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Childhood physical abuse and midlife physical health: Testing a multi-pathway life course model☆

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ABSTRACT

Although prior research has established that childhood abuse adversely affects midlife physical health, it is unclear *how* abuse continues to harm health decades after the abuse has ended. In this project, I assess four life course pathways (health behaviors, cognition, mental health, and social relation) that plausibly link childhood physical abuse to three midlife physical health outcomes (bronchitis diagnosis, ulcer diagnosis, and general physical health). These three outcomes are etiologically distinct, leading to unique testable hypotheses. Multivariate models controlling for childhood background and early adversity were estimated using data from over 3000 respondents in the Wisconsin Longitudinal Study, USA. The results indicate that midlife social relations and cognition do not function as pathways for any outcome. However, smoking is a crucial pathway connecting childhood abuse with bronchitis; mental health is important for ulcers; and BMI, smoking, and mental health are paramount for general physical health. These findings suggest that abuse survivors' coping mechanisms can lead to an array of midlife health problems. Furthermore, the results validate the use of etiologically distinct outcomes for understanding plausible causal pathways when using cross-sectional data.

Introduction

The routes by which childhood physical abuse affects midlife physical health is an important case for social scientists and health researchers interested in understanding the complex and multi-directional influences of social and biological systems. Though childhood abuse is recognized as a public health problem, the literature investigating the relationship between childhood abuse and adult health is incomplete. Most notably, few studies have assessed *how* childhood abuse has lifelong health effects, and no research has systematically assessed the relative importance of multiple mechanisms. As Kendall-Tackett notes, "Recent studies have established that childhood abuse makes people sick. The next logical question to ask is 'Why'" (Kendall-Tackett, 2003; p. xiii). Childhood physical abuse may directly cause poor adult health, especially in cases of extreme abuse that cause enduring forms of bodily harm. Yet, childhood physical abuse may also act indirectly as the catalyst for a range of social, behavioral, and emotional problems that are more proximate causes of midlife morbidity. Identifying and targeting potentially modifiable connections between childhood abuse and adult health can help reduce the lifelong burden of childhood violence.

This is the first project to assess multiple pathways between childhood physical abuse and different aspects of midlife physical health. The specific outcomes include a bronchitis diagnosis, an ulcer diagnosis, and a measure of general health. Three measures of midlife physical health are used to explore whether childhood physical abuse can predict different long-term adult health problems through an individual's more proximate response to childhood abuse (i.e., smoking vs. depression vs. dropping out of school). Importantly, these health outcomes have distinct etiologies, thereby permitting the development of testable hypotheses about plausible causal mechanisms linking childhood abuse with midlife health.

I focus on childhood physical abuse because it is relatively common for men and women (6%–30% of people in community-based samples report childhood physical abuse), and is more narrowly defined than inclusive measures of "childhood adversity"

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(Springer, Sheridan, Kuo, & Carnes, 2003). Without this conscious circumscription, it would be impossible to ascertain whether differential relationships between "childhood adversity" and the three outcomes were due to different types of "adversity" rather than due to a multiplicity of mechanisms associated with a *specific* type of childhood adversity (i.e., childhood physical abuse).

In sum, the current project has three primary substantive objectives: (1) to assess the effect of childhood physical abuse on midlife health, controlling for potentially confounding family background and childhood adversity variables, (2) to trace the health behaviors, cognition, mental health, and social relations pathways through which childhood physical abuse may affect midlife physical health, and (3) to examine gender differences in outcomes and pathways. Through addressing these substantive objectives, I also assess the feasibility of using etiologically based hypotheses to test life course processes when using cross-sectional data, and I examine whether abuse-related sample attrition necessitates a focus on midlife health, rather than later life health.

Childhood physical abuse and midlife health outcomes

Childhood abuse is linked with poor adult health outcomes; however, most research has focused on the mental health effects of sexual abuse in women (Neuman & Houskamp, 1996; Springer et al., 2003). The independent health effects of childhood physical abuse among men and women have received less attention and most of this research has been conducted on clinical samples. The best evidence from general population surveys of men and women is that childhood physical abuse leads to increased psychiatric disorders, risky health behaviors, and adult physical health conditions (Goodwin, Hoven, Murison, & Hotopf, 2003; MacMillan et al., 1999; Shaw & Krause, 2002; Thompson, Kingree, & Desai, 2004).

Many studies examine the health effects of childhood abuse in samples of women only, despite the fact that childhood physical abuse is equally prevalent for boys and girls (U.S. Department of Health and Human Services, Administration on Children, Youth and Families, 2002). The few studies that have specifically explored sex differences yield discrepant results including no sex differences and abused women suffering more (Irving & Ferraro, 2006; Springer, Sheridan, Kuo, & Carnes, 2007; Thompson et al., 2004).

Adverse health consequences of childhood abuse are not necessarily equivalent – or even present – throughout all of adulthood; yet, little research examines age variations in the health effects of childhood adversity. In one notable exception using a decade of prospective longitudinal data, O'Rand and Hamil-Luker (2005) establish that early life adversity hastens the onset of adult health problems. These results mirror the well-established findings that socioeconomic disparities in health are greatest at midlife and converge at later life (Beckett, 2000; House et al., 1994). The two main reasons for heightened health disparities at midlife are early onset of health problems and selective attrition due to adversity. These findings suggest that midlife is substantively and methodologically the best time to study the relationship between childhood abuse and adult health inequalities – however, this has not yet been directly tested.

Plausible pathways linking childhood physical abuse to adult physical health

Research has begun to explore mechanisms through which childhood abuse could affect adult physical health, though most research focuses on childhood sexual abuse (Kendall-Tackett, 2003). These mechanisms generally include behavioral strategies to cope with memories of violence (i.e., smoking), emotional and psychological responses (i.e., depression and anxiety), and secondary stressors (i.e., poor adult relationships) (Kendall-Tackett, 2003; Pearlin, 1989).

