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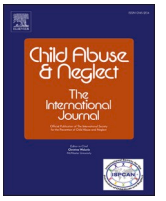

Childhood sexual abuse and cholesterol risk: Testing body mass index as a mediator across gender in a national sample of adults☆,☆☆

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ABSTRACT

Background: Childhood sexual abuse (CSA) is associated with health problems, including cardiometabolic outcomes. Findings directly linking CSA to cholesterol levels are mixed, and identifying mediating pathways is the next logical step. Body mass index (BMI) is one possible mediator, given its association with both CSA and cardiometabolic outcomes. Gendered effects of CSA indicates that BMI may operate differently in men and women.
Objective: We tested BMI as a mediator linking CSA to high-density lipoprotein (HDL) and low-density lipoprotein (LDL) using a multiple group structural equation model stratified across gender to test the indirect effects.
Participants and setting: We utilized a sample of 1054 adults (54.7 % women) from the study of Midlife Development in the United States, who were drawn from the general population.
Methods: Using two waves of data, participants responded to a questionnaire assessing CSA, provided measurements from which to calculate BMI, and a fasting blood sample from which cholesterol levels were measured.
Results: The indirect effects in the overall sample yielded a significant effect from CSA to HDL via BMI ($\beta = -0.03$, 95 % CI [−0.050, −0.010]), but not LDL ($\beta = 0.006$, 95 % CI [−0.002, 0.014]). The indirect effect from CSA to HDL cholesterol was significant among women ($\beta = -0.04$, 95 % CI [−0.066, −0.012]) only. Indirect effects to LDL among both genders were both non-significant.
Conclusions: BMI appears to be a possible mediator linking CSA to lower HDL cholesterol among women suggesting BMI could be a point of trauma-informed prevention and intervention especially impactful.

1. Introduction

Childhood sexual abuse (CSA) is a threat to public health resulting in substantial economic, interpersonal, psychological, and

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physical burden. Although there is variation in definitions of CSA (Briere, 1992; DiLillo, 2001; Cicchetti & Toth, 2005; Mathews & Collin-Vézina, 2019), sexual abuse is frequently defined by forced or unwanted sexual contact and inappropriate exposure of sexually explicit material. CSA has been linked to the leading causes of death, including cardiovascular disease (Fuller-Thomson et al., 2012; Irish et al., 2010; Jakubowski et al., 2021; Rich-Edwards et al., 2012). Cardiometabolic biomarkers are critical pre-clinical indicators of cardiovascular risk (Félix-Redondo et al., 2013) and specific forms of maltreatment, such as CSA, may impact these biomarkers resulting in increased risk for cardiovascular disease and premature mortality (Fuller-Thomson et al., 2012; Jakubowski et al., 2021; Rich-Edwards et al., 2012). High-density lipoprotein (HDL) and low-density lipoprotein (LDL) are two distinct forms of cholesterol that demonstrate differential effects on health. HDL is colloquially termed "good cholesterol" and has health benefits, while LDL is referred to as the "bad cholesterol" which is a risk factor for cardiovascular disease (Center for Disease Control, 2024).

One notable issue in existing research on CSA and cholesterol is that research has almost exclusively examined CSA as a predictor of cholesterol without considering mediating pathways or differences between populations. When considering midlife and older adults, a time period when cholesterol rises (Grundy et al., 2019), there is a substantial amount of time between sexual abuse in childhood and cholesterol levels in midlife. There is little theoretical work to justify a direct association between CSA and HDL and LDL, rather the effects are likely to be conditional or indirect. Body mass index (BMI) is a possible mechanism that may connect CSA to cholesterol. These propositions, however, have seldom been tested. Additionally, gender differences exist in the prevalence rates of CSA (Stoltenborgh et al., 2011) and in the association between CSA and BMI (Elsenburg et al., 2023), indicating the potentially mediating role of BMI may differ for men and women. Identification of BMI as a possible mediator linking CSA to cholesterol levels in middle adulthood across gender would serve to 1) identify specific types of adverse childhood experiences, such as CSA, as a particularly important risk factor for higher BMI, 2) identify BMI as a potential target for prevention and intervention efforts and utilize a trauma-informed approach to combat the lack of weight loss found among CSA survivors (e.g., Oppong et al., 2006), and 3) document for whom prevention efforts may be most effective. Consideration of only direct effects of CSA on cholesterol places prevention and intervention efforts solely on the prevention of sexual abuse and such efforts have theoretical and methodological issues that have resulted in mixed effects (Topping & Barron, 2009; Zeuthen & Hagelskjær, 2013). Therefore, the purpose of the current study was 1) to examine BMI as a mediating pathway linking CSA to HDL and LDL cholesterol, and 2) examine possible gender differences between men and women using a large national sample of adults from the study of Midlife Development in the United States (MIDUS).

