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Early socioeconomic adversity and cardiometabolic risk in young adults: mediating roles of risky health lifestyle and depressive symptoms

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Abstract The study examined the mediating roles of risky health lifestyle and depressive symptoms in relation to childhood/adolescence adversity and young adult cardiometabolic risk with data from the National Longitudinal Study of Adolescent to Adult Health (n = 9421). Four classes of youth emerged from a latent class analysis with varying early adversity patterns: (a) both low disadvantaged SES and stressful experience (54.8%), (b) high disadvantaged SES and low stressful experience (31.0%), (c) low disadvantaged SES and high stressful experience (10.9%), and (d) both high disadvantaged SES and stressful experience (3.3%). Early adversity had multiple direct and indirect effects on CM risk for those experiencing SES-related adversities. Instead, early adversity generated mediational processes between adversity and CM risks through risky health lifestyle and depressive symptoms for those experiencing stressful experience. Implications for intervention when dealing with youths who have experienced multiple forms of early adversity are discussed.

Keywords Childhood/adolescence adversity · Risky health lifestyle · Depressive symptoms · Cardiometabolic risk · Latent class analysis · Path model

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Introduction

Cardiometabolic risk (hereafter referred as CM risk) is considered a major contributor to the leading causes of death in the United States (Xu et al., 2014) and, relatedly, a risk factor for early mortality (Saydah et al., 2013). Past studies have consistently reported that adversity in childhood and adolescence (hereafter referred as early adversity) is strongly linked to CM risk in adulthood. However, less is known about the influence of specific types of early adversity and the mediating effects of risky health lifestyle and depressive symptoms on CM risk. Identifying distinct adversity groups and understanding the longitudinal mechanisms linking early adversity to health risk in adulthood has implications for the design of successful interventions to reduce CM risk.

Direct effects of early adversity on young adult CM risk

The long-term effects of early adversity on young adult CM risk is expected to exist across two aspects of early adversity, which we conceptualize as (1) *disadvantaged socioeconomic status* (SES) reflected by multiple SES markers and (2) *stressful life circumstances* measured by several specific experiences. Numerous studies have found that elements of disadvantaged SES, including family economic hardship, low parental education, parents' marital status, and adverse community characteristics impact youth's CM risk (Burdette & Needham, 2012; Shin & Miller, 2012). For instance, adolescents from low income families often lack health resources, such as proper housing, clean environment, affordable health insurance, access to health care, and prevention programs as well as parental health knowledge and information necessary for healthy

child rearing (Wen & Maloney, 2011). A large body of evidence implicates poverty in childhood/adolescence as a strong predictor of later CM risks (Cohen et al., 2010).

Instead, child/adolescent stressful life experiences, such as parental abuse and social negligence, can generate negative emotions for youth, and these emotions generally invoke physiological processes (McEwen, 1998). Over time, these stress responses lead to consistent activation of the hypothalamic-pituitary-adrenocortical (HPA) axis and the sympathetic-adrenal medullary (SAM) system with increased magnitude and duration in the physiological responses (McEwen, 2012). Such conditions are implicated in the development of CM risk through physiological dysregulation, including exaggerated stress hormones, altered insulin sensitivity, increased blood pressure, and inflated central adiposity as well as elevated inflammation (McEwen, 2012; Miller et al., 2011).

Previous studies have consistently suggested multiplicative, or joint effects of these two distinct types of adversity on health outcomes (Doom et al., 2017; Kittleson et al., 2006; Miller et al., 2011). Although the long-term effects of early adversity on CM risks in young adulthood is well documented (e.g., Wickrama et al., 2015a), less is known about multiplicative effects of two distinct adversities (SES disadvantages and stressful experiences) on CM risk in young adulthood. The main purpose of this study is to identify the distinct patterns of early adversity and how these distinct patterns are related to CM risk in young adulthood.

Multiple mediating effects of early adversity on CM risk

Multiple mediation processes may explain long-lasting effects of early adversities on subsequent CM risk, including behavioral and psychological mechanisms (Wickrama et al., 2013). In the stress paradigm, risky health behaviors such as smoking, overeating, and inactivity represent forms of behavioral coping mechanisms with stress (Pampel et al., 2010). Studies show that health risk behaviors are seen as major contributors to CM risk as reflected by elevated levels of biomarkers such as BMI, blood pressure, heart rate, HbA1C, cholesterol (Anand et al., 2015). Poor eating and sedentary behaviors have been shown to have pro-inflammatory effects (Stringhini et al., 2013), which also result in elevated levels of biomarkers, particularly high BMI (Nazmi et al., 2009). Given that health-related behaviors in adolescence are likely to persist into adulthood (Telama et al., 1997), we expect that a risky health lifestyle from adolescence to young adulthood will have a cumulative detrimental influence on CM risk over time (Anand et al., 2015). In addition, early adversities may influence CM risk through psychological distress. Compared to other youth, those who are exposed to early adversities encounter more psychological distress (i.e., depressive symptoms) during adolescence (Guerry & Hastings, 2011). The link between depressive symptom from adolescence to young adulthood and CM risk in adulthood is well documented (Wickrama et al., 2014).

These two mediational pathways (i.e., risky health lifestyle and depressive symptoms) imply that risky health lifestyle and depressive symptoms independently function as parallel mediation processes leading to CM risk in young adulthood. However, given the positive associations between risky health behaviors and depressive symptoms (Pampel et al., 2010), these two mediational pathways may also be connected, suggesting additional sequential processes *between* these two mediational pathways. For example, early adversities lead to increased psychological distress (i.e., depressive symptoms), such as feeling trapped, angry, hopeless, and frustrated (Prawitz et al., 2013). Adolescents' risky health behaviors (e.g., substance use, poor eating habits, and physical inactivity) may reflect an externalized reaction, or coping mechanism, to this negative internal process (Krueger & Chang, 2008). Taken together, we expect mutual associations between adolescents' risky health lifestyle and depressive symptoms over time leading to the production of parallel and sequential mediational processes linking early adversities and CM risk in young adulthood.