Health behaviors are the most researched plausible mechanism; however, the vast majority of this research examines health behaviors as outcomes and then infers that health behaviors mediate the relationship between child adversities and adult health outcomes. In a rare exception, Dong, Dube, Felitti, Giles, and Anda (2003) specifically modeled mediation and found that drinking, drug use, and risky sex accounted for up to 50% of the relationship between adverse childhood experiences and liver disease. Unfortunately, the study only included adults with a regular health care provider, limiting the generalizability of the findings. Furthermore, Dong et al. (2003) examined a composite childhood adversity measure, making it impossible to discern pathways specifically associated with childhood physical abuse.

Adult social relationships are another possible pathway through which childhood physical abuse can cause adult health problems. Childhood abuse has been associated with poor marital quality, social isolation, and negative family interactions (Kendall-Tackett, 2003; Shaw & Krause, 2002) – all of which can adversely affect adult health. Although the social relations literature focuses almost exclusively on childhood sexual abuse, some social relations have been linked with childhood physical abuse and could plausibly mediate the link between childhood physical abuse and adult physical health (Shaw & Krause, 2002). For example, Shaw and Krause (2002) found that negative family interactions partially attenuated the relationship between childhood physical abuse and adult chronic health problems.

Childhood abuse may also have consequences for cognitive resources, which could in turn affect adult physical health. Childhood abuse is associated with poor educational outcomes (Perez & Widom, 1994) and there is an extensive body of literature linking education with adult health (Marmot, Ryff, Bumpass, Shipley, & Marks, 1997). While prior childhood abuse studies have included education as a control variable, none have explicitly assessed the potential mediating effect of cognitive resources.

Mental health is a potential route through which childhood physical abuse could exert negative effects on adult physical health. Goodwin et al. (2003) examined mental health as a plausible pathway connecting childhood physical abuse to specific physical health outcomes using a cross-sectional, population-based sample. They found that the relationship between childhood physical abuse and recurring stomach problems, ulcers, and migraines remained significant but was substantially attenuated with the inclusion of panic attacks, generalized anxiety disorder, major depression, and alcohol/substance use disorders.

Few studies have examined gender differences in pathways connecting childhood abuse to adult physical health. Irving and Ferraro (2006) found evidence that personal control attenuated the effect of childhood physical violence by non-parents in a female-only sample but not in a male-only sample. However, this gender difference was not statistically significant.

In sum, few population-based studies have explored any possible pathway linking childhood physical abuse to a specific health outcome, and no prior research has assessed the relative importance of different plausible pathways for multiple outcomes. In addition, it is unknown whether these plausible pathways vary by gender. Documenting plausible mechanisms linking childhood abuse with adult health is crucial for understanding how early disadvantage persists across a lifetime and for developing targeted interventions to alleviate the lifelong health burden of childhood abuse.

Multi-pathway life course model

Based on existing literature, I synthesized an array of possible mechanisms linking childhood physical abuse to midlife physical

health. I bring these diverse mechanisms into a single model, permitting comparison of different mediators by gender and controlling for family background and childhood adversities. This multi-pathway life course model contains four potential pathways: health behaviors, cognition, mental health, and social relations. The health behaviors pathway includes obesity, smoking, and problem drinking. The cognition pathway includes education and midlife cognitive ability. The mental health pathway includes scales of depressive symptoms, anxiety, and anger. The social relations pathway includes marital quality, social support, emotional support, social activities with family, and social activities with friends.

I chose three dependent variables for these analyses. Bronchitis/emphysema was selected as an outcome because it is primarily caused by one of the potential mediators – smoking. According to the Centers for Disease Control and Prevention (CDC), chronic bronchitis and emphysema account for 59% of all smoking-attributable diseases (CDC, 2003). Ulcers were chosen as the second diagnostic outcome for two main reasons. First, the relationship between childhood physical abuse and ulcers is widely accepted and well-established across multiple datasets and samples (Drossman et al., 1990; Goodwin et al., 2003). Second, the prevailing biopsychosocial understanding of ulcer etiology leads to empirical predictions that differ substantially from bronchitis/emphysema. Specifically, research has established that a bacterium called Helicobacter pylori (Hp) is implicated in the vast majority of ulcers (NIH Consensus Development Panel on Helicobacter pylori in Peptic Ulcer Disease, 1994). Although Hp is present in almost all patients with ulcers, the reverse logic is not true; only 20% of people with Hp infection develop ulcers. The primary determinant of ulcer development given Hp infection is biological sensitization to stress resulting from prior traumatic events combined with current stress, smoking, drinking, and/or sleeplessness (Levenstein, 2000; Murison, 2001; Overmier & Murison, 2005). Importantly, animal experiments suggest that gastrointestinal vulnerability due to prior trauma is a stronger predictor than current stressors (Overmier & Murison, 2005).

These different etiologies provide analytic leverage to assess the proposed causal ordering of the multi-pathway model despite using largely cross-sectional data. In other words, the causal model of mediation is supported each time the data validate an etiologically based prediction about specific pathways and specific outcomes. For example, the implied causal model is supported if smoking is a stronger predictor and mediator for a bronchitis/emphysema diagnosis vs. an ulcer diagnosis. In addition, the etiology of ulcers suggests that current stress and coping behaviors (such as smoking and drinking) may modestly attenuate the relationship between childhood physical abuse and ulcers. Therefore, the causal model predicts that the mental health and health behavior pathways should only partially mediate the relationship between abuse and ulcer diagnoses. Finally, the causal model is supported if multiple pathways are implicated in the connection between childhood physical abuse and the summary indicator of medical diagnoses – because this summary indicator is a general physical health outcome. In sum, using etiologically distinct outcomes and epidemiologically established mediators enables a variety of tests for causal ordering.

