redlining, and disinvestment within the urban core for decades. While significant investment is now being made, Cleveland remains one of the poorest cities, with a poverty rate well above the national rate and the child poverty rate. Many neighborhoods have scarce resources for healthy living, such as healthy food retail, safe places to play, and safe routes to school.

Measures

Adversity measures.—Adversity measures include *trauma*, comprised of six inter- and intrapersonal trauma items, and *instability*, comprised of six items related to socioeconomic instability, each assessed over the past year and summed to create a unique index. Trauma and instability indices were developed from available secondary data based on existing theory and research.

Trauma.: Starting in Y1, exposure to trauma over the past year was measured each year using the Adolescent Life Change Scale [32]. Youth were asked whether or not they experienced the following six traumatic life experiences: badly hurt or sick, arrested by the police, victim of a crime, death of a parent, sibling, or close friend.

Instability.: Similarly, each year, six items were used to measure instability in the past year: marital separation and divorce (parent report), whether and how many times youth changed schools, whether and how many times the family moved residences (administrative records and parent-report), food insecurity with hunger (United States Department of Agriculture Food Security Survey Module for Children) [33], and fear of crime in their neighborhood (Neighborhood Walkability Scale) [34]. Youth were considered to have "fear of crime in the neighborhood" if they responded *strongly agree* (on a scale of 1-*strongly agree* to 4-*strongly disagree*) to any of the six items related to unstable neighborhood safety (e.g., there is a high crime rate in my neighborhood; the crime rate in my neighborhood makes it unsafe to go on walks alone or with someone at night).

From these data, four unique indices were created within each of the trauma and instability domains: (1) number of trauma exposures and the number of instability exposures at Y1 (baseline); (2) an accumulative index of total number of traumatic and instability exposures across all three years; and (3) two dichotomous indicators to compare those with high (3+) and moderate (2+) levels of exposure in each of the three time points compared to those with fewer and less frequent traumatic or instability experiences. This approach allowed us to determine if outcomes differed for those with high or moderate levels compared to lower levels of persistent exposure to trauma or instability.

Youth CMR trajectories.—CMR outcomes were assessed using objective biomarker measures to capture CMR (BMI, HDL, LDL, blood pressure, triglycerides, CRP, and insulin sensitivity) yearly from baseline (Y0) through the third and final year of the study (Y3). BMI, measured by trained study personnel/staff, was calculated by dividing weight (measured to the nearest 0.1 kg) by height (m^2; measured to nearest 0.1 cm). Systolic and diastolic blood pressure was measured using the OMRON HEM-705-CPN Digital Blood Pressure Monitor, an automated blood pressure measurement device. The second and third readings were recorded to the nearest integer and averaged. Blood specimens were collected by a trained phlebotomist after 8 hours of fasting and analyzed by Northwest Lipid Metabolism and Diabetes Research Laboratories. Blood draws were conducted at baseline, year one, and year three only, yielding biomedical measures of HDL and LDL cholesterol, triglycerides, high-sensitivity CRP, and insulin resistance (homeostatic model assessment for insulin resistance). Additional details on procedures can be found in prior publications [30,35].

Linear slope factor scores were generated to assess the longitudinal trajectory of change for each CMR outcome separately [30]. A slope trajectory over 3 years was calculated for each participant for each CMR outcome. Interpolation and imputation were used to calculate slopes for missing CMR outcome variables. Imputation of BMI was conducted for 14 participants missing all BMI follow-up data by using 13 prespecified imputation variables and 1,000 imputations. Slopes were interpolated for BMI measures available at multiple assessment points. For all other health trajectories, 100 runs were used for the computation of slopes.

Demographic characteristics.: Demographic characteristics were included as covariates based on research suggesting potential confounding with key study variables: parent-

reported education level (<high school degree, high school degree, some college or college degree [reference group]), child age, child sex dichotomized as male and female (reference group-male), and child BMI (when BMI was not the outcome) [36–38]. We also controlled for effects of participation in each of the two intervention arms, comparing to the control group. Due to potential sex/gender differences in risk exposure, with increased risk for females [39], interaction effects of trauma and instability with child sex on CMR outcomes were examined.

Data analysis

Multivariable linear regression analysis was conducted in SPSS V.25. For each CMR outcome (i.e., ΔY0-Y3), regression models tested: (1) Y1 baseline trauma and instability exposures; (2) accumulative index of traumatic and instability exposures across all three years; and (3) 2+ or 3+ traumatic or instability experiences in each year for 3 years. Regression models further adjusted to demographic, intervention/control group, child BMI (except for BMI outcome models), and baseline (Y0) outcome.