Distinct longitudinal mechanisms of early adversity on CM risk

Previous studies suggest distinct longitudinal mechanisms linking types of early adversity and CM risk (Doom et al., 2017). For example, in terms of SES-related disadvantages, family SES reflects the family structure, which is fairly stable over the life course (Miller et al., 2011), and typically has persistent effects on later health outcomes (Doom et al., 2017). That is, low-SES families are a chronic stressor for youth with persistent effects on their CM risk in later adulthood (Cohen et al., 2010). Moreover, because childhood/adolescence is a sensitive period in the life course, experiencing SES disadvantage in this life stage can have strong effects on health (Miller et al., 2011). A recent study suggest that the health risks associated with low SES in adolescence persist even when people experience upward social mobility (Wickrama et al., 2016). In this vein, we expect that SES-related adversities will have relatively long-term direct effects on CM risk in young adulthood even after adjusting for the multiple mediating effects of health risk behaviors and depressive symptoms.

In contrast, stressful circumstances such as sexual-, physical-, and/or emotional-abuse emphasize individual

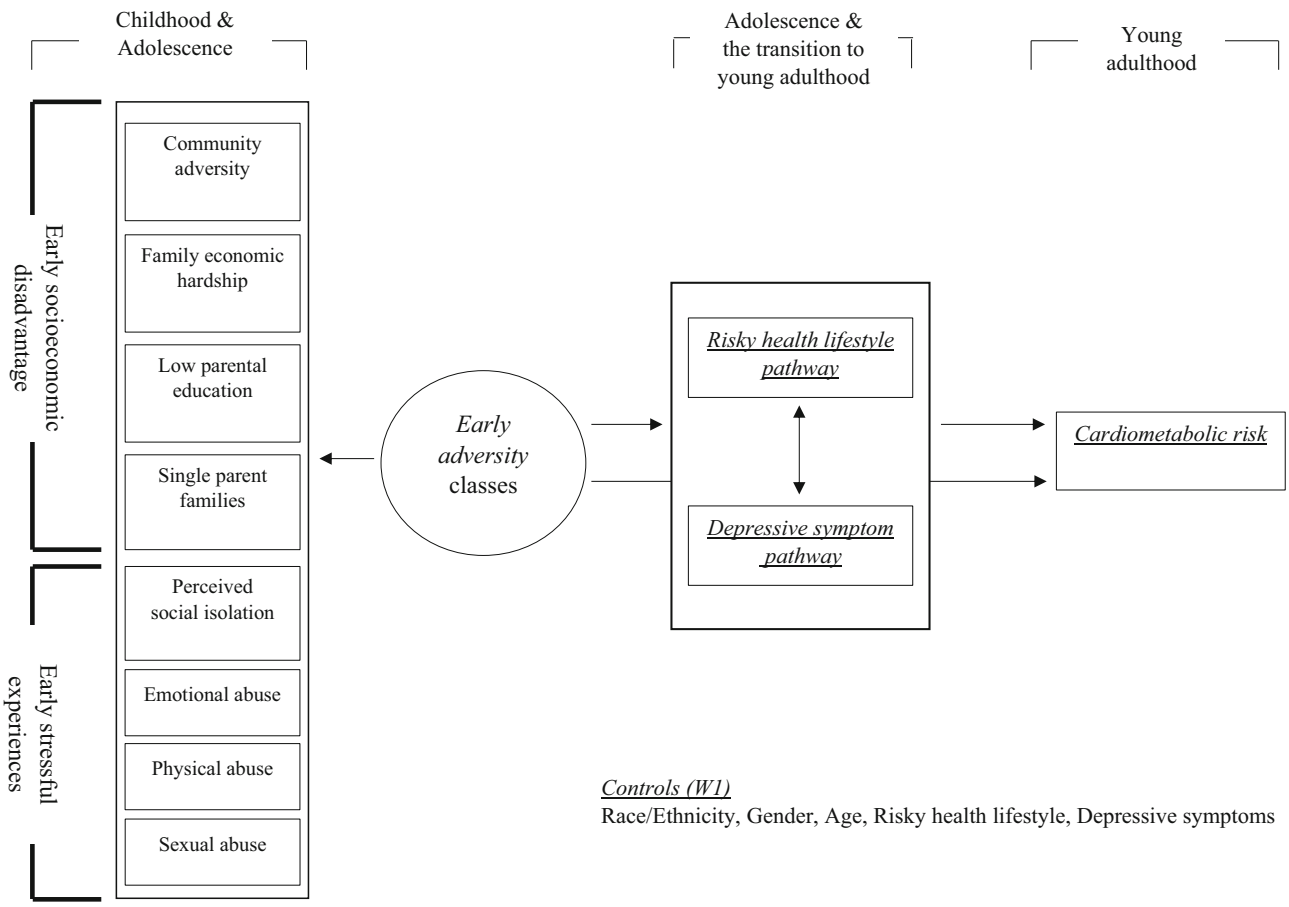

Fig. 1 Hypothesized model

experiences (Doom et al., 2017). These early stressful experiences may generate more short-term detrimental outcomes (e.g., negative parent–child relationship, conduct problems, and substance use), which, in turn, is related to elevated CM risk later in life (Doom et al., 2017). These proximal effects emphasize multiple mediational pathways linking early stressful experiences to health outcomes in adulthood, rather than persistent effects of early adversity itself. Taken together, we expect distinct longitudinal mechanisms operate between early disadvantaged SES and stressful experiences. The overall hypothesized model is depicted in Fig. 1.