Attrition analyses

Past research suggests that the health effects of early life adversity are most pronounced at midlife due to differential attrition and hastened onset of health problems; however, it is unclear whether this relationship holds for childhood abuse. Unfortunately, there are too few respondents with bronchitis or ulcer diagnoses past midlife to assess the plausibility of differential age of onset due to childhood abuse. However, it is possible to explore whether people who were physically abused as children are more likely to leave the study between midlife and later life *and* whether differential attrition is due to mortality and morbidity. If abuse survivors are more likely to leave the study for health reasons, this would support the importance of examining midlife health because a focus on later life health would miss a substantial portion of abuse-related health problems and, therefore, provide a biased view of the relationship between childhood abuse and adult health.

Data and methods

Data for the current project come from the Wisconsin Longitudinal Study (WLS) – a large, longitudinal study of education, careers, health, and aging (Sewell, Hauser, Springer, & Hauser, 2003). The WLS is well suited for exploring the long-term health effects of childhood abuse because it is a population-based sample of men and women selected without regard to health status. Briefly, the WLS began with a 1/3 random sample ($N = 10{,}317$) of adolescent males and females who graduated from Wisconsin high schools in 1957. In 1975, one random sibling was selected for each graduate and these siblings were then interviewed in 1977, 1994, and 2005. The 1994 sibling mail questionnaire (but not the graduate questionnaire) contained items on childhood physical abuse; therefore, the current analyses are based on data from these sibling respondents. The specific samples used for this project were truncated to people who had complete data on all variables used in these analyses (see Appendix A for sample selection information). The medical diagnoses models have a sample of 3317 respondents, bronchitis models have a sample of 3393 respondents, and ulcer models have a sample of 3403 respondents. There were no significant differences in any of the variables due to missing data, thereby lessening concern about missing data bias (results available upon request).

The all cause attrition analyses were based on the sample who: completed both the 1994 phone and mail surveys; had complete data on childhood physical abuse, demographics, and childhood context variables; and who either completed both or neither the 2005 mail and 2005 phone surveys ($n = 3635$). The death and illness attrition analyses were further restricted to respondents who did not complete the 2005 mail and phone surveys due to death or severe illness ($n = 2892$).

Independent variable

Childhood physical abuse is the main independent variable of interest and was measured in 1994 with questions derived from the Conflict Tactics Scale (Straus, Gelles, & Steinmetz, 1981). In separate questions about each parent, respondents were asked whether their mother or father "slapped, shoved, or threw things at them" before age 17. Those who reported "some" or "a lot" of abuse by either or both parents, as opposed to "a little" or "not at all," were counted as having experienced childhood physical abuse. Dichotomizing childhood physical abuse to exclude infrequent abuse is consistent with prior research (Dong et al., 2003; Felitti et al., 1998; Goodwin et al., 2003; Irving & Ferraro, 2006) and helps assure that the measure captures abuse rather than corporal punishment parenting practices that were common when the respondents were children. Nonetheless, sensitivity tests demonstrated that the results were robust to an array of abuse specifications including dichotomous measures cut at different degrees of abuse severity and polytomous operationalizations of abuse frequency (results available upon request).

Dependent variables

Medical diagnoses were measured in 1994 using a modified version of the Duke Older Adults Research Survey (Duke University Center for the Study of Aging and Human Development, 1978). Respondents reported whether a medical professional had ever diagnosed them with any of the following 17 medical conditions (allergies, anemia, arthritis/rheumatism, asthma, serious back trouble, bronchitis/emphysema, cancer, circulation problems, colitis, diabetes, heart trouble, high blood pressure, high cholesterol, kidney/bladder problems, chronic liver trouble, multiple sclerosis, and ulcer). Accounting for control variables, childhood physical abuse significantly predicted allergies, arthritis/rheumatism, asthma, bronchitis/emphysema, circulation problems, high blood pressure, and ulcers.

The medical diagnoses measure was a sum of all diagnoses for respondents who answered each of the diagnoses questions. Because the medical diagnoses summary indicator was highly skewed, I used a started natural log to make it more normally distributed. However, the results were robust to a variety of specifications including a non-logged summary indicator, a summary indicator not limited to respondents who answered all questions, and dichotomous indicators (results available upon request). I analyzed self-reported bronchitis/emphysema and ulcer diagnoses as dichotomous outcomes.

Mediators

All of the mediating variables were measured during the 1994 survey, except education, which was taken from the 1977 surveys. When 1977 education data were not available – largely because the participant did not join the study until 1994 – the 1994 variable was substituted.

I assessed three potential mediating health behaviors: BMI categories calculated at the CDC recommended cut points (normal < 25; overweight $= 25$–29.9; obese > 29.9); whether the respondents reported that they were current smokers, former smokers, or nonsmokers; and whether they reported any one of five drinking problems (dummy variable). Examples of drinking problems included: "Has drinking caused a problem for you at work?" and "Has drinking created problems between you and your spouse, children, parents, or other near relatives?"

Cognition variables included midlife cognitive ability and educational attainment. Midlife cognitive ability was measured by a 10-item, abbreviated version of the similarities subset of the Wechsler Adult Intelligence Scale (WAIS) (Wechsler, 1987). The WAIS similarities test asks how two things are alike (i.e., table and chair) with responses coded "0", "1", or "2" based on the correctness and abstractness of answers. Educational attainment was coded in years with the exception that all bachelor's degrees were coded as 16 and all education past a master's degree was coded as 19.

