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Midlife self-reported social support as a buffer against premature mortality risks associated with childhood abuse

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Research has linked childhood abuse to a plethora of adverse health outcomes in adulthood[1,2]. However, whether positive experiences in adulthood much beyond cessation of abuse exposure can offset these adverse health risks remains unclear. Using a sample of 6,078 adults from the National Survey of Midlife Development in the United States (MIDUS), we examined whether adult self-reported social support decreased mortality risk associated with self-reported exposure to three types of childhood abuse: severe physical abuse, modest physical abuse and emotional abuse. Greater self-reported social support was related to reduced mortality risk; however, this relation was qualified by exposure to childhood abuse. For each type of abuse, self-reported social support was linked to a larger reduction in mortality risk among individuals reporting childhood abuse compared with those reporting minimal or no exposure to abuse. These findings suggest that supportive relationships in midlife can partly offset the mortality risks that seem to be set in motion by childhood experiences of abuse.

Childhood abuse is a relatively common occurrence in the United States. The lifetime prevalence of physical abuse is estimated to be between 16% and 18.1%; emotional abuse is more prevalent, with estimates ranging from 23.9% to 35.1%[3,4]. The short- and longer-term mental health consequences of abuse have been extensively documented[1,5,6]. More recently, studies have linked childhood abuse with physical health problems during adulthood, including higher rates of morbidity from respiratory disorders, some cancers and cardiovascular disease, as well as premature mortality during midlife[2,7,8]. Given the apparent health consequences of abuse, a pressing question is whether there are processes capable of buffering against, compensating for or reversing its effects[9].

Positive social relationships marked by high levels of warmth and support have been shown to mitigate the associations between a range of early life stressors (such as socioeconomic disadvantage, neglect, loss of a parent and parental divorce) and alterations in biological functioning thought to contribute to the development and progression of disease[10–13]. Many fewer studies have focused specifically on abuse, but the few that have similarly point to supportive relationships as an effective buffer. For example, in a study focusing on maternal and paternal harshness that included abuse, warmth from one parent attenuated the associations between harshness from the second parent and declining self-reported overall physical health and increasing BMI over the course of adolescence[14]. In a study that focused specifically on long-term health risks, childhood abuse was associated with more signs of multisystem dysregulation at midlife; importantly, this association was attenuated among those reporting high levels of parental warmth and affection[15].

It should be noted that these past studies have focused on parental warmth and support concurrent with exposure to adversity early in life. However, there exists a decades-long 'incubation' period between exposure to abuse during childhood and the emergence of health problems such as heart disease, cancer and stroke, which typically have their first onset in the middle and later stages of the lifespan. Notably, positive social experiences during these middle and later life stages have been linked to better health outcomes in many domains for which early abuse seems to confer risk[16,17]. As such, supportive relationships during middle and later decades of life may decelerate the poor health trajectories initially set in motion by early experiences of adversity, or compensate for them through other processes. We are aware of only one study that has examined this question in humans, and it did indeed find that a supportive family environment in adulthood protected women who were sexually abused as children from alterations in morning cortisol output[18]. Further support comes from rodent studies showing that environmental enrichment during adolescence and adulthood can reverse the effects of low maternal care during the postnatal period on adult amygdala and hippocampal plasticity[19,20]. Another notable limitation of previous studies is that they have focused on intermediary biological markers as outcomes. Although useful indicators of risk, these biomarkers do not reflect actual disease or disability, which raises questions about the clinical relevance of this phenomenon. This is an important question to address, as it can speak to the plausibility and significance of reversibility later in life[9].

We attempt to answer this question in the present study using data from the National Survey of Midlife Development in the United States (MIDUS). In previous analyses of this dataset, we reported that among women at midlife, reports of being abused during childhood were associated with a 1.2–1.6-fold higher risk of mortality over the subsequent two decades[7]. Extending those results, the current investigation tested whether self-reported social support during adulthood mitigated the mortality risks associated with self-reported abuse. Given that health effects of abuse are hypothesized to differ as a function of the nature and/or severity of the maltreatment[21,22], we examined different types of abuse (severe physical abuse, moderate physical abuse and emotional abuse) separately. We also considered a viable alternative explanation for any buffering associated with social support: namely, that it reflected the protective influence of other positive psychological resources associated with mortality, including positive affect, perceived control and purpose in life[23–25].