1.1. Theoretical and conceptual underpinnings

Wiss et al. (2022) proffered that sexual abuse becomes biologically embedded, triggering a cascade of physiological effects (e.g., inflammation) that contribute to food addiction, eating disorders, and emotional eating. In response to powerlessness, betrayal, and stigmatization (Finkelhor & Browne, 1985) individuals may utilize food as a source of control or comfort through disordered eating behaviors, such as binge eating (Hodge & Baker, 2021). Studies have found that CSA is associated with a spectrum of using food to cope ranging from overeating (Emery et al., 2021) to food addiction (Kiefer et al., 2021). Chronic stress can lead to an increase in the food-associated drive and desire to consume "comfort foods" to mitigate the stress response (Dallman et al., 2005) which can offer short term regulation but may lead to long term consequences, such as weight gain, due to a positive energy balance (Kazmierski et al., 2022). Additionally, other mechanisms including health behavior (e.g., alcohol use, smoking) can contribute to increased risk for being overweight or obese and ultimately lead to poorer cardiometabolic health (Scott-Storey, 2013).

1.2. Childhood sexual abuse and cardiometabolic health

Although there is substantial variation in CSA prevalence rates due to prospective vs retrospective reports, conceptual definition of sexual abuse (e.g., contact vs non-contact), and who data were collected from including official records (e.g., CPS, hospital) versus self-report or collateral (e.g., parent) reports, there is agreement that CSA is common in the United States. According to official reports of sexual abuse, the National Incidence Study-4 (NIS-4; Sedlak et al., 2010) found that approximately 300,000 children experienced sexual abuse in 2005–2006. Among adults in the United States and Canada, 20 % of female and 8 % of male participants retrospectively reported CSA (Stoltenborgh et al., 2011).

Despite studies connecting CSA to cardiovascular health (e.g., Rich-Edwards et al., 2012), the relationship between CSA and cholesterol has only recently been studied. While there are studies that have indeed found a relationship between CSA and cholesterol (van Reedt Dortland et al., 2012), findings more frequently indicate that CSA is not directly associated with HDL and LDL (Kisely et al., 2023; Li et al., 2021; Miller & Lacey, 2022; O'Leary et al., 2023). For example, (Goncalves Soares et al., 2021) examined the associations between CSA and cardiometabolic health and found that CSA was not associated with HDL or LDL among young adults. These findings have been replicated in younger adults (Kisely et al., 2023), and extended to midlife adults (Li et al., 2021; Miller & Lacey, 2022). Similar findings have been likewise noted in the National Child Development Study (or 1958 British Cohort), a prospective study of adults born in Wales, England, and Scotland in 1958 (see Power et al., 2020 for review).

The consistent lack of findings linking CSA to cholesterol levels has several possible interpretations. The first and the seldom mentioned is that there is not an association between CSA and cholesterol, meaning that CSA is not directly or indirectly associated with cholesterol levels among adults. Instead, there may be dose-response relationship where CSA, combined with other forms of childhood adversity, is associated with poorer cholesterol profiles (Miller & Lacey, 2022; O'Leary et al., 2023). A dose-response relationship underlying the relationship between adverse childhood experiences (ACEs) and HDL and LDL assumes that 1) each ACE exerts a similar effect on the distal outcome, and 2) ACEs operate under the same mechanism(s) and greater exposure to adversity increases the mechanism(s) that ultimately impact health (McLaughlin et al., 2021). Second, there may be domain specific mechanisms

where individual forms of ACEs (e.g., sexual abuse) affect different mechanisms (e.g., BMI) that influence levels of cardiometabolic biomarkers (McLaughlin et al., 2021). Given the considerable amount of chronological time between CSA and cholesterol levels in midlife, relatively recent theoretical work suggests an indirect or conditional relationship (Scott-Storey, 2013; Wiss et al., 2022). Thus, it is not surprising that associations between CSA and cholesterol are small and non-significant, which is consistent with other biomarkers, such as inflammation (Chiang et al., 2022). Instead, testing possible intervening factors and processes can highlight pathways of risk and resilience. Research has seldom tested mediating mechanisms (e.g., smoking, alcohol use, and BMI) linking CSA to cholesterol and instead the possible mechanisms are omitted or inappropriately implemented as covariates (e.g., Li et al., 2021; O'Leary et al., 2023), thereby potentially masking the effects of CSA on cholesterol. Together, these limitations miss an opportunity to identify possible mediating pathways (Jakubowski et al., 2021) and CSA has been consistently associated with greater BMI (Danese & Tan, 2014) and may be one such pathway.