The mental health pathway included scales for depressive symptoms, anxiety, and anger. Depressive symptoms were assessed with a 20-item Center for Epidemiologic Studies Depression Scale (CES-D). Anxiety and anger were each assessed with 10 items from Spielberger's Anxiety and Anger scales (Radloff, 1977). For all mental health items, respondents were asked to report how many days in the past week they experienced each item. Respondents were included in the analyses if they answered at least 10 of the 20 depression questions, although the results were substantively equivalent for the sample that completed all 20 items. To construct each mental health scale, I divided the total score by the number of items answered, multiplied this value by the number of items in the scale, and rounded the result.

I included several measures for the social relations pathway. Marital closeness was assessed for married respondents and was coded "1" if respondents felt "very close" or "close" to their current spouse, compared to "somewhat close" or "not at all close".[1] The social support variable was a dichotomous indicator coded "1" if the respondent had someone other than a spouse from whom they could borrow $250, ask for help with a personal problem, *and* ask for help if sick for a week or more. Emotional support was coded "1" if someone had both a person in their family and a friend outside of their family with whom they could "really share [his/her] private feelings and concerns." Finally, I included one measure of social activities with friends and one measure of social activities with family – both operationalized as the number of days engaged in social activities [with friends or family] during the past four weeks.

Control variables

Demographic controls included sex and age of the respondent at the 1994 mail survey. In addition, childhood context was accounted for by including three variables that had previously been determined to parsimoniously capture the influence of ten different childhood adversity and family background measures: father's education (in years) when respondent was 16, growing up on a farm (yes/no), and parents having serious marital problems during the respondent's childhood (yes/no) (Springer et al., 2007). Unfortunately, the 1994 instrument did not contain childhood sexual abuse measures or valid indicators of childhood emotional abuse.

Attrition

I constructed two dichotomous indicators to assess whether abuse survivors were more likely than non-abused respondents to leave the study between 1994 and 2005. Both items were coded "0" if the respondent completed both the 2005 phone and mail survey. The first indicator assessed general non-response (all cause attrition) and was coded "1" if the respondent did not complete the 2005 telephone and mail survey, for any reason. The second item measured non-response due to morbidity or mortality and was coded "1" if the respondent died between 1994 and 2005 or was too ill to complete the 2005 phone and mail survey.

Analytic strategy

I began the analyses by assessing the possibility of abuse-related attrition between midlife and later life controlling for sex, age, and childhood context. I then turned to the mediation models. Theoretically plausible mediators were determined based on prior literature; however, statistical analyses were necessary to discern which variables analytically qualified as mediators. I utilized Baron and Kenny's (1986) well-established causal steps approach to identify qualifying mediators.

After determining qualifying mediators, I conducted five nested analyses. First, I examined the baseline effect of childhood physical abuse on each physical health outcome, accounting for age and sex. Second, I controlled for childhood context to best establish the independent effect of childhood physical abuse. Third, I examined each mediating pathway linking childhood physical abuse to each outcome, controlling for childhood context. In the fourth stage of analyses, I assessed the attenuating effect of all qualifying mediator

[1] As described in the analytic strategy section, each potential mediator was tested individually as detailed by Baron and Kenny (1986). Therefore, when assessing marital closeness, I examined only married couples.

variables in the same model, accounting for all controls. Finally, I examined gender differences.

To examine gender differences, I started by determining whether childhood physical abuse predicted each outcome in single-sex samples. I then tested an interaction between childhood physical abuse and sex for each outcome to determine whether the consequences of abuse differed significantly for men and women. Lastly, I assessed whether the pathways varied by sex in cases where abuse predicted the outcome in both single-sex samples or when the interaction of sex and abuse was significant.

Finally, to further assess the relative importance of smoking as a mediator, I conducted Sobel–Goodman tests for smoking on each outcome, controlling for all qualifying variables. Ordinary least squares regression was used to analyze the summary indicator of medical diagnoses and logistic regression was used for the specific diagnoses and attrition analyses. All analyses were conducted with Stata SE (StataSE, 2006).

Results

Attrition analyses

Respondents who reported childhood physical abuse in 1994 were significantly more likely than non-abused respondents to leave the study prior to the 2005 survey in general, and specifically due to death or illness (Appendix B). The magnitude of these effects was not trivial (i.e., OR = 1.47), further demonstrating the adverse health effects of childhood physical abuse and underscoring the importance of focusing on midlife rather than later life health.

Descriptive statistics

Table 1 presents the descriptive and bivariate statistics (by abuse status) for outcomes, control variables, and variables that qualified as analytic mediators for at least one outcome. Twelve percent of all respondents reported childhood physical abuse (396/3317 = 12%); abuse survivors grew up with less-educated fathers and were more likely to report parental marital problems than non-abused respondents. Abuse survivors had significantly worse physical health than non-abused respondents (2.6 medical diagnoses vs. 2.0 medical diagnoses, respectively). Almost one out of five abuse survivors reported a bronchitis/emphysema diagnosis compared to 13% of non-abused respondents. Ulcers were also more common among abuse survivors with 13% reporting an ulcer diagnosis compared to 8% of non-abused respondents.

In terms of mediators, none of the cognition or social relations variables qualified as mediators for any of the outcomes (Baron & Kenny, 1986). In addition, problem drinking was never a mediator (results available upon request). However, for the qualifying mediators, abuse survivors generally fared worse. They were significantly more likely to be obese (32%), current smokers (21%), and former smokers (45%) compared to non-abused respondents (23%, 17%, and 37%, respectively). Abuse survivors also reported significantly worse mental health than non-abused respondents. In sum, the bivariate results show that abuse survivors had worse physical health, poorer mental health, and greater health risks than non-abused respondents.