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Table 1 | Sample characteristics and descriptive data of study variables

	n (%)	Mean (s.d.)
Age		46.78 (12.90)
Gender		
Female	3,191 (52.5)	
Male	2,887 (47.5)	
Ethnicity		
European American	5,518 (90.8)	
African American	312 (5.1)	
Other	248 (4.1)	
Education		
<High school	595 (9.8)	
High school	1,674 (27.5)	
Some college	1,845 (30.4)	
College degree or some graduate school	1,296 (21.3)	
Master's or professional degree	668 (11.0)	
Medical conditions		
History of heart problems	784 (12.9)	
History of cancer	437 (7.2)	
Depression	760 (12.5)	
Health behaviours		
History of smoking regularly	3,111 (51.2)	
History of regular alcohol use	2,526 (41.6)	
Childhood abuse		
Emotional abuse	2,188 (36.0)	
Moderate physical abuse	1,594 (26.2)	
Severe physical abuse	695 (11.4)	
Social support		3.45 (0.46)
Deceased	1,038 (17.1)	

Analyses were based on data collected in 1995–1996 during the first wave of MIDUS, an ongoing national study on the development of health and well-being from midlife to older adulthood. Participants included 6,078 adults who completed questionnaires assessing childhood physical and emotional abuse and current social support, and who provided information on covariates, including demographic characteristics (age, gender, race and educational attainment), medical history (heart problems, cancer and depression) and health behaviours (smoking and alcohol use). Data on mortality were collected over the next two decades, to October 2015. Descriptive information on the sample is presented in Table 1. At baseline, when participants reported on abuse and support, they were approximately 47 years old. The gender distribution was fairly balanced, and the majority of participants were of European descent and had at least a high school diploma. Over a third of the sample reported experiencing some type of abuse during childhood, with emotional abuse being the most common type of abuse experienced. In general, participants reported high levels of midlife social support from all sources. Of the participants included in the present study, nearly a fifth died over the roughly 20-year follow-up period.

Cox proportional hazard models were estimated to test the buffering role of midlife social support against mortality risk associated with childhood abuse. These models provide estimates of hazard ratios (HR), which represent the change in probability of death at any particular point in time given a one-unit increase on the predictor variable. An HR less than one indicates decreased likelihood of death whereas an HR greater than one indicates increased likelihood of death.

To determine the relative contributions of abuse, social support and their interaction to mortality risk above and beyond traditional risk factors, we first examined the associations between mortality risk and covariates, which included demographic characteristics (age, gender, race and educational attainment), medical history (heart problems, cancer and depression) and health behaviours (smoking and alcohol use). As shown in Table 2, older age, male gender and lower educational attainment were associated with higher mortality risk. African Americans compared with European Americans were also at higher risk for mortality. As expected, medical conditions, including heart disease, cancer and depression, were associated with higher mortality risk. With respect to health behaviours, smoking, but not alcohol consumption, was associated with higher risk for mortality.

To this base model, we added abuse, self-reported social support and their interactions in subsequent steps, focusing first on self-reported severe physical abuse. As displayed in Table 3, severe physical abuse was not associated with mortality risk, as we previously reported[7]. Higher social support, however, was associated with lower mortality. Consistent with hypotheses, there was a significant severe physical abuse by social support interaction, suggesting a buffering effect of social support. Indeed, follow-up tests indicated that the association between social support and reduced mortality risk varied according to reported experiences of childhood severe physical abuse. Specifically, the association between social support and lower mortality was stronger among individuals reporting severe abuse (HR = 0.74, 95% CI = 0.64–0.85, $P < 0.001$) compared with those who did not (HR = 0.92, 95% CI = 0.86–0.98, $P = 0.016$).

Paralleling the findings above, self-reported moderate physical abuse was not related to mortality whereas self-reported social support was, as displayed in Table 3. In line with our hypotheses, there was a significant interaction between moderate physical abuse and social support. As above, the association between social support and lower mortality was stronger among those reporting exposure to moderate physical abuse in childhood (HR = 0.81, 95% CI = 0.73–0.89, $P < .001$) versus those not endorsing such abuse (HR = 0.92, 95% CI = 0.86–0.99, $P = 0.031$).

Consistent with the patterns above, self-reported emotional abuse on its own was unrelated to mortality (Table 3). However, there was a main effect of self-reported social support and an interaction effect between emotional abuse and social support. Again, the association between self-reported social support and lower mortality was stronger among individuals who reported exposure to childhood emotional abuse (HR = 0.80, 95% CI = 0.73–0.88, $P < .001$) compared with those who did not (HR = 0.93, 95% CI = 0.86–1.00, $P = .051$).