Methods

Sample

Data for this study came from a nationally representative sample of adolescents participating in the National Longitudinal Study of Adolescent to Adult Health (Add Health). At Wave 1, 20,745 adolescents participated from a stratified, random sample of US schools and completed an in-home interview that collected data on a range of topics. A parent of the participant also completed an in-home interview at Wave 1. We used data from 9421 adolescents who participated in Waves 1 (1995), 2 (1996), and 3 (2001) and provided biomarker data at Wave 4 (2008). More information about Add Health is available at http://www.cpc.unc.edu/addhealth.

Measures

Childhood/adolescent adversity

We defined early adversity including community/family SES adversity (referred as disadvantaged SES) and stressful life experiences in childhood and early adolescence. Using interview data from Wave 1 unless otherwise stated below, four dichotomous measures of disadvantaged SES were created. Unless otherwise noted, consistent with previous research, binary splits were created using the top 25% percentile or equivalent scores as a cutpoint (Lee et al., 2018; Horan & Widom, 2015). Specific *disadvan-*

taged SES measures include: (1) community adversity: sum of four dichotomized indicators asking about the high/low community proportions of families living in poverty, single families, adults employed in the service industry, and unemployed men; (2) family economic hardship: sum of five dichotomous items asking if any household member received economic assistance such as food stamps and housing subsidies in the past month; (3) low parental education: composite score of father's and mother's education levels (coded high school diploma or less education as 1 and others as 0); (4) single parent family: single, widowed, divorced, or separated parents were coded as 1. These socio-economic adversity items have been widely used in other studies to assess early socio-economic adversity (Brody et al., 2013; Lee et al., 2018; Wickrama et al., 2015b).

Four dichotomous measures of adolescent *stressful experiences* were also created. Three maltreatment items were used in previous study as indicators of stressful experience: emotional abuse, physical abuse, and sexual abuse (Scheidell et al., 2018). In addition, we included a measure of low social support as a proxy for social isolation (Matthews et al., 2016). A research indicates that social isolation may act as another stressful experience causing physiological dysregulation (Pugh et al., 2001), resulting in increased CM risk in young adulthood (Caspi et al., 2006). Unless otherwise stated below, dichotomous indicators were created by adapting the criteria of Scheidell et al. (2018). Therefore, *stressful experience* measures include: (1) social isolation (Wave 1; Adkins et al., 2012): 3 items assessing relationships with friends, teachers, and family (alpha = .75; responses ranged from 1 "very much" to 5 "not at all"; e.g., How much do you feel that your teachers care about you; coded the top quartile as 1); (2) emotional abuse (Wave 3): parent/adult caregiver said things that really hurt one's feelings or made one feel unloved before the age of 12 $\geq$ six times; (3) physical abuse (Wave 3): slapped, hit, kicked or thrown by a parent/adult caregiver before the age of 12 $\geq$ six times; (4) sexual abuse (Wave 3): parent/adult caregiver touching the participant or forcing the participant to touch him or her in sexual way, or forcing sexual relations before the age of 12 $\geq$ 1 time.

Risky health lifestyle

Sum scores were computed separately for Waves 1 through 3 from seven dichotomous health risk behaviors. Specific behaviors were: smoking, binge-drinking, marijuana use, improper sleep duration, unhealthy eating, physical inactivity, and sedentary behaviors. These indicators were selected to be consistent with previous research (Frech, 2012; Lawrence et al., 2017; Wickrama et al., 2017). Unless otherwise stated below, dichotomous indicators were created by adapting the criteria of Frech (2012). Then, all dichotomous indicators were summed to capture a single variable at each time point assessing overall risky health lifestyle. Each variable is described in detail below.

Regarding smoking, respondents were assigned a value of 1 if they reported using tobacco products at all in the last 30 days. Binge drinking was coded as 1 if they reported at least one instance of heavy episodic drinking (e.g., 5 or more drinks in one sitting) in the past 12 months. Marijuana use was coded as 1 if the individual reported using marijuana one or more times in the last 30 days. Respondents reported how many hours of sleep they usually get. Improper sleep was coded as 1 based on previous finding (Hirshkowitz et al., 2015; under the age of 20, outside the recommended 8–11 h; aged 20 or older outside the recommended 6–9 h). For Waves 2 and 3, unhealthy eating (Wickrama et al., 2017) was measured by a mean split of two items that assessed how often the respondents consumed fast food and skipped breakfast in the past 7 days (Scale responses ranged from 0 = no days to 7 = 7 days). At Wave 1, unhealthy eating was only measured by skipping breakfast (on a weekday morning) due to data availability (coded 1 if respondents reported "Nothing to eat or drinking"). Having breakfast is an important indicator of healthy eating (Spence, 2017). Physical inactivity was coded as 1 if the respondent reported engaging in physical activities on less than three occasions in the past week. Sedentary behavior (Lawrence et al., 2017) was coded as 1 if the respondent scored in the top quartile (around 28 + h) on a sum score of the total number of hours in the past week spent TV viewing, video viewing, and playing computer games.

Depressive symptoms

Depressive symptoms were measured at Waves 1, 2, and 3 using an abridged 9-item assessment from the Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977). Respondents indicated how often they experienced certain feelings or behaviors in the previous 7 days. Sample items include: "You were bothered by things that usually don't bother you," and "You had trouble keeping your mind on what you were doing." Responses ranged from never or rarely = 0 to most of the time or all of the time = 3. Scores were summed and averaged with higher scores reflecting more depressive symptomology (alphas were .80, .81, and .83 for Waves 1 through 3, respectively).

Young adults' cardiometabolic (CM) risk

Because biomarkers have an additive predictive value (Zakynthinos & Pappa, 2004), we measured CM risk by computing an aggregate score of ten regulatory biomarkers of cardiovascular and metabolic systems at Wave 4 (2008) for which the participant was in the highest risk quartile (Seplaki et al., 2004). This aggregated biological measure captures (a) multiple system cumulative physiological dysregulation and inflammation and (b) the longitudinal context, or weathering, over the early life course (Wickrama et al., 2015a, b). Outliers for each of the biomarker variables (z scores > 3) were removed.