Plausible pathways linking childhood physical abuse and medical diagnoses at midlife

Childhood physical abuse predicted a 20% increase in medical diagnoses accounting for demographics, and a 17% increase accounting for demographics and childhood context (columns 1 and 2 in Table 2). Health behaviors – obesity, in particular – attenuated the effect of childhood physical abuse, although abuse continued to significantly predict a 13% increase in medical diagnoses accounting for health behaviors (column 3 in Table 2). Mental health mediators also partially attenuated the relationship between childhood physical abuse and medical diagnoses (column 4 in Table 2). Depression, but not anxiety or anger, predicted medical diagnoses accounting for abuse and controls. In the final model, childhood physical abuse continued to predict a 10% increase in medical diagnoses accounting for *all* variables (column 5 in Table 2).

Turning to sex differences, childhood physical abuse significantly predicted an increase in medical diagnoses in both the male-only and female-only samples; however, the interaction of sex and childhood physical abuse was not significant in the pooled sample. Further analyses indicated no significant variations in the pathways by sex. These results suggest that childhood physical abuse affects the health of midlife men and women to the same degree and through similar mechanisms (results available upon request).

Table 1
Descriptive statistics for all variables used in analyses, by childhood physical abuse ($N = 3317$).

	Abuse survivors ($n = 396$)		Non-abused ($n = 2921$)	
	Mean	SD	Mean	SD
Demographics				
Age	53.91	6.78	53.80	7.31
Female (%)	0.53		0.52	
Childhood context				
Father's education*	9.50	3.37	9.93	3.37
Farm background (%)	0.18		0.20	
Parent marital Problems (%)**	0.43		0.14	
Outcomes				
Number of medical diagnoses**	2.58	2.08	2.04	2.00
Bronchitis/emphysema diagnosis (%)**	0.19		0.13	
Ulcer diagnosis (%)**	0.13		0.08	
Mediators				
Health behaviors				
Overweight (%)	0.38		0.41	
Obese (%)**	0.32		0.23	
Current smoker (%)*	0.21		0.17	
Former smoker (%)**	0.45		0.37	
Mental health				
Depression**	21.28	19.64	16.65	15.83
Anger**	9.72	10.35	7.56	8.50
Anxiety**	15.74	12.89	13.10	11.72

*$p < 0.05$; **$p < 0.01$.
Notes: means and standard deviations are presented for continuous measures; proportions are shown for categorical variables. Two-tailed *t*-tests were used to assess significant differences by childhood physical abuse.

Plausible pathways linking childhood physical abuse and bronchitis/emphysema at midlife

Childhood physical abuse was associated with a significant 57% increase in the odds of a bronchitis/emphysema diagnosis accounting for age and sex (column 1 in Table 3), compared to a still significant 44% increase after including childhood context variables (column 2 in Table 3). Controlling for health behaviors (column 3 in Table 3) completely mediated the relationship between childhood physical abuse and bronchitis/emphysema. As hypothesized, smoking strongly and significantly predicted a bronchitis/emphysema diagnosis. Specifically, current smoking (vs. having never smoked) was associated with a 95% increase in the odds of a bronchitis/emphysema diagnosis.

Table 2
Ordinary least squares regression analyses of plausible pathways linking childhood physical abuse and number of medical diagnoses at midlife ($n = 3317$).

	Demographics			Childhood context			Health behaviors			Mental health			Full model		
	Coef.	SE	%Δ	Coef.	SE	%Δ	Coef.	SE	%Δ	Coef.	SE	%Δ	Coef.	SE	%Δ
Childhood physical abuse	0.186**	0.032	20.4%	0.157**	0.033	17.0%	0.119**	0.033	12.6%	0.126**	0.033	13.4%	0.094**	0.033	9.9%
Demographics															
Age	0.016**	0.001	1.6%	0.017**	0.001	1.7%	0.016**	0.001	1.6%	0.019**	0.001	1.9%	0.018**	0.001	1.8%
Female	0.188**	0.021	20.7%	0.181**	0.021	19.8%	0.217**	0.022	24.2%	0.166**	0.021	18.1%	0.200**	0.022	22.1%
Childhood context															
Father's education				0.000	0.003	0.0%	0.001	0.003	0.1%	0.003	0.003	0.3%	0.003	0.003	0.3%
Farm background				−0.075**	0.027	−7.2%	−0.073**	0.027	−7.0%	−0.068*	0.027	−6.6%	−0.066*	0.026	−6.4%
Parent marital problems				0.097**	0.029	10.2%	0.100**	0.028	10.5%	0.081**	0.028	8.4%	0.085**	0.028	8.9%
Health behaviors															
Overweight							0.085**	0.024	8.9%				0.082**	0.024	8.5%
Obese							0.261**	0.029	29.8%				0.234**	0.028	26.4%
Current smoker							0.030	0.030	3.0%				0.012	0.029	1.2%
Former smoker							0.090**	0.023	9.4%				0.087**	0.023	9.1%
Mental health[a]															
Depression (×100)										0.659**	0.086	93.3%	0.629**	0.085	87.6%
Anger (×100)										0.282	0.151	32.6%	0.215	0.150	24.0%
Anxiety (×100)										0.010	0.126	1.0%	0.022	0.125	2.2%
Constant	−0.058	0.080	−5.6%	−0.079	0.092	−7.6%	−0.193*	0.093	−17.6%	−0.322**	0.094	−27.5%	−0.399**	0.095	−32.9%
Adjusted R^2		0.067			0.073			0.100			0.109			0.131	

*$p < 0.05$; **$p < 0.01$.
Notes: unstandardized regression coefficients for the started natural log of medical diagnoses are presented in the coefficient columns. The %Δ column indicates how much a one unit change in X affects the percent change in medical diagnoses based on the formula $(100 \times (\exp(B) - 1)$.
[a] The coefficients for the mental health variables are multiplied by 100 because the coefficients measure the effect of a one unit increase in the mental health scales and the coefficients are, therefore, quite small.

Unlike the health behaviors pathway, childhood physical abuse continued to significantly predict a bronchitis/emphysema diagnosis accounting for depression, anxiety, anger, and other control variables (column 4 in Table 3). In the full model (column 5 in Table 3), childhood physical abuse did not significantly predict a bronchitis/emphysema diagnosis, as expected given the already established attenuating effect of health behaviors.