We then tested whether social support might be a proxy for other protective factors associated with mortality. First, we created a psychological resources composite variable based on measures positive affect, perceived control and purpose in life, and then we statistically adjusted for this variable and examined whether it interacted with childhood abuse to predict mortality. Higher levels of psychological resources were associated with lower mortality (HR = 0.82, 95% CI = 0.75–0.89, $P < .001$), but did little to change the abuse by support interaction effects. The interactions remained significant and the hazard ratios remained relatively unchanged (severe physical abuse: HR = 0.80, 95% CI = 0.69–0.93, $P = 0.003$; moderate physical abuse: HR = 0.84, 95% CI = 0.77–0.92, $P = 0.048$; emotional abuse: HR = 0.89, 95% CI = 0.79–1.00, $P = 0.045$). Furthermore, there were no significant interactions between psychological resources and any of the abuse types (severe physical abuse: HR = 1.12, 95% CI = 0.89–1.40, $P = 0.327$; moderate physical abuse: HR = 1.07, 95% CI = 0.90–1.23, $P = 0.431$; emotional abuse: HR = 0.97, 95% CI = 0.82–1.15, $P = 0.731$).

Table 2 | Results of models predicting mortality risk from sociodemographic characteristics, medical conditions and health behaviours

	Model 1		Model 2		Model 3	
	HR (95% CI)	*P*	HR (95% CI)	*P*	HR (95% CI)	*P*
Age	2.81 (2.26–3.50)	<0.001	2.38 (1.90–2.97)	<0.001	2.38 (1.90–2.99)	<0.001
Age × time	1.09 (1.05–1.27)	0.065	1.15 (1.04–1.26)	0.004	1.16 (1.05–1.27)	0.003
Female[a]	0.69 (0.61–0.78)	<0.001	0.71 (0.62–0.80)	<0.001	0.81 (0.71–0.92)	0.002
African American[b]	1.47 (1.13–1.91)	0.004	1.63 (1.25–2.12)	<0.001	1.74 (1.34–2.26)	<0.001
Other[b]	0.99 (0.67–1.48)	0.965	0.92 (0.62–1.37)	0.68	0.95 (0.63–1.41)	0.783
Education	0.81 (0.76–0.86)	<0.001	0.82 (0.77–0.87)	<0.001	0.84 (0.79–0.90)	<0.001
Heart disease			1.96 (1.71–2.25)	<0.001	1.90 (1.65–2.18)	<0.001
Cancer			2.97 (1.89–4.67)	<0.001	2.90 (1.85–4.55)	<0.001
Cancer × time			0.68 (0.56–0.84)	<0.001	0.68 (0.56–0.84)	<0.001
Depression			1.34 (1.10–1.63)	0.003	1.26 (1.04–1.53)	0.021
Smoking					1.73 (1.51–1.98)	<0.001
Alcohol use					1.06 (0.92–1.21)	0.427

Gender was coded as 0 = male and 1 = female. [b]Race was dummy-coded with European Americans as the reference group.

We also examined each psychological resource variable individually, and similarly found little evidence that social support acted as a proxy for each psychological resource variable. When adjusting for positive affect, sense of control and purpose in life individually, the interaction effects between childhood abuse and social support remained, although several were slightly attenuated (Supplementary Tables 1–3). Similarly, interactions between childhood abuse types and sense of control and positive affect were not significant (Supplementary Tables 4 and 5). For purpose in life, there was no significant interaction with emotional abuse; however, significant interactions with moderate and severe physical abuse emerged (Supplementary Table 6). Notably, both the physical abuse by social support interactions and the physical abuse by purpose in life interactions remained significant when they were entered into the same model (Supplementary Table 7), suggesting independent moderating effects of social support and purpose in life. Indeed, unlike social support, the association between purpose in life and reduced mortality risk was not evident among those reporting severe physical abuse (HR = 1.06, 95% CI = 0.89–1.26, $P = 0.499$) and moderate physical abuse (HR = 1.02, 95% CI = 0.90–1.15, $P = 0.739$). Rather, this association was observed only among those reporting no physical abuse (severe: HR = 0.87, 95% CI = 0.81–0.92, $P < 0.001$; moderate: HR = 0.83, 95% CI = 0.78–0.90, $P < 0.001$).