The biomarkers assessed include systolic blood pressure, diastolic blood pressure, pulse rate, glycohemoglobin, glucose, triglycerides, high-density lipoprotein, low-density lipoprotein, high sensitivity C-reactive protein level, and BMI. Systolic and diastolic blood pressure (mmHg) and pulse rate measurements were taken on the right arm, absent contraindications in a rested/seated position by trained field interviewers using oscillometric blood pressure monitors. Using standard procedures, trained and certified interviewers obtained whole blood spots for dried blood analysis. From these samples, glycohemoglobin, an integrated measure of blood glucose control over the preceding 2–3 months, total glucose values, triglycerides, low-density lipoprotein, high-density lipoprotein, and high sensitivity C-reactive protein level were assayed. Trained interviewers also obtained measurements of respondents' height and weight, and this information was used to compute their BMI, the ratio of weight to height squared ($lb \times 703/in^2$).

Control variables

Our analyses also accounted for time invarying covariates, including respondents' gender (male = 0, female = 1), age (continuous), and race/ethnicity. Regarding race/ethnicity, the dichotomous variables for each of the minority statuses were included as independent variables in the regression equation resulting in regression coefficients that can be interpreted with reference to Whites.

Data analytic strategy

First, latent class analysis (LCA; McLachlan & Peel, 2000) was used to identify the unobserved distinct patterns of early adversity using the 8 indicators that we mentioned above. For the model evaluation (Wickrama et al., 2016), we used several fit indices such as sample size adjusted BIC (lower value is preferred), entropy ($\geq .70$ is acceptable), and the Lo–Mendell–Rubin Likelihood Ratio Test (LMR-LRT; significant p value indicates the k class model is better than $k - 1$ class model). Second, to investigate long-term effects of early adversity on CM risks in young adulthood, the path model was tested to estimate direct effects of early adversity (Wave 1) on CM risk (Wave 4) using class membership in LCA (step 1). Third, to investigate the parallel mediation processes, we added risky health lifestyle and depressive symptoms as mediators assessed at Waves 2 and 3 into the direct path model that we described in step 2. To investigate sequential processes, we incorporated cross-lagged associations between the mediators (risky health lifestyle and depressive symptoms) into the mediational path model.

The current study also incorporated several demographic variables such as race/ethnicity (White = reference), gender (Male = reference), and age. Also, we specified early risky health lifestyle and depressive symptoms (at Wave 1) as control variables to estimate unique mediational pathways. For model evaluation, the comparative fit index (CFI; $\geq .90$ acceptable), Tucker-Lewis Index (TLI; $\geq .90$ acceptable), and the root mean square of error approximation (RMSEA; $\leq .08$ acceptable) were used (Little, 2013).

All analyses were conducted in Mplus (version 8.00). All coefficients were adjusted by using the complex sample design (TYPE = COMPLEX method in Mplus) to take into account stratified cluster-sampling of Add Health data. Also, sample weights from Wave 4 were utilized to account for longitudinal attrition. Missing data were managed using multiple imputation for Waves 1–3 (except for the cumulative CM risk measure for which the missing rate was 1.3%). The analytical sample included only non-imputed CM risk data in Wave 4. On average, 5.9% of data was imputed.

Results

Latent class analysis

We conducted a series of LCAs by specifying two- through six-class models of early adversity and comparing all available model fit indices. Based on the results, the four-class model was selected as the optimal class model (see Table 1). The estimated proportions of the adversity indicators for each of the four classes are shown in Table 2. Around half of the youth were exposed to low SES disadvantage and also experienced a low level of stressful experiences (Class 1; $n = 5160$, 54.8%). Youth classified as Class 2 were exposed to relatively high SES disadvantage but experienced a low level of stressful experiences ($n = 2918$, 31.0%). Youth in Class 3 were generally

Table 1 Model fit indices for early adversity latent class analyses

Fit statistics	2 classes	3 classes	4 classes	5 classes	6 classes[a]
# of free parameters	17	26	35	44	53
BIC	76,955.279	75,039.416	74,886.092	74,890.734	74,918.454
SSABIC	76,901.256	74,956.793	74,774.868	74,750.909	74,750.028
Entropy	0.542	0.668	0.700	0.640	0.641
Adj. LMR-LRT (p value)	2461.751 ($p < .001$)	1974.247 ($p < .001$)	232.853 (p < .001)	76.782 ($p < .05$)	53.981 ($p < .001$)
Group size (n, %)					
Class 1	3283, 34.8%	1145, 12.0%	320, 3.3%	489, 5.2%	264, 2.8%
Class 2	6138, 65.2%	2814, 30.0%	1023, 10.9%	837, 8.9%	453, 4.8%
Class 3		5462, 58.0%	2918, 31.0%	1699, 18.0%	511, 5.4%
Class 4			5160, 54.8%	2367, 25.1%	1974, 21.0%
Class 5				4029, 42.8%	3083, 32.7%
Class 6					3136, 33.3%

Total n = 9421

AIC Akaike Information Criteria, *BIC* Bayesian Information Criteria, *SSABIC* Sample-Size Adjusted BIC, *Adj. LMR–LRT* Adjusted Lo–Mendall–Rubin Loglikelihood Ratio Test

[a]No repeated log-likelihood (local maximization)

exposed to low SES disadvantage but experienced more stressful experiences (n = 1023, 10.9%). Class 4 was comprised of youth with high levels of both SES disadvantage and stressful experiences (n = 320, 3.3%). Additionally, participant demographic characteristics for each class are shown in Table 2.