1.3. Mediating role of body mass index

Although research has demonstrated some mixed findings (Chiu et al., 2023), a substantial proportion of studies have found an association between CSA and BMI (Rohde et al., 2008; Schroeder et al., 2021), including meta-analytic evidence (Danese & Tan, 2014). Varying forms of childhood abuse have been linked to being underweight and overweight, but CSA has stronger associations with being overweight and obese compared to being underweight (Roenholt et al., 2012). The relationship between CSA and BMI in adolescence is inconsistent, but becomes more stable in adulthood (Elsenburg et al., 2023; Sokol et al., 2019). Data from numerous large-scale studies, including the ACEs study (Williamson et al., 2002) and ADD health, indicate that CSA is associated with a greater BMI (Kisely et al., 2023). There is also longitudinal evidence connecting CSA to BMI over time. A recent study found that CSA was associated with a greater BMI as well as steeper increase in BMI over time for women who experienced sexual abuse compared to women who had not; however, the relationship between sexual abuse and BMI in men was not significant (Elsenburg et al., 2023). Other studies have demonstrated similar effects across gender, finding CSA was associated with a lower BMI among men, but greater BMI in women (Sokol et al., 2019). These findings suggest that BMI may serve as a mediator connecting CSA to LDL and HDL values, although the impact of BMI may affect men and women differently.

BMI has been implicated in adult cardiometabolic health among midlife adults (Li et al., 2021), including altered cholesterol absorption, synthesis, and transport (see McAuley, 2020 for review), and may serve as a possible pathway linking CSA to unhealthy cholesterol levels. In fact, estimates indicate that 60–70 % of obese individuals and 50–60 % of overweight individuals have dyslipidemia (Bays et al., 2013). LDL and HDL cholesterol are commonly measured lipoproteins used to indicate the body's ability to balance the production and clearance of triglycerides that are consumed from the diet and synthesized endogenously. Findings are inconsistent for BMI and LDL cholesterol; some studies indicate higher BMI is associated with higher levels of LDL cholesterol (Varbo et al., 2015) while others found no significant associations between BMI and LDL (Shamai et al., 2011). Higher BMI has noted associations with decreased HDL cholesterol (Rashid & Genest, 2007; Varbo et al., 2015). Additionally, studies found differences among men and women with stronger associations for women. For example, Shamai et al. (2011) found significant associations for BMI and HDL in women, but not men.

The relationship between cholesterol metabolism and obesity is complex. A meta-analysis of randomized control trials of interventions designed to treat obesity in adults found that weight loss of 1 kg was associated with a decrease in LDL cholesterol and an increase in HDL cholesterol (Hasan et al., 2020) and multiple physiological pathways help explain this connection. When an individual's energy intake exceeds their needs, the body stores excess energy as triglycerides in adipose tissue. Excess adipose tissue can result in a pro-inflammatory state and is associated with insulin resistance which increases lipolysis, the breakdown of fat, resulting in an increase in free fatty acids in the blood (Vekic et al., 2019). This initiates the formation and release of very-low density lipoprotein (VLDL) particles, which are converted to LDL particles. Ultimately, these changes can lead to unwanted modifications in the composition of lipoproteins, such as HDL and LDL cholesterol (Feingold & Grunfeld, 2015).

1.4. The present study

The current study examined BMI as a possible pathway linking sexual abuse to HDL and LDL stratified across gender. Regarding the hypotheses, first, using the full sample of midlife adults drawn from the general population, it was hypothesized that 1) CSA would be directly associated with higher levels of BMI, and 2) BMI would be associated with lower levels of HDL and greater levels of LDL. No direct effects were hypothesized from CSA to HDL and LDL; instead, the associations from CSA to cholesterol will be accounted for by BMI and the covariates. When decomposing the associations across gender, we expect that CSA will be associated with BMI in women but not men (e.g., moderate), but BMI will be associated with greater LDL and lower HDL in both women and men. By extension, it is expected that BMI will be a mediator linking CSA to HDL and LDL in women only.

To elucidate the unique direct and indirect effects, numerous covariates were employed. We included sociodemographic factors (e.g., age, race, and household income), modeled other potential mediators including health behavior (alcohol use and smoking status) and educational achievement (Scott-Storey, 2013). Additionally, common causes of independent and dependent variables (e.g., third variable problem; DiLillo, 2001) should be controlled for to rule out spurious associations and in the current study we included eating habits as a covariate, which can be a potential cause of both BMI and cholesterol. Many existing studies (e.g., Kisely et al., 2023; Li et al., 2019) did not adjust for important covariates, such as waist to hip ratio, use of cholesterol medication, and family history of high cholesterol, which are highly relevant covariates (Spann et al., 2014). Thus, we controlled for cholesterol specific factors including cholesterol medication use and family history of high cholesterol. Finally, health disparities research has indicated there are

disproportionately worse health outcomes among adults living in rural communities (Cross et al., 2020; Cross et al., 2021), so we controlled for living in a rural community.