Results

Sample characteristics

Sample characteristics are provided in Table 1. Youth in the sample were primarily female (57.8%, n =208) and non-Hispanic Black (76.7%, n = 276), followed by Hispanic (16.4%, n = 59), non-Hispanic white (3.9%, n = 14), multi-racial (2.2%, n = 8), or of another race (0.8%, n = 3). Over 70% of the families were participating in the Supplemental Nutrition Assistance Program (SNAP) or the Special Supplemental Nutrition Assistance Program for Women, Infants, and Children (WIC) at baseline, and 53% of the index parents had some college education or more. At baseline, the mean BMI percentile was 95.69 (SD = 3.72). More than three-fourths of youth (75.3%) reported at least one trauma or instability exposure at Y1, 38.7% reported two or more, and 13.6% reported three or more. Specifically, youth on average reported 0.44 (SD = 0.82) trauma-specific experiences, with 30.9% having one or more and 7.1% having two or more traumatic experiences. Youth reported 0.97 (SD = 0.91) instability-specific experiences on average, with 64.9% having at least 1%, and 25.7% having two or more instability experiences. Trauma and instability had a small, positive correlation when accumulated over 3 years but were not significantly correlated at baseline (baseline: r = 0.10, p = .07; accumulated over 3 years: r = 0.13, p = .03).

Descriptive statistics for all CMR indicators at Y0 are provided in Table 1. Based on the eligibility criteria for the primary study [35], all youth had a baseline BMI that met criteria for overweight or obesity on average (M = 27.13, SD = 4.87). Average HDL and LDL cholesterol levels were 47.57 (SD = 11.35) and 88.79 (SD = 24.56), respectively, and were within acceptable ranges at each follow-up period (HDL >35 mg/dL and LDL <130 mg/dL) [40]. Blood pressure average values were also in the acceptable range for age at baseline and each follow-up (90th–94th percentiles confer possible risks in adolescents) [41,42]. Most triglyceride average levels were also below risk markers of 90 mg/dL (Y0 M = 82.55, SD = 48.18) [43], as was CRP (Y0 M = 0.22, SD = 0.33; >3 indicates infection or disease) [44] at each wave of data collection. Insulin sensitivity average levels were above cutoff levels for

metabolic syndrome (risk >2) [45] at baseline (Y0 $M = 4.00$, $SD = 2.40$) and each follow-up period for most adolescents.

Main results

Prospective effects of trauma and instability.—Regression models examining the independent effects of trauma and instability reported at year one show that higher instability scores were significantly associated with increased BMI ($b = 0.37$, $p < .001$), decreased HDL ($b = -0.43$, $p = .01$), and increased CRP ($b = 0.03$, $p = .001$), net of covariates. Results are shown in Table 2. Trauma reported for Y0-Y1 was not associated with any of the CMR trajectories. Associations between trauma and instability and CMR trajectories did not differ by child sex. Negatively significant associations between baseline values of the slope outcome indicate the spread of the CMR values at baseline was greater than at follow-up (i.e., the variance of CRP at baseline is greater than at follow-up).

Effects of accumulative and persistent trauma and instability exposure.—As shown in Table 3, higher accumulation of instability exposures ($b = 0.16$, $p < .001$) during years one through three were associated with positive (i.e., increases) BMI trajectories but no other CMR trajectories. Accumulative measures of trauma were not associated with any of the CMR trajectories.

Results assessing sensitivity to the accumulation of trauma and instability exposure over 3 years by comparing those with two or more exposures and those with three or more exposures in each of the three years to those with fewer exposures are shown in Table 4. Having 3+ instability experiences in each of the 3 years was associated with higher BMI slope ($b = 0.53$, $p = .001$), higher CRP ($b = 0.05$, $p = .01$), and lower HDL ($b = -0.88$, $p = .01$), compared to those with fewer than three instability experiences each year for 3 years. We also found having two or more traumatic exposures in each of the 3 years was independently significantly associated with the increasing slope of triglycerides ($b = 0.007$, $p = .004$).

Discussion

Results indicate that negative impacts of adversity on CMR may primarily be driven by exposure to instability rather than the traumatic forms of adversity included in our study. Both prospective and longitudinal indicators of instability-specific adversity were negatively associated with BMI, HDL, and CRP trajectories. Unexpectedly, while traumatic exposures were negatively associated with triglyceride levels when exposed to two or more traumas, this domain of adversity was not significantly associated with any other CMR outcomes.

Contrary to much of the existing literature, higher trauma scores were not significantly associated with most markers of adolescent CMR. Differences may be explained in part by the age of adolescents, such that impacts of trauma-specific adversity on CMR may not take effect until later, settling in during young adulthood and adulthood [2]. Findings also support the idea that children are highly resilient and can and often do survive high levels of trauma without negative impacts on CMR. Specifically, risk may not be incurred until youth are older and exposure levels are very high (i.e., over four exposures) [1]. The

lack of measures of other forms of interpersonal trauma, such as abuse or neglect, may also help explain null findings. Of the few known studies examining adversity and CMR factors during childhood, both use expansive measures of adverse or stressful childhood environments that capture elements of both trauma and instability as a single measure. For example, impacts of psychosocial stress on higher BMI z-score were found by age 10 years [46], and more negative life events, assessed as over 70 items representing varying degrees of parent and family physical or mental health and well-being, increased risk of being overweight by age 15 years [4]. This suggests that early indicators of CMR may be driven or exacerbated by instability, rather than accumulated exposure to more acute forms of trauma measured in the present study (i.e., badly hurt/sick, arrested, victim of crime, or death of family/friend). Findings should be interpreted in the context of measurement limitations, as the measure of trauma in the present study is limited to six items. Other existing measures of trauma and adversity vary widely across studies (e.g., from 10 to 70 possible types of stressful life events) [2,4–6,47]. Consistency in measurement is needed to better assess how the present study findings relate to existing research.