Path analyses

The direct effect model was tested after setting the low SES disadvantage and low stressful experience group (Class 1) as the reference group. The model fit was good (CFI/TLI = 1.00/1.00; RMSEA = .00). The results are shown in Fig. 2. After adjusting for the effects of early risky health lifestyle and depressive symptoms, all three adversity classes *at Wave 1* were significantly associated with more CM risk *at Wave 4* (β [high SES disadvantage/low stressful experience] = .05; 95% CI .02, .07; β [low SES disadvantage/high stressful experience] = .04, 95% CI .02, .06; β [high SES disadvantage/high stressful experience] = .03, 95% CI .01, .05) compared to the low SES disadvantage/low stressful experience group (i.e., the reference group; Class 1).

Next, risky health lifestyle and depressive symptoms (*at Waves 2 and 3*) were specified as mediators in the direct effect model. The results are shown in Fig. 3. The model had acceptable fit (CFI/TLI = .97/.90; RMSEA = .04). The results showed that the two SES-related adversity classes (Classes 2 and 4) remained at greater CM risk compared to the low adversity reference class even after adjusting for the effects of risky health lifestyle and depressive symptoms. However, the association between low disadvantaged SES/high stressful experiences class and CM risk was not statistically significant (β = .01; 95% CI − .01, .04) after incorporating these mediators.

The multiple mediational processes of adversity were then tested. We detected two specific mediation mechanisms with risky health lifestyle. First, all three adversity classes (Classes 2, 3, and 4) *at Wave 1* were uniquely associated with CM risk *at Wave 4* through risky health lifestyle *at Waves 2 and 3* (b [high SES disadvantage/low stressful experience] = .005; 95% CI .003, .008; b [low SES disadvantage/high stressful experience] = .003, 95% CI .001, .005; b [high SES disadvantage/high stressful experience] = .005, 95% CI .003, .009). The second mechanism was that the two adversity classes related to stressful experiences (Classes 3 and 4) *at Wave 1* directly influenced risky health lifestyle *at Wave 3*, which in turn positively influenced CM risk (b [low SES disadvantage/high stressful experience] = .003, 95% CI .001, .005; b [high SES disadvantage/high stressful experience] = .006, 95% CI .001, .010). Given the significant effect of risky health lifestyle *at Wave 2* on CM risk (β = .04; 95% CI .01, .06), we also estimated mediation effects of adversity classes on CM risk through risky health lifestyle *at Wave 2*, which showed non-significant mediation effects (average mediation effect b = .00; average 95% CI − .01, .01).

Similar mediation mechanisms were also found for depressive symptoms. All three adversity classes were associated with CM risk *at Wave 4* through depressive symptoms *at Waves 2 and 3* (b [high SES disadvantage/low stressful experience] = .003; 95% CI .001, .005; b [low SES disadvantage/high stressful experience] = .002, 95% CI .001, .003; b [high SES disadvantage/high stressful

Table 2 Estimated proportions of class indicators for the co-occurrence between early socioeconomic adversities and stressful experiences

Class indicators	Overall (n = 9421)		Low SES disadvantage/low stressful experiences[a] (*n* = 5160, 54.8%)		High SES disadvantage/low stressful experience[b] (*n* = 2918, 31.0%)		Low SES disadvantage/high stressful experience[c] (*n* = 1023, 10.9%)		High SES disadvantage/high stressful experiences[d] (*n* = 320, 3.3%)		*p* value
	Prob	M (SE)	Prob	M (SE)	Prob	M (SE)	Prob	M (SE)	Prob	M (SE)	
Socioeconomic adversities											
Community adversity	.25		.21		.58		.20		.61		$< .001^{a=c<b=d}$
Family economic hardship	.28		.06		.71		.17		.79		$< .001^{a=c<b=d}$
Low parental education	.45		.27		.76		.30		.83		$< .001^{a=c<b=d}$
Single parent	.30		.12		.67		.23		.79		$< .001^{a=c<b<d}$
Stressful experiences											
Social isolation	.34		.29		.27		.52		.47		$< .001^{a=b<c=d}$
Emotional abuse	.24		.10		.11		.97		.98		$< .001^{a=b<c=d}$
Sexual abuse	.04		.00		.02		.14		.20		$< .001^{a=b<c=d}$
Physical abuse	.09		.00		.00		.50		.62		$< .001^{a=b<c=d}$
Demographics											
Female	.54		.53		.55		.55		.59		.29
Age		15.70 (.01)		15.64 (.02)		15.78 (.03)		15.78 (.05)		15.71 (.13)	.31
Race											
Black	20.9		14.8		33.3		14.2		25.1		$< .05^{a=c<d<b}$
Hispanic	15.4		13.4		18.4		14.1		23.1		$< .05^{a=c<b=d}$
Asian	6.5		6.7		5.2		9.5		5.3		$< .05^{b=d<a<c}$
Native and others	2.7		2.3		3.0		3.4		4.0		$< .05^{a=b=c<d}$
White	54.5		62.8		40.1		58.8		42.5		$< .05^{b=d<a=c}$
Risky health lifestyle (W1)		1.70 (1.35)		1.51 (1.30)		1.83 (1.35)		1.95 (1.46)		1.90 (1.37)	$< .001^{a<b=c=d}$
Depressive symptom (W1)		.65 (.47)		.57 (.43)		.72 (.48)		.75 (.51)		.83 (.50)	$< .001^{a<b=d<c}$

As a post hoc test, Bonferroni tests and Wald-tests were conducted for continuous- and binary variables, respectively

M mean, *SE* standard error

experience] = .002, 95% CI .001, .003). Additionally, all three adversity classes *at Wave 1* influenced depressive symptoms *at Wave 3*, which in turn was implicated in greater CM risk at Wave 4 (*b* [high SES disadvantage/low stressful experience] = .010; 95% CI .001, .019; *b* [low SES disadvantage/high stressful experience] = .004, 95% CI .001, .007; *b* [high SES disadvantage/high stressful experience] = .012, 95% CI .001, .023).