2. Method

Data from the current study are from the longitudinal study of midlife development in the United States (MIDUS), a study focused on understanding biopsychosocial determinants of aging and health. The MIDUS study has been continually funded by the John D. and Catherine T. MacArthur Foundation since 1995–1996. The MIDUS has collected data every 9 years starting in 1995–1996 (MIDUS 1) with subsequent waves in 2004–2006 (MIDUS 2) and 2013–2014 (MIDUS 3). The original MIDUS data collection was comprised of individuals from four discrete groups: (1) a national random digit dialing (RDD) sample ($n = 3487$); (2) city oversamples in the United States ($n = 757$); (3) siblings of individuals from the RDD sample ($n = 950$); and (4) a national RDD sample of twin pairs ($n = 1914$). MIDUS 2 retained 4963 participants and MIDUS 3 retained 3294 individuals. In addition to the primary data collection, following MIDUS 2 (2005–2009; $n = 1255$) biological samples were collected from a subset of individuals who participated at MIDUS 2 ($n = 1054$) and a new sample of African Americans residing in Milwaukee ($n = 201$). Participants were included in the study if they participated in the biomarker at MIDUS 2 and MIDUS 2 biomarker study (n = 1054). The Milwaukee participants (n = 201) were not included due to not participating in MIDUS 2, thus not providing data on BMI.

2.1. Participants

Adults included in the current analysis (n = 1054) had a mean age of 55.26 ($SD = 11.78$), 54.7 % were women ($n = 577$), 92.8 % were White adults and the remaining were racial or ethnic minority (2.6 % Black, 1.3 % Native American, 0.3 % Asian, 2.8 % reported "other" and 0.1 % refused), and 760 adults were married (72.1 %). Regarding educational achievement, 37 (3.5 %) participants did not graduate high school, 217 (20.6 %) reported either a high school diploma or GED, 224 (21.3 %) reported some college but no degree, 83 (7.9 %) reported an associate's degree or technical degree, 241 (22.9 %) reported a college degree, 48 (4.6 %) reported some graduate school but did not earn a degree, and 201 (19.1 %) participants reported earning either a master's or doctorate; three people did not report their educational achievement. The mean household income was $76,384.60 ($SD = $60,050.10). Most participants ($n = 980$) resided in urban or suburban areas (93 %), while 74 participants lived in rural areas (7 %).

2.2. Measures

2.2.1. Childhood sexual abuse

The Childhood Trauma Questionnaire (CTQ; Bernstein et al., 1994, 2003) is a 25-item scale assessing five types of childhood abuse and neglect prior to the age of 18. Subscales include emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect. Items are scored on a five-point Likert scale, ranging from (1) '*Never*' to (5) '*Very Frequently*.' The CTQ has been found to have adequate test-retest reliability, convergent validity, construct validity, and criterion-related validity (Bernstein et al., 1994, 2003). For the current study, we utilized the clinical cutoff score (scores of 8 or greater) to indicate clinically significant sexual abuse (Walker et al., 1999). An example item was "Someone molested me." ω of the sexual abuse subscale was 0.95 for the total sum score from which the cutoff score was derived.

2.2.2. Body mass index

BMI of participants was calculated by dividing their self-reported weight (kg) by their height (meters) squared. BMI was measured at MIDUS 2.

2.2.3. HDL and LDL

Participants in the MIDUS 2 biomarker stayed overnight at one of three general clinical research centers. HDL and LDL were drawn from fasting blood samples collected from each participant before breakfast on Day 2 of their stay. Frozen samples were stored in a −60 °C to −80 °C freezer until shipped on dry ice to the MIDUS Biocore Lab. The frozen serum and plasma in 1 mL aliquots were shipped to the MIDUS Biocore Lab monthly for the following biomarker assays. More detailed information regarding the blood draws can be found in the biomarker documentation. HDL and LDL were measured at the MIDUS 2 biomarker.

2.3. Grouping variable

2.3.1. Gender

Participants' gender was coded as either being a male (1) or female (2).

2.4. Covariates

Covariates included childhood emotional and physical abuse and neglect (CTQ subscales), age, race (racial minority/White), marital status (not married/married), educational achievement, household income (thousands of dollars), current cholesterol medication use (yes/no), alcohol use, smoking status (smoker/non-smoker), family history of high cholesterol (yes/no), household dysfunction in childhood, rurality (rural/non-rural), healthy eating habits, and waist to hip ratio. For more detail on how covariates we

assessed, please see the online supplementary material.

2.5. Data considerations

There are several methodological considerations worthy of brief discussion. First, within the MIDUS data there is a large subsamples of both twins and siblings, which are non-independent data (e.g., correlated residuals) due to shared childhood experiences (e.g., maltreatment) and genetics, which underestimated the standard errors resulting in a type 1 error. Given that research questions under investigation were at level 1 (individual level) not at level 2 (family level), multilevel modeling (MLM) would not be an appropriate analytic method and MLM induces additional assumptions at level 2 (e.g., endogeneity) that may or may not be met (see McNeish et al., 2017 for in-depth discussion). To correct the standard errors, we used TYPE = COMPLEX in Mplus, which provides cluster correct standard errors (McNeish et al., 2017). Second, missing data in the current study were minimal and covariance coverage was >95.6 % for all bivariate pairs of variables. The data were considered missing at random (MAR), which presumes that variables in the model are predictive of missingness. Given the extensive number of covariates, several of which have been associated with participant dropout in the MIDUS (Song et al., 2021), we believe this is a tenable assumption. Additionally, when there are low levels of missing data, the use of modern missing data methods will not make meaningful differences in parameter estimates, standard errors, or increase statistical power (Enders, 2023). Third, we utilized coefficient Omega (ω; Hancock & An, 2020) because assumptions of Cronbach's alpha (e.g., unidimensionality, tau-equivalence) are almost never met in practice, resulting in lower reliability estimates (McNeish, 2018). Lastly, due to the large number of covariates used, measurement of the covariates is provided in the online supplementary material.