In our study, higher levels of instability and specifically three or more instability experiences each year over a 3-year period, were associated with increases in BMI and CRP and decreases in HDL trajectories over the same time period. Consistent with prior research, these findings suggest that the more chronic the exposure to destabilizing instability experiences, the stronger the likely effect on adverse CMR outcomes [2,4,25,48]. Our hypothesis was confirmed, suggesting long-term stress activation is more likely to occur in children experiencing recurrent instability particularly in both home and community settings and in the occurrence of ongoing unpredictability [49]. However, because the percentage of those experiencing 3+ adverse exposures was small, a larger sample may be necessary to detect a difference at higher levels of exposure. Exposures that create environments of instability, such as changing schools, fear of crime in the neighborhood, or a change in household composition (i.e. parental separation or divorce), can negatively impact adolescent CMR. It is also possible that instability contributes to the lack of material resources, which in turn can impact health behaviors that contribute to CMR. When the physical and emotional stress of instability is chronic and families face constant unpredictability in basic needs or safety, the long-term risks of toxic stress, including allostatic overload, and it is “wear and tear” on the body’s organ systems increase [50]. If the stress response from instability exposure is activated during the early adolescent stage or experienced chronically over the course of adolescence, the effects of toxic stress are more likely to take effect on the body and increase specific aspects of CMR.

Strengths and limitations

The use of eight indicators of CMR allows for assessment of how the distribution of health disparities can differ by CMR factor and take different trajectories over time, providing detailed information on the impacts of two distinct forms of adversity on different markers of health in a high-risk population [27]. Based on the study findings, BMI could also be a mediator of the association between adversity exposure and other CMR outcomes. However, even though BMI is the most common metric for assessing weight status, the measure has many limitations including the inability to distinguish between muscle and fat or to

account for body fat distribution, in addition to the over-reliance on categories with arbitrary cut-points (underweight, normal weight, overweight, and obesity) [51]. A strength of the present study is the inclusion of multiple markers of CMR rather than reliance on a single indicator such as BMI [28].

Exposures were not weighted due to mixed prior research on which aspects of these novel measures are more impactful in the association with CMR outcomes. Future research should examine the reliability and validity of item-level exposures within each trauma and instability dimension to better assess differences in single-type exposures on study outcomes, as well as individual perceptions, to determine how exposures should be weighted. Although study measures of trauma and instability may not encompass all aspects of what constitutes adversity among high-risk youth, the dimensional characterization of adversity into trauma and instability provides an important starting point for distinguishing effects of distinct forms of adversity on CMR during adolescence. The data collected on youth gender was limited to male/female and may not capture the full range of gender identities represented by participants. Our study was further limited in its ability to capture the impacts of discrimination and acculturation as important and distinct forms of adversity that may help explain the underlying mechanisms of the adversity-CMR correlations that exist for U.S. racial and ethnic minority populations. Future research needs larger samples with more diversity to account for race and ethnicity. Due to the number of hypotheses and CMR outcomes tested, there is an increased risk for type 1 error (i.e., finding a false-positive result) [52]. However, alpha-adjustment for multiple comparisons reduces the significance value to very stringent levels and increases the chances of a type 2 error [53]. Instead, examination of effect sizes and confidence intervals is recommended for interpretation beyond statistical significance alone, and present study findings should be taken in consideration with the broader evidence-base (i.e., not relying on results of this single study to draw conclusions), particularly because constructs are novel (i.e., instability) [53]. Lastly, the study was a secondary analysis of data from an intervention study which could impact results even though the intervention did not significantly effect CMR [30]. Possible effects of participation in intervention groups were accounted for in study control variables.

This is the first study to our knowledge to document associations between instability on adolescent CMR, specifically among overweight and obese youth during early adolescence. Most existing research uses retrospective reports of the first 18 years of life or used cross-sectional designs, not accounting for the effects of adversity during sensitive periods of development in early adolescence or the degree of adversity that can be experienced over a single year in high-risk contexts. High study retention (over 90%) and the longitudinal design of the present study further enhances the robustness of the findings, while taking into consideration that causal inferences cannot be drawn from the study design and the study sample was recruited from a Midwestern, city which may limit generalizability.