Finally, we found a sequential mediation pathway incorporating both risky health lifestyle and depressive symptoms. That is, all three adversity classes were associated with risky health lifestyle *at Wave 3* through depressive symptoms *at Wave 2*. This mediated pathways significantly predicted CM risk *at Wave 4* (*b* [high SES disadvantage/low stressful experience] = .001; 95% CI .000, .002; *b* [low SES disadvantage/high stressful experience] = .001, 95% CI .000, .002; *b* [high SES disadvantage/high stressful experience] = .001, 95% CI .000, .003).

Discussion

In this study, we investigated longitudinal mechanisms involving risky health lifestyle and depressive symptoms through which unique patterns of early adversity influence young adults' CM risk using a nationally representative sample of youth. Several findings emerged from our investigation. First, using a latent class analysis (LCA) approach, we identified the existence of four distinct adversity patterns. The direct effect model shows that different patterns of early adversity existed, each with a unique combination of early SES disadvantage and

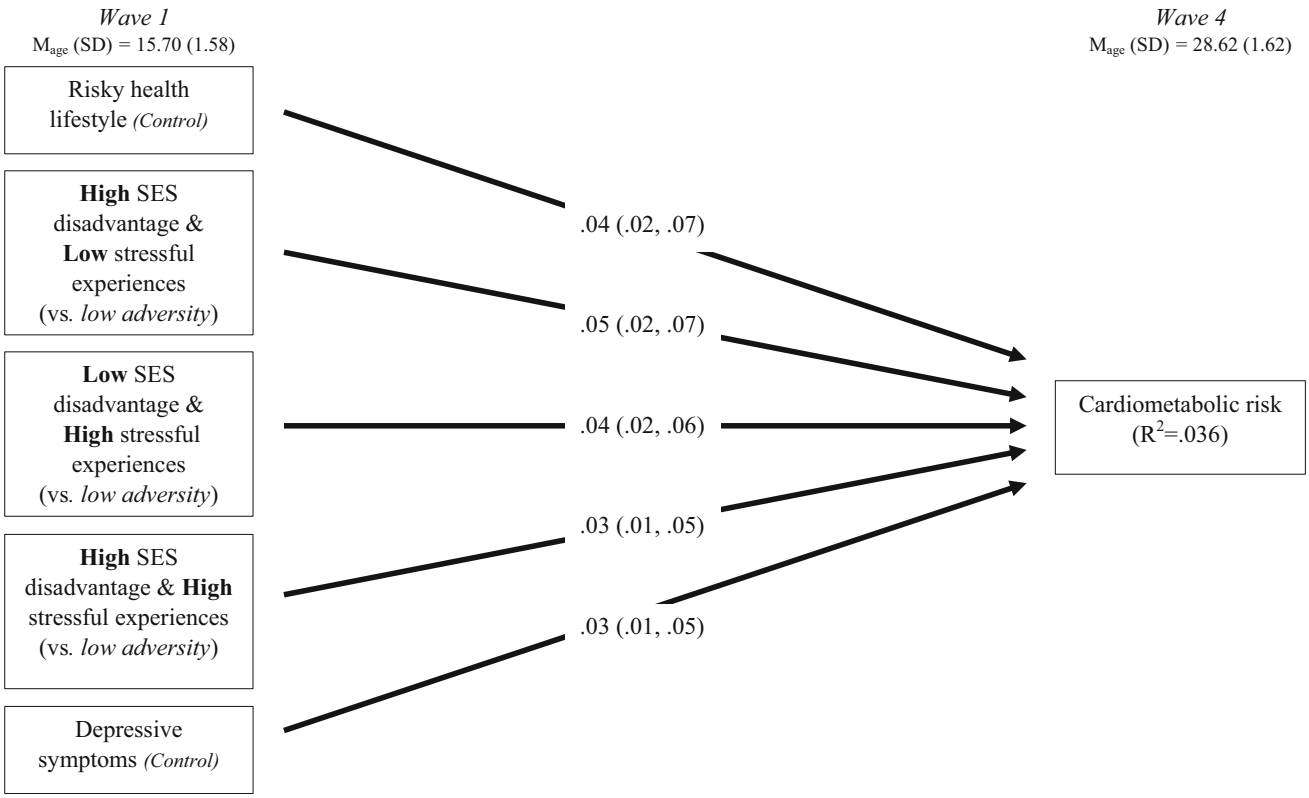

Fig. 2 Direct effects of early adversity patterns on cardiometabolic risk (n = 9421). *Note*. All coefficients are standardized. 95% CIs are shown in parentheses. The reference group is: low SES disadvantage and low stressful experiences. Youths' race/ethnicity, gender, and age were controlled. Bold paths denote statistical significance at $p < .01$. CFI/TLI = 1.00/1.00. RMSEA = .00

stressful experiences. These adversity patterns were uniquely related to subsequent CM risk in young adulthood (see Fig. 2), indicating the multiplicative (joint) effects of the two types of adversity (i.e., adversity related to SES and stressful experiences) on health outcomes in young adulthood. Taken together, our findings extend previous studies by showing multiplicative (joint) effects of two types of early adversity using advanced statistical methods. LCA is an effective approach to test these multiplicative effects by identifying a small set of underlying subgroups characterized by multiple indicators (Lanza & Rhoades, 2013).

Next, the results uncovered mediating processes of both risky health lifestyle and depressive symptoms from adolescence to transition to young adulthood (Waves 2 and 3) *indicating three unique* mediating mechanisms. The first mediation mechanism suggests that both SES- and experience-related early adversities (Classes 2, 3, and 4) serve as triggers creating multiple risk exposures (not only a series of risky health lifestyle but also psychological distress) that persist from adolescence through young adulthood (Waves 2 and 3), which in turn uniquely increases CM risk in young adulthood. These two mediational pathways clearly provide evidence for *parallel mediation processes*, implying that there are multiple pathways to CM risks in young adulthood. In the current study, incorporating multiple mediational processes clearly showed that early adversities are connected with *multiple risk exposures* (risky health lifestyle and mental health problems) at *multiple life stages* (during adolescence to transition to young adulthood), which influence CM risk in young adulthood. Previous findings showed the parallel processes by using multiple risk mediators at a single life stage (transition to young adulthood; Doom et al., 2017). The finding extends previous studies by showing that early adversities generate multiple risks (parallel mediation processes) at multiple life stages.