3. Statistical analysis

3.1. Analytic approach

Our first analytic step was to generate descriptive and bivariate statistics in SPSS. Descriptive and bivariate statistics included means, standard deviations, correlations, and *t*-tests or chi-square testing study variables across gender. To test the hypotheses, a measured variable path analysis was specified in Mplus v 8.10. To evaluate model-data fit, numerous statistics are commonly used, including the chi-square statistic, comparative fix index (CFI), standardized root mean square residual (SRMR), and root mean square error of approximation (RMSEA). Although Hu and Bentler (1999) provided general findings for cutoff values regarding model-data fit using confirmatory factor analysis, they cautioned against overgeneralization to other SEM models, which has largely been ignored. Omnibus measures of fit (e.g., CFI, RMSEA) provide an overall picture of model-data fit, but their performance is influenced by the strength of the covariation among variables, model complexity, and sample size (e.g., Kenny et al., 2015; Van Laar & Braeken, 2021). Further, global fit indices fail to identify local misspecification that can have cascading effects to other parts of the model and examination of the residual matrix (expected – observed covariance matrix) can identify local misfit. Therefore, model-data fit was evaluated based on global fit indices as well as the residual matrix while considering model complexity and degrees of freedom. In the first set of analyses, a single group SEM model was tested using the entire sample and the second set of analyses stratified paths across gender by utilizing a multiple group path analysis. For all analyses we report completely standardized coefficients, but for pathways including a binary predictor (e.g., CSA to BMI), coefficients are partially standardized (only dependent variable was standardized), which allows for the regression weights to be interpreted as a standardized mean difference (e.g., Cohen's *d*).

To address possible multivariate non-normality of the residuals, numerous methods of estimation have been proposed. Methods include robust maximum likelihood (MLR), asymptotic distribution free estimators (ADF; Browne, 1984), and bootstrapping (MacKinnon et al., 2004). We opted for MLR because ADF requires large sample sizes (1000 for simple models and 5000 for more in complex models) and bootstrapping is prone to type 1 errors (Satorra & Bentler, 2001; West et al., 1995). The final step in the analytic approach was to run sensitivity analyses across additional conditions (e.g., cholesterol cutoff values, cholesterol medication use) to determine the stability and generalizability of the results.

Table 1
Bivariate correlations among independent, mediating, and outcome variables.

	Entire sample				Breakdown across gender			
	1.	2.	3.	4.	1.	2.	3.	4.
1. Sexual abuse	–				–	0.16***	−0.03	0.01
2. Body mass index	0.11***	–			0.09	–	−0.41***	0.07
3. Low-density lipoprotein	0.06*	−0.39***	–		−0.01	−0.37***	–	−0.14***
4. High-density lipoprotein	0.02	0.03	−0.09**	–	0.04	−0.01	−0.05	–

Note. In the breakdown across gender, males are presented below the diagonal and females are presented above the diagonal.
* $p < .05$.
** $p < .01$.
*** $p < .001$.

4. Results

4.1. Descriptive and bivariate results

Table 1 displays the correlations for the entire sample and across gender. Table 2 displays the gender differences between men and women across the independent, mediating, and outcome variables. Using the CTQ cutoff scores, we found that 8 % of men and 22.7 % of women reported sexual abuse.

4.2. Structural equation modeling

4.2.1. Single group structural equation model

The SEM analyzing the entire sample demonstrated good model data fit using omnibus measures χ^2 (2) = 0.88, p = .64, CFI = 1, TLI = 1, RMSEA = 0, 90 % CI [0.000, 0.048], SRMR = 0.002. The direct effects from CSA to HDL and LDL were not estimated, resulting in 2 degrees of freedom. Examination of the residual matrix indicated no local misspecification (e.g., no significant z-scores comparing observed vs expected covariances). Thus, cumulative evidence suggests an adequate fitting model with no apparent misspecifications. Results are displayed in Fig. 1. CSA was associated with BMI such that adults who reported higher levels of CSA also reported higher levels of BMI (β = 0.11, p = .003). Greater BMI was, in turn, associated with lower levels of HDL (β = −0.27, p = .003) indicating that adults with a higher BMI also tended to report lower levels of HDL. In contrast, BMI was not associated with LDL (β = 0.06, p = .08). Regarding the indirect effects, CSA was indirectly associated with HDL via BMI (β = −0.03, 95 % CI [−0.050, −0.010]), but was not indirectly associated with LDL (β = 0.006, 95 % CI [−0.002, 0.014]). Parameter estimates of the covariates are presented in the online supplementary material.