Conclusions

Surprisingly, environments of instability (i.e., parent separation/divorce, changed schools, moved, food insecurity, fear of crime) rather than experiences of trauma (i.e., badly hurt/

sick, arrested, victim of crime, death of family/friend) were found to be more impactful targets of CMR prevention, specifically during the early adolescent stage. However, environments of instability most often go undetected, even when adverse experiences are screened for routinely. Based on study findings, next steps for research are to examine whether instability may mediate the association between trauma and CMR, as well as other potential mediating and moderating mechanisms across the child's social ecology, such as parenting factors, parenting stress, or other aspects of the health environment (e.g., neighborhood, school, peer groups, family) that can help elucidate the complex pathways from dimensional adversity exposure to better health outcomes [54]. Future research is needed on what can be done to reduce exposure to and impacts of instability, such as programs designed to support families experiencing changes in housing, schools, or personal finances. Including measures of instability in assessments of social risk in pediatric health settings [55] may assist with identification of needs and referrals to available resources, and ultimately buffers the effects of such stressors on cardiometabolic health. Importantly, an examination of factors that can interrupt the path from adversity to CMR in high-risk groups will be an essential next step in addressing disparities and promoting health across the lifespan.

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IMPLICATIONS AND CONTRIBUTION

Findings indicate that like traumatic events (e.g., victim of crime, death of family/friend), instability (e.g., parent separation, changing schools, moving) can impact adolescent cardiometabolic health. Because environments of instability are less visible and often undetected, support of families experiencing such changes in housing, schools, and household structure is needed.

Table 1

Sample characteristics and descriptive statistics (n = 360)

Sample characteristics (n = 360)	n/M	%/SD
Demographics		
Adolescent sex (female)	208	57.8%
Adolescent age	11.56	0.57
Race/ethnicity		
Non-Hispanic Black	276	76.7%
Hispanic	59	16.4%
Non-Hispanic White	14	3.9%
"Other"	11	3.0%
Adolescent BMI percentile	95.69	3.72
Parent education		
<High school	65	18.1%
High school degree	101	28.1%
Some college/college degree	194	53.9%
Trauma and instability		
Y1–Y3 trauma 0–8	1.17	1.50
2+ exposures	80	28.4%
3+ exposures	39	13.8%
Y1–Y3 instability 0–8	1.89	1.64
2+ exposures	195	54.2%
3+ exposures	113	31.4%
CMR factors at baseline		
BMI		
85th–94th percentile	111	30.8%
95th percentile or higher	249	69.2%
Cholesterol–HDL (mg/dL)		
Normal (>45)	174	55.1%
Borderline (40–45)	62	19.6%
Too low (<40)	80	25.3%
Cholesterol–LDL (mg/dL)		
Normal (<110)	271	85.8%
Borderline (110–129)	45	12.5%
Blood pressure		
Normal	265	90.6%
Prehypertensive	74	5.6%
Stage 1 hypertension	18	3.9%
Triglycerides		
Normal (<90 mg/dL)	222	61.7%
Borderline (90–<130 mg/dL)	45	12.5%
Too high (>130 mg/dL)	49	13.6%
C-reactive Protein (mg/dL)		
Within range (<1.04)	304	96.2%
At risk (1.04 or higher)	12	3.8%
Insulin sensitivity		
Not at risk (≤2)	11	3.5%

Sample characteristics (*n* = 360)	n/M	%/SD
At risk (>2)	305	96.5%

BMI = body mass index; CMR = cardiometabolic risk; HDL = high-density lipoprotein; LDL = low-density lipoprotein.

Table 2

Prospective associations baseline trauma and instability indices with CMR trajectories

	b	SE	β	95% CI	*P*
Outcome: BMI slope (kg/m^2)					
Trauma	0.09	0.09	0.06	−0.08, 0.26	.30
Instability	0.37	0.08	0.27	0.22, 0.52	**<.001*****
Intervention 1	−0.03	0.18	−0.01	−0.38, 0.31	.85
Intervention 2	0.15	0.17	0.06	−0.19, 0.49	.39
Child age[a]	−0.01	0.01	−0.05	−0.03, 0.01	.36
Child sex[b]	−0.20	0.14	−0.08	−0.48, 0.09	.18
<High school[c]	−0.13	0.19	−0.04	−0.51, 0.25	.49
High school	−0.37	0.17	−0.13	−0.70, −0.04	**.03***
Baseline[d]	0.01	0.02	0.05	−0.02, 0.04	.41
R^2 = 0.11	*F*(df) = 3.92 (9, 309)				**<.001*****
Outcome: HDL Slope (mg/dL)					
Trauma	−0.08	0.20	−0.02	−0.48, 0.32	.70
Instability	−0.43	0.17	−0.13	−0.77, −0.09	**.01***
Intervention 1	−0.29	0.39	−0.04	−1.05, 0.48	.46
Intervention 2	−0.58	0.38	−0.09	−1.33, 0.17	.13
Child age[a]	0.01	0.02	0.02	−0.04, 0.06	.72
Child sex[b]	−1.26	0.32	−0.21	−1.89, −0.63	**<.001*****
<High school[c]	−0.62	0.42	−0.08	−1.45, 0.21	.15
High school	0.56	0.37	0.08	−0.18, 1.29	.14
Child BMI	−0.05	0.03	−0.08	−0.11, 0.02	.15
Baseline[d]	−0.11	0.01	−0.40	−0.13, −0.08	**<.001*****
R^2 = 0.24	*F*(df) = 9.10(10, 298)				**<.001*****
Outcome: LDL Slope (mg/dL)					
Trauma	−0.64	0.50	−0.07	−0.82, 2.94	.20
Instability	0.43	0.42	0.05	−0.40, 1.26	.32