Focusing on sex differences, childhood physical abuse predicted a bronchitis/emphysema diagnosis in the female-only sample but not in the male-only sample; however, the interaction of sex and childhood physical abuse in the pooled sample was not significant (results available upon request). Possible sex differences in pathways were not tested because childhood abuse only predicted bronchitis/emphysema in the female sample.

Plausible pathways linking childhood physical abuse and ulcers at midlife

Childhood physical abuse predicted a significant 76% increase in the odds of an ulcer diagnosis accounting for age and sex (column 1

Table 3
Logistic regression analyses of plausible pathways linking childhood physical abuse and bronchitis/emphysema at midlife ($n = 3393$).

	Demographics		Childhood context		Health behaviors		Mental health		Full model	
	OR	95% C.I.	OR	95% C.I.	OR	95% C.I.	OR	95% C.I.	OR	95% C.I.
Childhood physical abuse	1.57**	(1.20–2.05)	1.44*	(1.08–1.90)	1.28	(0.97–1.70)	1.37*	(1.03–0.181)	1.24	(0.93–1.65)
Demographics										
Age	1.00	(0.98–1.00)	1.00	(0.98–1.00)	1.00	(0.98–1.01)	1.00	(0.99–1.02)	1.00	(0.99–1.02)
Female	2.05**	(1.67–2.51)	2.01**	(1.64–2.47)	2.22**	(1.79–2.75)	1.97**	(1.60–2.42)	2.16**	(1.74–2.69)
Childhood context										
Father's education			1.01	(0.97–1.38)	1.01	(0.98–1.04)	1.01	(0.98–1.04)	1.01	(0.98–1.04)
Farm background			0.93	(0.72–1.21)	0.96	(0.74–1.24)	0.94	(0.72–1.22)	0.96	(0.74–1.25)
Parent marital problems			1.34*	(1.05–1.71)	1.35*	(1.05–1.72)	1.31*	(1.02–1.67)	1.31*	(1.03–1.68)
Health behaviors										
Overweight					1.13	(0.89–1.43)			1.12	(0.88–1.42)
Obese					1.78**	(1.38–2.30)			1.71**	(1.32–2.20)
Current smoker					1.95**	(1.49–2.55)			1.91**	(1.46–2.50)
Former smoker					1.41**	(1.12–1.77)			1.39**	(1.11–1.75)
Mental health										
Depression							1.00	(1.00–1.01)	1.00	(0.99–1.01)
Anger							1.01	(1.00–1.02)	1.01	(1.00–1.02)
Anxiety							1.00	(0.99–1.02)	1.01	(0.99–1.02)
Pseudo R^2	0.022		0.024		0.040		0.031		0.045	
Chi^2; d.f.	60.37; 3		66.88; 6		111.80; 10		84.82; 9		124.66; 13	

*$p < 0.05$; **$p < 0.01$.
Notes: odds ratios (exponential log-odds) and 95% confidence intervals are presented.

Table 4
Logistic regression analyses of plausible pathways linking childhood physical abuse and ulcers at midlife ($n = 3403$).

	Demographics		Childhood context		Health behaviors		Mental health		Full model	
	OR	95% C.I.	OR	95% C.I.	OR	95% C.I.	OR	95% C.I.	OR	95% C.I.
Childhood physical abuse	1.76**	(1.28–2.40)	1.68**	(1.22–2.33)	1.61**	(1.16–2.35)	1.55**	(1.12–2.16)	1.50*	(1.08–2.09)
Demographics										
Age	0.99	(0.98–1.01)	0.99	(0.97–1.00)	0.99	(0.98–1.01)	0.99	(0.98–1.01)	0.99	(0.98–1.01)
Female	1.19	(0.93–1.51)	1.17	(0.92–1.49)	1.23	(0.97–1.57)	1.14	(0.89–1.45)	1.20	(0.93–1.53)
Childhood context										
Father's education			0.98	(0.94–1.01)	0.98	(0.94–1.01)	0.98	(0.94–1.02)	0.98	(0.94–1.02)
Farm background			0.90	(0.66–1.23)	0.94	(0.69–1.29)	0.92	(0.67–1.26)	0.95	(0.70–1.31)
Parent marital problems			1.11	(0.82–1.50)	1.09	(0.80–1.48)	1.06	(0.78–1.44)	1.05	(0.77–1.42)
Health behaviors										
Current smoker					1.87**	(1.36–2.57)			1.79**	(1.30–2.47)
Former smoker					1.34*	(1.02–1.77)			1.35*	(1.02–1.78)
Mental health										
Depression							1.02**	(1.01–1.03)	1.02**	(1.01–1.03)
Anger							1.00	(0.98–1.02)	1.00	(0.98–1.01)
Anxiety							1.00	(0.98–1.01)	1.00	(0.98–1.01)
Pseudo R^2	0.007		0.008		0.016		0.025		0.031	
Chi^2; d.f.	14.55; 3		16.71; 6		31.52; 8		51.02; 9		63.74; 11	

*$P < 0.05$; **$p < 0.01$.
Notes: odds ratios (exponential log-odds) and 95% confidence intervals are presented.

in Table 4) and a significant 68% increase in the odds of an ulcer diagnosis accounting for childhood context, age, and sex (column 2 in Table 4). Childhood physical abuse remained a significant predictor of an ulcer diagnosis through all mediation analyses, with only smoking and depression modestly attenuating the association. Childhood physical abuse still predicted a 50% increase in the odds of an ulcer diagnosis in the full model, demonstrating that the plausible pathways leave a large effect unaccounted for (column 5 in Table 4).