Second mediating mechanism is related to transition period to young adulthood. For example, youth who experienced early adversity related to stressful experiences (Class 3 and 4) generally reported a riskier health lifestyle and more depressive symptoms during the transition period to young adulthood (at Wave 3), even after accounting for same mediators at Wave 2, which in turn detrimentally influenced their CM risk in young adulthood. Similarly, youth who experienced early adversity related to SES disadvantages (Class 2) reported more depressive symptoms during the transition period to young adulthood (at

Wave 3) compared to those experienced who experienced few adversities, even after accounting for same depressive symptoms at Wave 2, which in turn detrimentally influenced their CM risk in young adulthood. These findings suggest that early adversities uniquely influence behavioral and psychological problems during the *transition period to young adulthood*, further increasing their CM risk in young adulthood. This influence may be due to early adversity provoking a precocious transition to adulthood (e.g., early pregnancy, early employment, and school dropout) (Lee et al., 2018), which may coincide with an increase in risky health behaviors and depressive symptoms. Future studies should explore the potential effects of precocious transition events in greater detail along with the identified mediational processes.

The third mediation mechanism showed significant associations between depressive symptoms at Wave 2 and risky health lifestyle at Wave 3. This is evidence of a *sequential* mediational process stemming from early adversity (Pampel et al., 2010). That is, early adversity may trigger depressive symptoms and depressed youth are more likely to exhibit a risky health lifestyle during the transition period to young adulthood, resulting in CM risk in adulthood.

In addition, our findings pertaining to these three mediation effects suggest unique longitudinal mechanisms linking distinct patterns of early adversity to CM risks in young adulthood. As shown in Fig. 3, SES-related adversities produced unique direct and indirect effects on CM risks in young adulthood. For example, compared to individuals who experienced minimal adversity in adolescence (Class 1), those who experienced SES disadvantages during adolescence (Classes 2 and 4) were at elevated CM risk in young adulthood even after adjusting for mediational effects of risky health lifestyle and depressive symptoms, which is evidence of the long-term direct effects of early adversity on CM risk. This is consistent with the findings of past studies, reporting that youth in low-SES families have persistent long-term CM risk in adulthood (Cohen et al., 2010). In addition, the findings show that early SES disadvantages (Classes 2 and 4) were linked to risky health lifestyle and depressive symptoms in adolescence (Wave 2), which in turn influence these same risk factors during the transition period to young adulthood (Wave 3). These