	b	SE	β	95% CI	P
Intervention 1	1.06	0.96	0.07	−0.82, 2.94	.27
Intervention 2	−0.42	0.94	−0.03	−2.27, 1.43	.66
Child age[a]	−0.06	0.06	−0.05	−0.17, 0.06	.35
Child sex[b]	−2.13	0.79	−0.15	−3.69, −0.57	**.01** *
<High school[c]	−0.19	1.04	−0.01	−2.23, 1.86	.86
High school	−0.66	0.92	−0.04	−2.46, 1.15	.48
Child BMI	0.10	0.08	0.07	−0.05, 0.24	.21
Baseline[d]	−0.12	0.02	−0.39	−0.15, −0.08	**<.001** ***
$R^2 = 0.21$	F(df) = 7.63 (10, 298)				**<.001** ***
Outcome: BP- systolic slope (%tile)					
Trauma	0.35	0.55	0.03	−0.73, 1.43	.52
Instability	0.10	0.48	0.01	−0.85, 1.05	.83
Intervention 1	0.60	1.09	0.03	−1.56, 2.75	.59
Intervention 2	0.39	1.07	0.02	−1.72, 2.50	.72
Child age[a]	−0.13	0.07	−0.10	−0.27, 0.002	.05
Child sex[b]	1.83	0.91	0.11	0.05, 3.61	.04
<High school[c]	0.33	1.20	0.02	−2.03, 2.69	.78
High school	−1.96	1.05	−0.10	−4.01, 0.10	.06
Child BMI	0.30	0.09	0.18	0.13, 0.47	**.001** **
Baseline[d]	−0.16	0.02	−0.46	−0.20, −0.13	**<.001** ***
$R^2 = 0.23$	F(df) = 8.70(10, 309)				**<.001** ***
Outcome: BP- diastolic slope (%tile)					
Trauma	0.53	0.45	0.06	−0.37, 1.42	.25
Instability	0.45	0.40	0.06	−0.33, 1.23	.26
Intervention 1	0.33	0.91	0.02	−1.45, 2.12	.71
Intervention 2	−0.51	0.89	−0.03	−2.25, 1.23	.57
Child age[a]	−0.04	0.06	−0.04	−0.15, 0.07	.45
Child sex[b]	−1.65	0.75	−0.11	−3.12, −0.18	**.03** *
<High school[c]	0.53	0.99	0.03	−1.43, 2.48	.60

	b	SE	β	95% CI	*P*
High school	−1.94	0.86	−0.11	−3.64, −0.24	**.03***
Child BMI	0.24	0.07	0.17	0.09, 0.38	**.001****
Baseline[d]	−0.21	0.02	−0.56	−0.24, −0.17	**<.001*****
$R^2 = 0.31$	*F*(df) = 13.57 (10, 309)				**<.001*****
Outcome: triglycerides slope (mg/dL)					
Trauma	1.32	1.01	0.06	−0.67, 3.31	.19
Instability	1.54	0.85	0.09	−0.13, 3.20	.07
Intervention 1	−1.37	1.92	−0.04	−5.14, 2.41	.48
Intervention 2	−1.44	1.88	−0.04	−5.14, 2.27	.45
Child age[a]	0.12	0.12	0.05	−0.12, 0.36	.31
Child sex[b]	3.03	1.58	0.09	−0.07, 6.14	.06
<High school[c]	0.51	2.10	0.01	−3.62, 4.64	.81
High school	0.09	1.84	0.002	−3.54, 3.72	.96
Child BMI	0.06	0.15	0.02	−0.24, 0.36	.70
Baseline[d]	−0.19	0.02	−0.56	−0.22, −0.16	**<.001*****
$R^2 = 0.36$	*F*(df) = 15.88 (10, 298)				**<.001*****
Outcome: CRP slope (mg/dL)					
Trauma	−0.01	0.01	−0.05	−0.03, 0.01	.33
Instability	0.03	0.01	0.18	0.01, 0.04	**.001****
Intervention 1	0.001	0.02	0.004	−0.03, 0.04	.95
Intervention 2	0.01	0.02	0.04	−0.02, 0.05	.48
Child age[a]	−0.0002	0.001	−0.01	−0.002, 0.002	.85
Child sex[b]	−0.01	0.02	−0.03	−0.04, 0.02	.56
<High school[c]	0.01	0.02	0.01	−0.03, 0.04	.79
High school	−0.04	0.02	−0.13	−0.08, −0.01	**.01***
Child BMI	0.004	0.002	0.15	0.001, 0.01	**.02***
Baseline[d]	−0.18	0.03	−0.44	−0.23, −0.13	**<.001*****
$R^2 = 0.23$	*F*(df) = 8.76(10, 298)				**<.001*****