As with bronchitis/emphysema, childhood physical abuse significantly predicted an ulcer diagnosis at midlife in the female-only sample but not in the male-only sample. Furthermore, the effects were not significantly different from each other as evidenced by the non-significant interaction of childhood physical abuse and sex in the pooled sample (results available upon request).

Percent of association explained by each plausible pathway

Table 5 presents a direct comparison of plausible pathways by outcome. Specifically, the percentages in Table 5 compare the abuse coefficient for each plausible pathway (columns 3–5 in Tables 2–4) with the model accounting for demographics and childhood context (column 2 in Tables 2–4, respectively) for each outcome.[2]

As predicted, the results in Table 5 show that the health behaviors pathway (which contains smoking) explains more of the association between childhood physical abuse and bronchitis/emphysema than between childhood physical abuse and the other outcomes. Indeed, the health behaviors pathway completely mediates the relationship between childhood physical abuse and a bronchitis/emphysema diagnosis. Further analyses using Sobel–Goodman tests of mediation demonstrate that smoking itself accounts for 13% of the relationship between abuse and bronchitis/emphysema – accounting for *all* other variables (Preacher & Hayes, 2008). This is in contrast to 7% and 1% for an ulcer diagnosis and the summary indicator of diagnoses, respectively (results available upon request). These bootstrapping analyses provide additional support for the proposed theoretical framework. Finally, the results in Table 5 also support the hypothesis that the mediating variables account for less of the association between childhood physical abuse and ulcers than between childhood physical abuse and other outcomes: approximately 20% and 40%, respectively.

Discussion

The findings from this multi-pathway, life course analysis demonstrate plausible explanations for how childhood physical abuse adversely affects midlife physical health. The results suggest that health behaviors and mental health may be crucial links between early childhood trauma and midlife physical health – whereas cognition and positive midlife social relationships appear inconsequential. Furthermore, the results indicate that the strength of plausible pathways varies by outcome in etiologically predictable ways, thereby supporting the use of epidemiological evidence as conceptual leverage to assess plausible causal processes when using cross-sectional data. Finally, the results provide strong evidence that childhood physical abuse is associated with health-related sample attrition past midlife.

As expected, smoking was an influential mediator of the relationship between childhood physical abuse and bronchitis/emphysema, whereas the relationship between childhood physical abuse and the medical diagnoses summary indicator was substantially mediated by both health behaviors and mental health. These results suggest that childhood physical abuse survivors may be differentially susceptible to specific health problems dependent on coping mechanisms. Furthermore, as hypothesized, childhood physical abuse continued to predict ulcer diagnoses, with only modest attenuation by smoking and depression. The findings provide suggestive evidence that childhood physical abuse may serve as a traumatic event linked with gastrointestinal sensitization – sensitization that precipitates ulcers when activated by smoking or depression among people with Hp infections. The constellation of these findings suggests that examining sole outcomes or

[2] For example, the 24% in the top left corner of Table 5 is the coefficient of abuse in the health behaviors model from Table 2 divided by the abuse coefficient in the childhood context model, subtracted from one: $(1 - (0.119/0.157) = 0.24)$. This value indicates that 24% of the association between childhood physical abuse and medical diagnoses can be accounted for by health behaviors variables. Note that the percentages for the logistic regression models are calculated based on the coefficients, not odds ratios.

Table 5
Percent of association between childhood physical abuse and each outcome explained by plausible pathways.

	Medical diagnoses	Bronchitis/emphysema	Ulcer
Health behaviors	24%**	31%	8%**
Mental health	20%**	14%*	15%**
Full model	40%**	41%	21%*

Childhood physical abuse is still significant at $^{*}p < 0.05$; $^{**}p < 0.01$.
Note: percent explained compares plausible pathway models to models controlling for age, sex, and all family context variables. The percentages for bronchitis and ulcers are derived from the abuse coefficients, not the odds ratios presented in the models.

mechanisms by which abuse affects later health underestimates its influence. Future research should extend these analyses to explore other outcomes. For example, to further assess the validity of the life course framework, I conducted sensitivity analyses exploring possible mechanisms connecting childhood physical abuse with high blood pressure and circulation problems. As etiologically predicted, BMI was the most influential mediator for high blood pressure, and smoking was the most influential mediator for circulation problems (results available upon request).

The results of the current project extend beyond prior research to demonstrate that poor mental health is not only an outcome – but it is also a mechanism through which childhood physical abuse may harm midlife physical health. Furthermore, the results underscore the importance of examining multi-faceted components of mental health (Aneshensel, 2002; Horwitz, 2002). Although each individual measure of mental health (depression, anger, and anxiety) qualified as an analytic mediator for all outcomes when examined separately, not all remained significant when included simultaneously. Depression was an important plausible mediator for the summary indicator of medical diagnoses and for an ulcer diagnosis. However, neither anxiety nor anger was significant for any outcome. These results indicate that specific aspects of mental health – depression in particular – may be partly responsible for the long-term physical health effects of childhood physical abuse.

The current project also provides additional support for the growing body of research suggesting that socially determined health inequalities are best measured at midlife (House et al., 1994; O'Rand & Hamil-Luker, 2005). Specifically, the current results are among the first to document higher attrition among abuse survivors, largely due to higher rates of mortality and morbidity among those who experienced childhood abuse. One implication of these results is that a focus on later life health would miss a substantial portion of abuse-related health problems, and could provide a biased view of the relationship between childhood abuse and adult health.

Some of the key findings of this project are indicated by the absence of significance. Specifically, I did not find support for the mediating role of cognition or midlife social relations despite an array of measures for both possible pathways. The unimportance of the social relations pathway corresponds well with Shaw and Krause's (2002) finding that emotional support does not mediate the relationship between childhood physical abuse and adult chronic conditions. Importantly, however, Shaw and Krause (2002) did find evidence of mediation using a measure of negative family interactions. It is, therefore, plausible that positive social relations do not buffer the adverse effect of childhood physical abuse; however, negative social interactions may act as secondary stressors and extend the trauma of childhood physical abuse into adulthood (Pearlin, 1989).