4.2.2. Multiple group structural equation model stratified across gender

The SEM that stratified across gender demonstrated good model data fit using omnibus measures: χ^2 (4) = 0.921, p = .92, CFI = 1, TLI = 1, RMSEA = 0, 90 % CI [0.000, 0.022], SRMR = 0.004 and examination of the residual matrix indicated no local misspecification (e.g., no significant z-scores between observed vs expected covariances). In the male model, CSA was not associated with BMI (β = 0.04, p = .34). Higher levels of BMI were associated with lower levels of HDL (β = −0.31, p < .001), but was not associated with LDL (β = 0.00, p = .56). The indirect effect from CSA to HDL was non-significant (β = −0.01, 95 % CI [−0.041, 0.014]) as was the indirect effect from CSA to LDL (β = −0.001, 95 % CI [−0.003, 0.003]).

In the female model, CSA was associated with greater BMI (β = 0.13, p = .02) such that women who reported greater CSA, reported a higher BMI in adulthood. Women who reported higher levels of BMI also had lower levels of HDL (β = −0.30, p < .001) and higher levels of LDL (β = 0.09, p = .04). The indirect pathway from CSA to HDL was significant among women (β = −0.04, 95 % CI [−0.066, −0.012]), but the pathway to LDL was not significant (β = 0.01, 95 % CI [−0.002, 0.026]), indicating that CSA was associated with decreased HDL through BMI. Regarding gender as a moderator, none of the chi-square difference tests, which were adjusted for the scaling correction factor provided in MLR, were statistically significant. Although the pathway from sexual abuse to BMI was significant for women but not men, constraining the pathway to be equal in males and females resulted in a non-significant chi-square difference test [χ^2 (1) = 1.89, p = .17]. Likewise, gender did not moderate the pathway from BMI to HDL [χ^2 (1) = 1.18, p = .27] or LDL [χ^2 (1) = 0.56, p = .45] (Fig. 2).

4.3. Sensitivity analyses

Sensitivity analyses were run to determine the stability of the findings across additional conditions (see Thabane et al., 2013 for overview). Sensitivity analyses were only employed in the multiple group model. The sensitivity analyses were implemented to compare models employing statistical control of cholesterol factors and controlling by design (e.g., inclusion criteria). The first sensitivity analysis ran excluded participants who had LDL cholesterol levels above 160 (e.g., Chen et al., 2016), which resulted in an analytic sample of 979 (54.6 % women) of the 1054 adults. Complete results of sensitivity analyses are presented in the online supplementary material. Briefly, no substantive differences were identified regarding the indirect effects; the relationship between BMI and LDL among women, however, fell to non-significance, indicating that the association between BMI and LDL was not significant when excluding those with severely high LDL. The second sensitivity analysis (N = 678; 58.7 % women) we ran excluded adults who were currently taking cholesterol medication and there were no substantive findings different from the hypothesized SEM models other than sexual abuse demonstrating a positive, direct relationship with HDL among women, indicating women who experienced sexual abuse in childhood also had higher levels of HDL.

Table 2
Gender differences across independent, mediating, and outcome variables.

Variable	Male mean/n	Female mean/n	t/χ^2	p	Cohen d
Sexual abuse	38	131	42.12	<0.001	–
Body mass index	28.35	27.59	2.208	0027	0.14
High-density lipoprotein	46.73	61.16	14.44	<0.001	−0.90
Low-density lipoprotein	105.23	107.2	−0.902	0.184	−0.06