	b	SE	β	95% CI		P
Outcome: insulin sensitivity slope (mg/dL)						
Trauma	0.05	0.09	0.03	−0.12, 0.22		.56
Instability	0.002	0.07	0.001	−0.14, 0.15		.98
Intervention 1	0.05	0.17	0.02	−0.27, 0.38		.74
Intervention 2	0.003	0.16	0.001	−0.32, 0.32		.98
Child age[a]	−0.02	0.01	−0.10	−0.04, 0.001		.07
Child sex[b]	0.10	0.14	0.04	−0.17, 0.38		.46
<High school[c]	−0.19	0.18	−0.06	−0.55, 0.16		.28
High school	0.02	0.16	0.01	−0.29, 0.33		.91
Child BMI	0.03	0.01	0.14	0.01, 0.06		**.02***
Baseline[d]	−0.25	0.03	−0.47	−0.31, −0.19		**<.001*****
$R^2 = 0.21$	$F(df) = 7.82\ (10, 298)$					**<.001*****

Boldface indicates statistical significance

* $p < .05$

** $p < .01$

*** $p < .001$.

BMI = body mass index; CMR = cardiometabolic risk; HDL = high-density lipoprotein; CRP = C-reactive protein.

[a] Child age measured in months.

[b] Child sex reference group is male.

[c] Education reference group is college education or higher.

[d] Baseline indicator of the health outcome measured (e.g., baseline [Year 0] BMI when BMI was the outcome).

Table 3

Regression of CMR trajectories on 3-year accumulative indices (Y1-Y3) of trauma and instability

	b	SE	β	95% CI	*P*
Outcome: BMI slope (kg/m^2)					
Trauma	−0.04	0.05	0.22	−0.14, 0.05	.40
Instability	0.16	0.05	−0.05	0.08, 0.25	**<.001*****
Intervention 1	−0.04	0.18	−0.02	−0.38, 0.31	.82
Intervention 2	0.16	0.17	0.06	−0.17, 0.50	.34
Child age[a]	−0.01	0.01	−0.05	−0.03, 0.01	.45
Child sex[b]	−0.16	0.15	−0.07	−0.45, 0.13	.27
<High school[c]	−0.17	0.20	−0.05	−0.55, 0.21	.38
High school	−0.28	0.17	−0.10	−0.61, 0.05	.10
Baseline[d]	0.02	0.02	0.07	−0.01, 0.05	.22
R^2 = 0.08	*F*(df) = 2.65 (9, 281)				**<.001*****
Outcome: HDL slope (mg/dL)					
Trauma	−0.14	0.11	−0.07	−0.08, 0.35	.21
Instability	−0.14	0.10	−0.08	−0.33, 0.06	.18
Intervention 1	−0.16	0.39	−0.03	−0.92, 0.61	.69
Intervention 2	−0.36	0.38	−0.60	−1.11, 0.39	.35
Child age[a]	−0.001	0.03	−0.002	−0.05, 0.05	.97
Child sex[b]	−1.21	0.32	−0.21	−1.85, −0.58	**<.001*****
<High school[c]	−0.37	0.43	−0.05	−1.22, 0.48	.39
High school	0.21	0.38	0.03	−0.54, 0.95	.59
Child BMI	−0.04	0.03	−0.07	−0.10, 0.02	.21
Baseline[d]	−0.10	0.02	−0.39	−0.12, −0.07	**<.001*****
R^2 = 0.20	*F*(df) = 6.50 (10,272)				**<.001*****
Outcome: LDL slope (mg/dL)					
Trauma	−0.38	0.27	−0.080	−0.92, 0.15	.16
Instability	0.19	0.25	0.043	−0.31, 0.68	.46

	b	SE	β	95% CI	P
Intervention 1	0.69	0.98	0.04	−1.24, 2.62	.48
Intervention 2	−0.24	0.96	−0.01	−2.14, 1.65	.80
Child age[a]	−0.04	0.06	−0.04	−0.17, 0.08	.48
Child sex[b]	−1.56	0.83	−0.10	−3.19, 0.07	.06
<High school[c]	−0.09	1.08	−0.01	−2.22, 2.04	.93
High school	−0.71	0.96	−0.04	−2.59, 1.18	.46
Child BMI	0.14	0.08	0.10	−0.01, 0.29	.07
Baseline[d]	−0.11	0.02	−0.38	−0.15, −0.08	**<.001**[***]
$R^2 = 0.19$	$F(df) = 5.97$ (10,272)				**<.001**[***]
Outcome: BP- systolic slope (%tile)					
Trauma	0.18	0.30	0.03	−0.41, 0.76	.56
Instability	0.26	0.28	0.05	−0.28, 0.81	.35
Intervention 1	0.42	1.07	0.02	−1.69, 2.53	.70
Intervention 2	0.68	1.05	0.04	−1.38, 2.74	.52
Child age[a]	−0.12	0.07	−0.10	−0.25, 0.01	.08
Child sex[b]	2.27	0.90	0.14	0.50, 4.04	**.01**
<High school[c]	0.45	1.20	0.02	−1.90, 2.81	.71
High school	−1.06	1.03	−0.06	−3.08, 0.97	.31
Child BMI	0.33	0.08	0.22	0.17, 0.49	**<.001**[***]
Baseline[d]	−0.16	0.02	−0.47	−0.19, −0.12	**<.001**[***]
$R^2 = 0.24$	$F(df) = 8.57$ (10, 281)				**<.001**[***]
Outcome: BP- diastolic slope (%tile)					
Trauma	0.13	0.23	0.03	−0.33, 0.58	.59
Instability	0.18	0.22	0.04	−0.25, 0.60	.42
Intervention 1	0.59	0.85	0.04	−1.08, 2.25	.49
Intervention 2	−0.03	0.82	−0.002	−1.65, 1.59	.97
Child age[a]	−0.03	0.05	−0.03	−0.13, 0.08	.60
Child sex[b]	−1.24	0.71	−0.09	−2.63, 0.15	.08
<High school[c]	0.71	0.94	0.04	−1.15, 2.57	.45