The results of this project provide evidence that the long-term physical health effects of childhood physical abuse do not vary significantly by sex – either in magnitude or plausible pathways. Although there have been few previous statistical tests of gendered pathways, the current results run counter to research on the health effects of gendered coping strategies (Rieker & Bird, 2005). Future research should further explore the possibility that some types of abuse (i.e., sexual abuse) may operate differentially for men and women.

Methodologically, this project improves on other existing research. I used a population-based cohort sample of men and women selected without regard to health status. Furthermore, I deliberately chose a narrowly defined form of childhood adversity to best establish the potential multi-faceted influence of one specific type of childhood adversity. In order to examine an independent effect of childhood physical abuse, I controlled for other possible childhood adversities that likely coexist with childhood physical abuse. Finally, I conducted analyses for each outcome on a consistent sample with complete data on all variables. This consistent sample alleviates the possible problem that abuse coefficient reductions are due to sample differences rather than mediation (Irving & Ferraro, 2006; Shaw & Krause, 2002).

In addition to these strengths, the current project has several possible limitations. For example, the childhood physical abuse measure draws from only two items. However, these two items are from a well-validated scale (Straus et al., 1981) and have performed well in previous studies (Shaw & Krause, 2002). Furthermore, the childhood physical abuse measures may be confounded by other types of childhood abuse – including sexual and/or emotional abuse – despite controls for childhood context and adversity. If so, this suggests that the results should be interpreted as the effect of childhood abuse in general, rather than the influence of childhood physical abuse. Finally, the childhood abuse measure may be limited because it is based on retrospective self-reports. However, existing literature on abuse measurement suggests that underreporting of abuse is the most likely consequence of retrospective reporting and of using few items to assess abuse (Hardt & Rutter, 2004). Therefore, the largest possible consequence of both these measurement limitations is that the magnitude of the relationship between childhood physical abuse and adult physical health determined in this study is a *conservative underestimate* of the true relationship.

Mood congruency bias may also be a potential problem; this may occur, for example, if depressed individuals "selectively recall negative experiences and hence may exaggerate or misrepresent the presence of childhood adversity" (Brewin, Andrews, & Gotlib, 1993). However, a recent review article found scant evidence of mood congruency bias (Hardt & Rutter, 2004). Furthermore, the results in the current project provide evidence against mood congruency bias. Controlling for mental health never eliminated the effect of childhood physical abuse, providing strong evidence against mood congruency bias in the current project.

It is also important to consider the possible consequences of exploring physician reported diagnoses as health outcomes. Specifically, by utilizing diagnoses as outcomes, the physical health indicators are confounded with access to health care. In order to assess this bias, I conducted sensitivity tests controlling for health insurance coverage and having a physical exam within the last 12 months. The results did not change substantially, thereby alleviating concerns about biases associated with diagnoses (results available upon request).

Finally, the current project is limited in making strong causal statements because the data are primarily cross-sectional. However, the life course framework draws from well-established etiologies of different outcomes, allowing for theoretical and empirical tests of plausible causal ordering. For example, epidemiologically I should find that current smoking predicts bronchitis much more than it predicts ulcers. This does turn out to be true. Is the reverse as plausible? There is no epidemiological evidence to support the idea that bronchitis would cause smoking more than ulcers would cause smoking. It is worthwhile to emphasize that these findings were very similar when contrasting current smoking to a measure combining never and former smoking. This sensitivity

test is necessary because it is highly likely that reverse causation drives the relationship between bronchitis and former smoking. This accumulation of evidence demonstrates the utility of employing an etiologically based framework for analyzing cross-sectional mediation data. However, even though the results are consistent with the proposed pathways model, longitudinal analyses will be necessary to establish causality.

Despite these limitations, this project makes several significant contributions to the existing literature and has implications for future research and practice. Understanding how childhood abuse continues to adversely influence health can help shape social service and public health interventions designed to alleviate the burden carried by survivors of childhood physical abuse. The current findings suggest that modification of select pathway variables such as smoking, obesity, and mental health may reduce the lifelong health burden of childhood physical abuse. Unfortunately, however, the results also indicate that childhood physical abuse among midlife adults cannot be completely undone. Specifically, the ulcer results fit squarely within the contemporary biopsychosocial understanding of ulcer etiology and suggest that childhood physical abuse creates gastrointestinal vulnerability that cannot be reversed. In sum, the results of the current project demonstrate that the ill health effects of childhood parental violence persist long into adulthood through complex and multi-faceted mechanisms.

Appendix A. WLS sibling sample selection

	N (% of previous row)
Invited to complete the 1994 mail survey	5751 (100)
…and returned the mail survey	4039 (70.2)
…and had two parents and could report on parental marital problems	3935 (97.4)
…and had complete data on all variables	3317 (84.2)

Appendix B. Logistic regression analyses of childhood physical abuse-related attrition between midlife and later life

	All cause attrition ($n = 3635$)		Death & illness attrition ($n = 2892$)	
	OR	95% CI	OR	95% CI
Childhood physical abuse	1.34**	(1.07–1.67)	1.47*	(1.02–2.12)
Demographics				
Age	1.02**	(1.01–1.03)	1.11**	(1.09–1.13)
Female	0.81**	(0.70–0.94)	0.65**	(0.50–0.84)
Childhood context				
Father's education	0.97*	(0.95–1.00)	0.95*	(0.92–0.99)
Farm	0.80*	(0.66–0.97)	0.74	(0.53–1.03)
Parent marital problems	0.89	(0.73–1.08)	0.95	(0.67–1.35)

*$p < .05$; **$p < .01$.

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