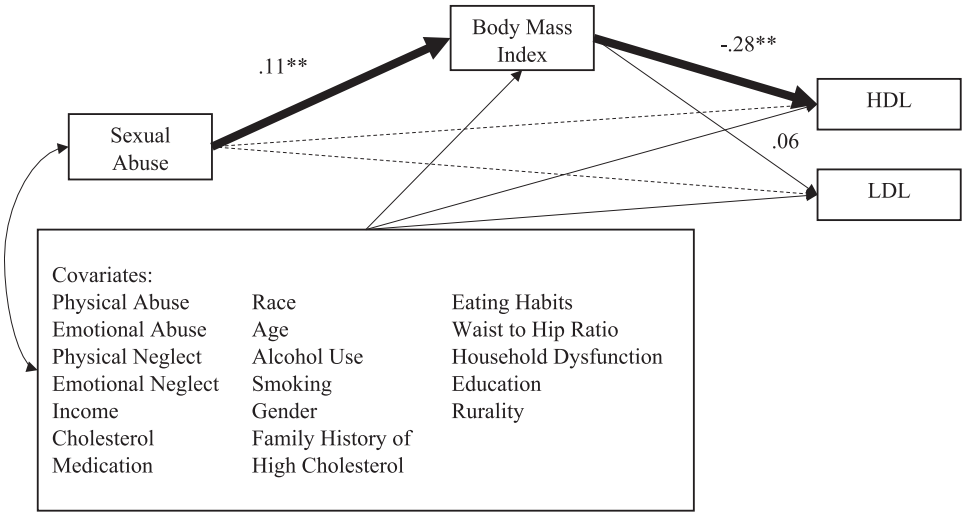

Fig. 1. Results of the structural equation model testing the indirect effects from childhood sexual abuse to cholesterol via BMI using the entire sample.
Note. Standardized regression coefficients are presented. Bold lines represent pathways involved in a significant indirect effect. For ease of presentation, regression coefficients for covariates are not presented.

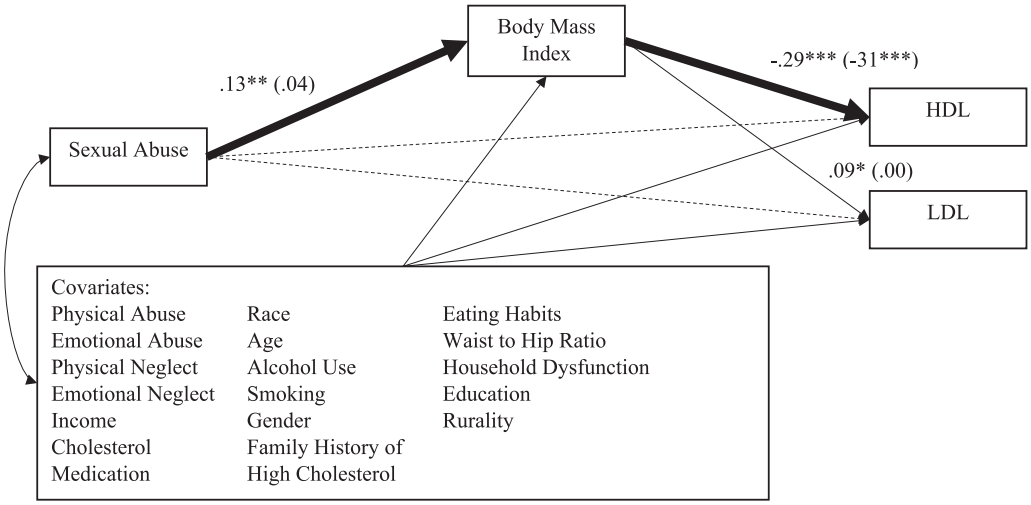

Fig. 2. Results of the structural equation model testing the indirect effects from childhood sexual abuse to cholesterol via BMI stratified across gender.
Note. Standardized regression coefficients are presented. Parameter estimates for men are in the parenthesis and women are outside the parentheses. Bold lines represent pathways involved in a significant indirect effect. For ease of presentation, regression coefficients for covariates are not presented.

5. Discussion

Extant research has largely found a null association between CSA and cholesterol, but the extended period of time between CSA and cardiometabolic health in midlife suggests a possible indirect relationship, working through intervening pathways. BMI was tested as a mediating pathway and it was found that CSA was associated with decreased HDL via greater BMI, but the indirect effect was only significant for women. Further strengthening conclusions, sensitivity analyses found that these effects were stable when removing those with high LDL cholesterol and those currently taking cholesterol medication. The effects of BMI on LDL, however, were more inconsistent, which is not surprising given that cholesterol medication targets LDL (Cholesterol Treatment Trialists' Collaboration et al., 2010; Stancu & Sima, 2001).

Prior literature has documented that CSA is associated with poorer physical health in adulthood (Irish et al., 2010), but literature has more scantly examined possible pathways leading to deleterious health outcomes, particularly across gender. To build on the

existing literature, the current study documented that BMI is a possible pathway linking CSA to cholesterol levels among women. Supporting the hypotheses and consistent with previous research, women who experienced CSA had higher levels of BMI (Richardson et al., 2014; Sokol et al., 2019), leaving them at risk for lower levels of HDL. The gender differences between CSA and BMI across gender may be a reflection of greater social pressure focused on physical appearance that is placed on women (Boone et al., 2011). Sexual objectification is a particularly salient characteristic in CSA (Finkelhor & Browne, 1985), which may lead to greater BMI among women. Specifically, the internal (e.g., negative cognitions) and external (e.g., sexual exploitation) objectification are psychologically and physiologically overwhelming, and a strategy for decreasing objectification is to rebel against the social standard for body weight and image as a form of self-protection. It should, however, be noted that gender did not moderate the pathway from CSA to BMI and the interpretations are based on the significant indirect effect and the standardized effect being substantially larger ($\beta = 0.13$ vs $\beta = 0.04$). Despite a large sample of adults, our study may be underpowered to detect such a small difference in parameter estimates (0.09). Given that the pathway from BMI to HDL was not significantly different between men and women, but the effect from CSA to BMI was only significant for women, there are reasons to believe that there may be important gender differences despite a non-significant moderating effect.