	b	SE	β	95% CI	*P*
High school	−1.15	0.81	−0.08	−2.75, 0.46	.16
Child BMI	0.26	0.07	0.21	0.13, 0.39	**<.001** ***
Baseline[d]	−0.18	0.02	−0.54	−0.21, −0.14	**<.001** ***
R^2 = 0.28	*F*(df) = 10−0.39 (10, 281)				**<.001** ***
Outcome: triglycerides slope (mg/dL)					
Trauma	−0.01	0.01	−0.05	−0.03, 0.01	.35
Instability	−0.01	0.02	−0.06	−0.06, 0.04	.68
Intervention 1	−2.05	1.85	−0.06	−5.69, 1.59	.27
Intervention 2	−1.90	1.81	−0.06	−5.47, 1.67	.30
Child age[a]	0.22	0.12	0.10	−0.01, 0.46	.06
Child sex[b]	4.48	1.54	0.14	1.44, 7.51	**.004** **
<High school[c]	−0.52	2.06	−0.01	−4.57, 3.54	.80
High school	−0.18	1.80	−0.01	−3.72, 3.54	.92
Child BMI	0.05	0.14	0.02	−0.24, 0.33	.76
Baseline[d]	−0.17	0.02	−0.54	−0.20, −0.14	**<.001** ***
R^2 = 0.35	*F*(df) = 14.15 (10, 272)				**<.001** ***
Outcome: C-reactive protein slope (mg/dL)					
Trauma	−0.01	0.01	−0.05	−0.03, 0.01	.35
Instability	−0.01	0.02	−0.06	−0.06, 0.04	.68
Intervention 1	−0.0001	0.02	<0.001	−0.04, 0.04	.997
Intervention 2	0.01	0.02	<0.001	−0.02, 0.05	.52
Child age[a]	−0.0001	0.001	−0.01	−0.002, 0.002	.87
Child sex[b]	−0.01	0.02	−0.03	−0.04, 0.02	.56
<High school[c]	0.005	0.02	−0.14	−0.042, 0.042	.81
High school	−0.04	0.02	−0.14	−0.08, −0.01	**.01** *
Child BMI	0.001	0.002	0.14	0.001, 0.007	**.02** *
Baseline[d]	−0.18	0.03	−0.44	−0.23, −0.13	**<.001** ***
R^2 = 0.20	*F*(df) = 6.44 (10,272)				**<.001** ***
Outcome: insulin sensitivity slope (mg/dL)					

	b	SE	β	95% CI	P
Trauma	0.001	0.05	0.002	−0.09, 0.09	.98
Instability	−0.01	0.04	−0.01	−0.09, 0.07	.85
Intervention 1	0.11	0.16	0.04	−0.21, 0.43	.49
Intervention 2	0.05	0.16	0.01	−0.27, 0.36	.77
Child age[a]	−0.01	0.01	−0.08	−0.03, 0.01	.18
Child sex[b]	0.12	0.14	0.08	−0.08, 0.47	.16
<High school[c]	−0.16	0.18	−0.05	−0.52, 0.19	.37
High school	0.01	0.16	0.003	−0.30, 0.32	.95
Child BMI	0.02	0.01	0.11	−0.002, 0.05	.07
Baseline[d]	−0.21	0.03	−0.44	−0.27, −0.16	**<.001*****
$R^2 = 0.20$	$F(df) = 6.66\ (10,272)$				**<.001*****

Boldface indicates statistical significance

* $p < .05$

** $p < .01$

*** $p < .001$.

BMI = body mass index; CMR = cardiometabolic risk; HDL = high-density lipoprotein.

[a] Child age measured in months.

[b] Child sex reference group is male.

[c] Education reference group is college education or higher.

[d] Baseline indicator of the health outcome measured (e.g., baseline [Year 0] BMI when BMI was the outcome).