We previously reported gender differences in the association between child abuse and premature mortality[7], with effects specific to women. Accordingly, we estimated follow-up models that included a three-way interaction between gender, self-reported childhood abuse types and self-reported social support. In models focusing on severe and moderate physical abuse, the interaction between childhood abuse and social support remained significant (severe physical abuse: HR = 0.76, 95% CI = 0.62–0.93, $P = 0.008$; moderate physical abuse: HR = 0.83, 95% CI = 0.71–0.98; $P = 0.030$), but there was no moderation of this effect by gender (severe physical abuse: HR = 1.13, 95% CI = 0.84–1.52, $P = 0.402$; moderate physical abuse: HR = 1.13, 95% CI = 0.88–1.44; $P = 0.333$). For emotional abuse, neither the two-way child abuse by support interaction (HR = 0.89, 95% CI = 0.76–1.05, $P = 0.173$) nor the three-way abuse by support by gender interaction (HR = 0.97, 95% CI = 0.77–1.23, $P = 0.799$) was significant.

Lastly, we examined whether the strength of the moderating effect of abuse varied by abuse type. To do so, we entered all three abuse by social support interactions into the same model. The results should be interpreted with some caution because of the fairly strong associations amongst types of abuse ($rs = 0.42$–0.56, $Ps < 0.001$). When all the terms were entered into a single covariate adjusted model, moderate physical abuse and emotional abuse no longer interacted with social support (moderate physical abuse: HR = 1.01, 95% CI = 0.86–1.20, $P = 0.864$; emotional abuse: HR = 0.93, 95% CI = 0.80–1.07; $P = 0.299$). There was a marginally significant interaction suggesting that severe physical abuse continued to moderate the link between support and mortality risk (HR = 0.84, 95% CI = 0.70–1.00, $P = 0.056$). Follow-up probing of this interaction indicated that higher social support was associated with lower mortality among both those with and without a history of severe physical abuse. However, this association was stronger among those reporting severe physical abuse (HR = 0.74, 95% CI = 0.64–0.85, $P < 0.001$) compared with those reporting minimal severe physical abuse (HR = 0.91, 95% CI = 0.85–0.98, $P = 0.008$).

The purpose of the current study was to determine whether reports of current social support in adulthood could offset mortality risk associated with reports of childhood experiences of parental abuse. In a national study of midlife adults, we observed interactions indicating that among those reporting childhood experiences of abuse, higher self-reported social support was related to lower mortality risks across nearly two decades. More specifically, a one standard deviation increase in social support was associated with a 26%, 19% and 20% decrease in mortality risk among individuals reporting childhood experiences of severe physical abuse, moderate physical abuse and emotional abuse, respectively. This effect was similar to those of some of the more traditional mortality risk factors. For instance, a one standard deviation increase in educational attainment was associated with a 16% decrease in mortality risk, and female gender was associated with a 19% reduction in mortality risk (although caution should be taken when comparing dichotomous and continuous predictors). By contrast, social support was associated with a more modest (7–8%) reduction of mortality risk among those without exposure to physical and emotional abuse. This pattern of findings is consistent with the buffering hypothesis, which posits that social support has beneficial effects only or primarily in the context of stress[26]. Notably, the buffering effects of social support were above those of traditional risk factors associated with both abuse and mortality, including educational attainment, history of heart problems and cancer, depression and health behaviours.

The observed findings converge with a substantial body of work demonstrating the salubrious effects of supportive relationships in

Table 3 | Results of models predicting mortality risk from childhood abuse, social support and their interactions