BMI has been identified as a possible pathway linking CSA to physical health problems in adulthood (McCarthy-Jones & McCarthy-Jones, 2014) and our study focuses specifically on HDL and LDL cholesterol. Hypotheses were partially supported, both men and women who reported a higher BMI reported significantly lower levels of HDL and gender did not moderate the association between BMI and HDL in the multiple group model. Our findings aligned with previous research that found a strong, negative association between BMI and HDL (Shamai et al., 2011; Vekic et al., 2023). This is significant considering low HDL levels are associated with negative cardiometabolic outcomes, even in individuals with healthy LDL values (Mascarenhas-Melo et al., 2013) and may be a better indicator of cardiometabolic risk. In both the primary analyses and in both sets of sensitivity analyses, the association between BMI and HDL was consistent. There were no meaningful fluctuations in the effect sizes of the regression coefficients (βs varied between 0.29 and 0.31). Thus, we found evidence that the relationship between BMI and HDL did not vary across gender or when those with clinical levels of cholesterol problems were removed, which is not all that surprising given the relatively small, negative correlation between HDL and LDL.

Interestingly, while prior research has suggested that BMI is also associated with greater LDL, although less so than HDL, we found that BMI was associated with LDL in women; however, this effect disappeared when removing those with cholesterol levels >160 and when women who were on cholesterol medication were removed, indicating the extreme values may strongly influence the linear association between BMI and LDL. For example, not reported in the results, there were significant mean differences in HDL (Cohen's $d = -0.28$), LDL (Cohen's $d = -0.45$), and BMI (Cohen's $d = 0.29$) among those who were currently on cholesterol medication and significant mean differences for HDL (Cohen's $d = -0.27$), and BMI (Cohen's $d = 0.30$), but not LDL (Cohen's $d = -0.10$) among those who have a diagnosed cholesterol problem. Those on cholesterol medication reported lower HDL, LDL, and higher BMI while those who have been diagnosed with a cholesterol problem have lower HDL, greater BMI, and lower but not significantly different LDL. These group differences indicate that more extreme values on BMI and HDL may account for the change in associations found in the sensitivity analyses. Alternatively, there may be a non-linear relationship between BMI and LDL that accounts for the non-significance. For example, Drapeau et al. (2006) found significantly lower LDL values in morbidly obese women (BMI > 40) compared to moderately obese women (BMI 30–40). We, however, examined scatter plots and did not find any indication of a non-linear association between BMI and cholesterol (e.g., quadratic). Additionally, unlike previous studies (e.g., Shamai et al., 2011), we controlled for currently taking cholesterol medication and family history of cholesterol, thus, it is likely that the association between BMI and LDL was attenuated, indicating that cholesterol medication is a possible mechanism of lower cholesterol among those with higher BMI levels. Further, the contributions of CSA on BMI among women were beyond the contribution of other forms of maltreatment and household dysfunction.

5.1. Limitations and future directions

Despite the strengths of the study, the current study is not without limitations. First, prospective and longitudinal measurement, particularly of sexual abuse, is needed. Using alternative methods to measure CSA (e.g., collateral report, official documentation). Estimating changes in BMI as well as prospective measurement of HDL and LDL is needed. Relatedly, we are unable to form conclusions about how BMI is associated with cholesterol over time due to not having cholesterol data at multiple time points. Thus, even though our study used two waves of data, associations are more likely to resemble cross-sectional associations. Additionally, although HDL and LDL are clinically used indicators of health, more consideration has been given to the complexity of the connection between lipoproteins and health outcomes. Obesity not only impacts total values of LDL and HDL, but also effects the composition of lipoprotein molecules, which may be more indicative of cardiovascular disease (Stadler & Marsche, 2020). This may be especially true for LDL cholesterol (Superko & Gadesam, 2008). Although this information was not collected within the MIDUS data, it suggests that future research should further explore the impact of BMI as a mediator between CSA and cholesterol composition. Lastly, our sample was mostly White, middle class midlife adults; future research would do well to examine possible racial and socioeconomic differences.

5.2. Conclusion

BMI appears to be a possible pathway linking CSA to cholesterol levels, specifically HDL among women. While findings suggest that development of trauma-informed prevention and intervention efforts to reduce BMI would be beneficial for women who were sexually abused, continued research is needed to longitudinally substantiate the conclusions of the current study. Likewise, clinicians including

therapists, physicians, and dietitians should carefully consider the effects of CSA on BMI in the work with their patients.

CRediT authorship contribution statement

Michael Fitzgerald: Writing – review & editing, Writing – original draft, Supervision, Methodology, Conceptualization. **Haley Hall:** Writing – review & editing, Writing – original draft.

Data availability

Data will be made available on request.

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