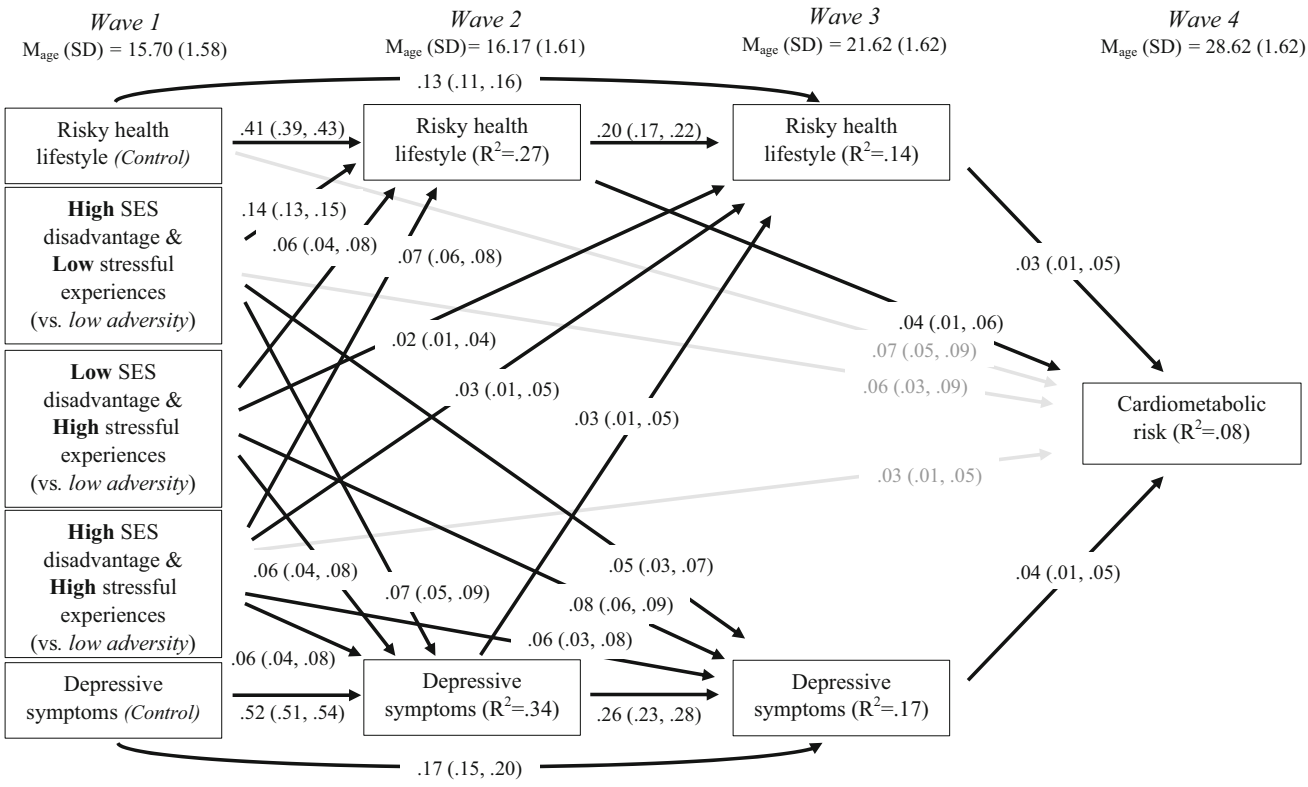

Fig. 3 Indirect effects of early adversity patterns on cardiometabolic risk (n = 9421). *Note*. All coefficients are standardized. 95% CIs are shown in parentheses. There reference group is: low SES disadvantage and low stressful experiences. Youths' race/ethnicity, gender, and age were controlled. Grayed paths represent direct effects of early adversity patterns. All paths denote statistical significance at $p < .01$. Non-significant paths are not shown in figure. Correlations between risky health lifestyle and depressive symptoms at Waves 2 and 3 were significant (*r*s, 95% CIs = .13, .11, .15, $p < .001$; .07, .05, .09, $p < .01$). CFI/TLI = .97/.90. RMSEA = .04

risk factors at Wave 3 then uniquely influenced CM risk in young adulthood (Wave 4). These direct and indirect effects of SES disadvantage suggest that early SES-related adversities increase CM risk in a cumulative fashion (addictive effects; Kuh et al., 2003).

In contrast, early *stressful experiences* (Classes 3 and 4) were only indirectly related to CM risk in young adulthood. In other words, early stressful experiences generate behavioral and psychological risks (i.e., risky health lifestyle and depressive symptoms) during adolescence and young adulthood. These risks have marked effects on CM risks in young adulthood (e.g., trigger effects; Kuh et al., 2003).

Importantly, as shown in Fig. 3, youth who experienced both early SES disadvantage and stressful experiences (Class 4) reported *multiple* risks (risky health lifestyle and depressive symptoms) at *multiple* life stage (adolescence to transition to adulthood to young adulthood), which detrimentally influence CM risk. In addition, long-term associations between these adversities and CM risk in young adulthood were still significant even after adjusting for direct and indirect effects of all other unique adversities (only SES disadvantages and only stressful experience). This result may indicate *synergistic* effects between these two adversity domains (SES disadvantages and stressful experiences) on CM risks in young adulthood. While previous studies suggested that both disadvantaged SES and stressful experience are uniquely associated with CM risks (Doom et al., 2017; Miller et al., 2011), our results identify variability in the distinct mechanisms between these two adversity types (SES disadvantages and stressful experiences) and CM risk in young adulthood.

Limitations and future research

The current study has several limitations. The first limitation is related to selection bias. As the Add Health cohort is a school-based sample, and our analyses included only those who participated in all four waves of data collection. Individuals who experienced greater adversity or very early adversity may have dropped out of school before Wave 1, or they may have higher rates of study attrition. The second limitation pertains to patterns of risky health lifestyle. Based on findings of previous studies (Frech, 2012), we treated risky health lifestyle as a cumulative index by summing multiple indicators. This single summed score of risky lifestyle rests on an assumption of linear and additive effects on CM risk. However, as in the case of early adversity, unobserved heterogeneous patterns of risky health lifestyle may also exist. Similarly, there may be heterogeneous patterns of depressive symptoms. Heterogeneity in risky health style and depressive symptoms implies that complex multiplicative effects may exist for associations between distinct patterns of early adversity and CM risks. However, given the high level of complexity in the multiple mediation model tested, this possibility was not investigated in the current study. Future studies should build on these findings to explore heterogeneity in risky health lifestyle and depressive symptoms and how this heterogeneity may relate to the identified adversity-CM risk association. Third, recall that in the current study, the CM risk variable was created by computing an aggregate score of ten regulatory biomarkers, and the missing data rate for this CM risk variable was 1.3%. However, missing data within individual biomarkers comprising the cumulative CM risk score averaged 12.85% and may have resulted in a slight underestimation of overall CM risk. However, we believe this effect would be relatively small. Finally, our findings do not address how changing health behaviors later in life may impact pre-existing CM risk. As the Add Health study extends to follow respondents later in life (including the recently collected Wave 5 data), future studies will be able to address to what extent CM outcomes relatively early in life are reversible depending on improved health behaviors later in life.

Public health implications

Our findings have important practical implications for prevention and intervention strategies. Based on our findings that early adversities are connected to directly and indirectly to CM risk later in life through multiple risk pathways, primary prevention of early adversity in childhood and adolescence is the ultimate and ideal goal. Furthermore, interventions should address diverse types of adversity when seeking to reduce CM risk in young adulthood. For example, our findings show that the early SES-related adversity as a chronic stressor has persistent and additive long-term effects on CM risks in young adulthood. These findings highlight the need for federal, state, and local level policies and programs aimed to prevent adolescents' socioeconomic failures, including truncation of education and financial problems, as well as parenting interventions designed to help families cope during times of economic distress.

In contrast, early stressful experiences (such as emotional, physical, sexual abuses, and social isolation) were found to generate more proximal behavioral and psychological outcomes (risky health lifestyle and depressive symptoms), which in turn has marked effects on increasing CM risk in young adulthood. These findings suggest modifiable ways to intervene with youth who have experienced early experiences of adversity. For example, promoting healthy lifestyle behaviors represent a practical and attainable course of intervention that can be implemented

through methods such as provisions of healthy food options, ensuring sufficient health care, and educating youth on the importance of positive health behaviors. Additionally, the continuity of depressive symptoms from adolescence into transition to young adulthood emphasizes the need for ongoing psychological care for depressed, or at-risk, adolescents. Consequently, while prevention program should incorporate distinct effects of early adversities in childhood and adolescence, interventions should also address behavioral and psychological problems to reduce CM risk in young adulthood.

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Compliance with ethical standards

Conflict of interest Tae Kyoung Lee, Kandauda A. S. Wickrama, and Catherine Walker O'Neal declare that they have no conflict of interest.

Human and animal rights and Informed consent All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Informed consent was obtained from all individual participants included in the study.

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