	Model 4		Model 5		Model 6	
	HR (95% CI)	*P*	HR (95% CI)	*P*	HR (95% CI)	*P*
	Severe physical abuse					
Age	2.45 (1.95–3.08)	<0.001	2.47 (1.96–3.10)	<0.001	2.47 (1.96–3.11)	<0.001
Age × time	1.15 (1.04–1.27)	0.006	1.15 (1.04–1.27)	0.005	1.15 (1.04–1.27)	0.005
Female[a]	0.81 (0.71–0.93)	0.002	0.82 (0.72–0.94)	0.004	0.82 (0.72–0.94)	0.004
African American[b]	1.70 (1.31–2.22)	<0.001	1.69 (1.30–2.20)	<0.001	1.71 (1.32–2.23)	<0.001
Other[b]	0.93 (0.62–1.37)	0.701	0.91 (0.61–1.35)	0.628	0.90 (0.60–1.33)	0.589
Education	0.85 (0.80–0.91)	<0.001	0.85 (0.80–0.91)	<0.001	0.85 (0.80–0.91)	<0.001
Heart disease	1.90 (1.65–2.18)	<0.001	1.90 (1.65–2.18)	<0.001	1.90 (1.66–2.18)	<0.001
Cancer	2.88 (1.83–4.54)	<0.001	2.92 (1.85–4.59)	<0.001	2.95 (1.87–4.64)	<0.001
Cancer × time	0.68 (0.55–0.83)	<0.001	0.68 (0.55–0.84)	<0.001	0.68 (0.55–0.83)	<0.001
Depression	1.25 (1.02–1.52)	0.028	1.19 (0.97–1.45)	0.091	1.18 (0.97–1.45)	0.100
Smoking	1.74 (1.52–2.00)	<0.001	1.75 (1.52–2.00)	<0.001	1.75 (1.52–2.00)	<0.001
Alcohol use	1.07 (0.93–1.22)	0.352	1.03 (0.90–1.18)	0.622	1.04 (0.91–1.17)	0.591
Severe physical abuse	1.16 (0.96–1.39)	0.117	1.11 (0.92–1.34)	0.262	1.02 (0.83–1.24)	0.885
Social support			0.88 (0.83–0.94)	<0.001	0.92 (0.86–0.98)	0.012
Severe physical abuse × social support					0.81 (0.70–0.94)	0.005
	Moderate physical abuse					
Age	2.39 (1.91–3.00)	<0.001	2.41 (1.92–3.02)	<0.001	2.41 (1.92–3.02)	<0.001
Age × Time	1.16 (1.05–1.28)	0.003	1.16 (1.05–1.28)	0.002	1.16 (1.05–1.28)	0.002
Female[a]	0.81 (0.71–0.92)	0.001	0.81 (0.71–0.93)	0.002	0.81 (0.71–0.93)	0.002
African American[b]	1.69 (1.29–2.20)	<0.001	1.68 (1.29–2.18)	<0.001	1.69 (1.29–2.20)	<0.001
Other[b]	0.97 (0.66–1.44)	0.891	0.95 (0.64–1.40)	0.782	0.94 (0.63–1.39)	0.746
Education	0.85 (0.80–0.90)	<0.001	0.85 (0.80–0.90)	<0.001	0.85 (0.80–0.90)	<0.001
Heart disease	1.89 (1.65–2.17)	<0.001	1.89 (1.64–2.17)	<0.001	1.89 (1.65–2.17)	<0.001
Cancer	2.86 (1.82–4.50)	<0.001	2.91 (1.85–4.57)	<0.001	2.93 (1.87–4.61)	<0.001
Cancer × time	0.68 (0.56–0.84)	<0.001	0.68 (0.56, -0.84)	<0.001	0.68 (0.56–0.83)	<0.001
Depression	1.25 (1.02–1.52)	0.031	1.18 (0.97–1.45)	0.102	1.18 (0.97–1.45)	0.100
Smoking	1.74 (1.51–1.99)	<0.001	1.74 (1.52–2.00)	<0.001	1.74 (1.51–1.99)	<0.001
Alcohol use	1.06 (0.93–1.22)	0.367	1.03 (0.90–1.18)	0.647	1.04 (0.91–1.19)	0.587
Moderate physical abuse	0.99 (0.86–1.15)	0.943	0.97 (0.85–1.12)	0.727	0.95 (0.83–1.04)	0.528
Social support			0.88 (0.83–0.94)	<0.001	0.92 (0.85–0.99)	0.025
Moderate physical abuse × social support					0.88 (0.78–1.00)	0.045
	Emotional abuse					
Age	2.38 (1.90–2.98)	<0.001	2.39 (1.91–3.00)	<0.001	2.40 (1.91–3.00)	<0.001
Age × time	1.16 (1.05–1.27)	0.003	1.16 (1.05–1.28)	0.003	1.16 (1.05–1.28)	0.003
Female[a]	0.81 (0.71–0.92)	0.002	0.82 (0.72–0.93)	0.003	0.82 (0.72–0.94)	0.004
African American[b]	1.74 (1.34–2.26)	<0.001	1.74 (1.34–2.26)	<0.001	1.75 (1.34–2.27)	<0.001
Other[b]	0.95 (0.64–1.42)	0.803	0.92 (0.62–1.37)	0.690	0.92 (0.62–1.37)	0.680
Education	0.84 (0.79–0.90)	<0.001	0.84 (0.79–0.90)	<0.001	0.84 (0.79–0.90)	<0.001
Heart disease	1.90 (1.65–2.18)	<0.001	1.90 (1.65–2.18)	<0.001	1.90 (1.65–2.18)	<0.001
Cancer	2.91 (1.85–4.56)	<0.001	2.95 (1.88–4.62)	<0.001	2.94 (1.88–4.61)	<0.001
Cancer × time	0.68 (0.56–0.84)	<0.001	0.68 (0.56–0.84)	<0.001	0.68 (0.56–0.84)	<0.001
Depression	1.27 (1.04–1.54)	0.019	1.20 (0.98–1.47)	0.070	1.19 (0.98–1.46)	0.084
Smoking	1.73 (1.51–1.98)	<0.001	1.73 (1.51–1.99)	<0.001	1.74 (1.51–1.99)	<0.001
Alcohol use	1.06 (0.92–1.21)	0.418	1.02 (0.89–1.17)	0.734	1.03 (0.90–1.18)	0.641
Emotional abuse	0.97 (0.85–1.12)	0.710	0.95 (0.83–1.09)	0.470	2.26 (0.96–5.33)	0.340
Social support			0.88 (0.83–0.93)	<0.001	0.92 (0.85–0.99)	0.027
Emotional abuse × social support					0.89 (0.79–1.00)	0.046