Table 4

Regression of CMR trajectories on 2+ or 3+ trauma & instability yearly over 3 years

	b	SE	β	95% CI	*p*
BMI (kg/m^2)					
Trauma 2+	<0.001	<0.001	0.05	<0.001, 0.001	.36
Instability 2+	0.22	0.14	0.09	−0.05, 0.49	.11
R^2 = 0.05					
F(df) = 1.80 (9, 345), p = .07					
Trauma 3+	−0.22	0.21	−0.06	−0.63, 0.19	.28
Instability 3+ **	0.53	0.16	0.20	0.21, 0.85	**.001** **
R^2 = 0.07					
F(df) = 9.08 (9, 281), p = .03 *					
HDL (mg/dL)					
Trauma 2+	−0.001	<0.001	−0.08	−0.002, <0.001	.14
Instability 2+	−0.37	0.31	−0.06	−0.97, 0.24	.24
R^2 = 0.23					
F(df) = 8.94 (10, 315), $p \leq .001$ ***					
Trauma 3+	0.75	0.46	0.09	−0.15, 1.65	.10
Instability 3+ *	−0.88	0.36	−0.14	−1.59, −0.18	**.01** *
R^2 = 0.22					
F(df) = 7.42 (10, 272), $p < .001$ ***					
LDL (mg/dL)					
Trauma 2+	−1.76	0.90	−0.11	−3.54, 0.02	.05
Instability 2+	−0.17	0.81	−0.01	−1.76, 1.42	.83
R^2 = 0.19					
F(df) = 6.18 (10, 272), $p < .001$					
Trauma 3+	−0.75	1.17	−0.04	−3.06, 1.56	.52
Instability 3+	0.24	0.91	0.02	−1.55, 2.04	.79
R^2 = 0.18					
F(df) = 5.74 (10, 272), $p < .001$ ***					

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	b	SE	β	95% CI	*p*
BP- Systolic (%tile)					
Trauma 2+	0.002	0.001	0.08	<0.001, 0.004	.09
Instability 2+	0.03	0.84	0.002	−1.63, 1.68	.97
R^2 = 0.25					
F(df) = 11.19 (10, 344), *p* < .001					
Trauma 3+	1.09	1.27	0.05	−1.41, 3.60	.39
Instability 3+	0.06	1.00	0.003	−1.91, 0.01	.96
R^2 = 0.24					
F(df) = 8.48 (10, 281), *p* < .001***					
BP- Diastolic (%tile)					
Trauma 2+	0.001	0.001	0.05	−0.001, 0.003	.28
Instability 2+	−0.001	0.71	<0.001	−1.40, 1.39	1.00
R^2 = 0.31					
F(df) = 15.33 (10, 344), *p* < .001					
Trauma 3+	0.59	1.002	0.03	−1.38, 2.56	.56
Instability 3+	0.13	0.79	0.01	−1.42, 1.68	.87
R^2 = 0.28					
F(df) = 10.30(10, 281), *p* < .001***					
Triglyceride					
Trauma 2+**	0.007	0.002	0.14	0.002, 0.01	**.004****
Instability 2+	1.03	1.60	0.03	−2.13, 4.18	.52
R^2 = 0.31					
F(df) = 13.47 (10, 315), *p* < .001					
Trauma 3+	− 0.51	2.20	−0.01	−4.84, 3.83	.82
Instability 3+	2.83	1.71	0.09	−0.54, 6.19	.10
R^2 = 0.35					
F(df) = 14.36 (10, 272), *p* < .001					
CRP					
Trauma 2+	−0.03	0.02	−0.11	−0.07, 0.001	.06
Instability 2+	0.02	0.02	0.06	−0.02, 0.05	.34

	b	SE	β	95% CI	*p*
R^2 = 0.19					
F(df) = 6.26 (10, 272), *p* < .001					
Trauma 3+	− 0.01	0.02	−0.11	−0.05, 0.04	.73
Instability 3+ *	0.05	0.02	0.15	0.01, 0.08	**.01** *
R^2 = 0.20					
F(df) = 6.53 (10, 272), *p* < .001					
Insulin sensitivity					
Trauma 2+	<0.001	<0.001	0.09	<0.001, 0.001	.07
Instability 2+	0.05	0.13	0.02	−0.21,0.30	.71
R^2 = 0.23					
F(df) = 9.08 (10, 315), *p* < .001					
Trauma 3+	−0.054	0.19	−0.02	−0.44, 0.33	.78
Instability 3+	0.11	0.15	0.04	−0.19, 0.40	.48
R^2 = 0.20					
F(df) = 6.72 (10, 272), ***p*< .001** ***					

All models adjusted for covariates: parent education level, child age, child sex, child BMI, baseline outcome indicator, participation in intervention study and child sex. Parameter estimates for covariates are available upon request.

Boldface indicates statistical significance

* *p* < .05

** *p* < .01

*** *p* < .001.

BMI = body mass index; CMR = cardiometabolic risk; HDL = high-density lipoprotein; CRP = C-reactive protein.