[a]Gender was coded as 0 = male and 1 = female. [b]Race was dummy-coded with European Americans as the reference group.

the face of early adversity on psychosocial, behavioural and biological functioning[10,11,13,15,27,28]. However, many of these studies focus on positive relationships in relatively close proximity to the adversity. This approach explicitly assumes that buffering processes occur alongside the adversity exposure or shortly thereafter. The present study builds on these previous studies by focusing on self-reported social support at later stages in life, decades after childhood experiences of adversity presumably occurred, and by extending the buffering effects to a clinically important outcome, namely mortality. Our results suggest the possibility that resources much later in life can serve a buffering function long after the stressor has ended. If substantiated in future research, this observation suggests that strengthening social relationships for middle-aged adults could help offset risks associated with adversities occurring much earlier.

The present findings also converge with developmental theories of resilience that conceptualize resilience as a process that includes recovery or restoration, which can take considerable time to manifest after adversity exposure[29]. Importantly, changes in the individual and in their context and experiences (including relationships with others) can alter pathways to resilience, trajectories of risk associated with a particular adversity, and capacity to adapt to subsequent challenges or threats[29]. These views of resilience as a dynamic process provide a framework for understanding how social support in adulthood could buffer against mortality risk among those with a history of childhood abuse. Indeed, supportive relationships in adulthood could help abuse victims overcome the multitude of developmental sequelae associated with their childhood experiences. Research shows these sequelae unfold across the lifespan and can include lower educational attainment, difficulties with employment and smaller earnings, as well as higher incidence of psychiatric conditions including major depression, anxiety disorders and substance abuse[30,31]. These demographic and psychiatric sequelae are themselves associated with alterations in biological processes, engagement in unhealthy lifestyle behaviours and poor health outcomes, including premature mortality[32,33]. In addition to increasing exposure to threatening conditions, early abuse may also increase sensitivity to them[34,35]. For instance, childhood abuse has been associated with higher negative emotional reactivity to everyday stress[36], which, in turn, has been shown to increase risk for premature mortality[37]. With respect to biological sensitivity, there is evidence suggesting that early adversity may bias certain immune cells (monocytes and macrophages) towards a pro-inflammatory state, such that when they encounter subsequent threats, they mount exaggerated inflammatory responses and are less sensitive to anti-inflammatory signals, which ultimately fosters a state of low-grade inflammation and increases risk for diseases such as atherosclerosis[38]. Given the available evidence[39–42], it seems likely that supportive adult relationships mitigate the health impact of these sequelae across the middle and later stages of the lifespan. Future research should test this empirically and identify the behavioural (smoking, weight gain and exercise) and biological (autonomic, cardiovascular and immunologic) processes through which this mitigation occurs.

The present study is not without limitations. First, causal inferences cannot be made given the observational design. Although we had a truly prospective design, adjusted for relevant confounds and considered alternative explanations regarding psychological resources, these features do not entirely ameliorate interpretational challenges. The pattern we interpret as buffering by social support could simply reflect a group of especially hardy or resilient individuals, who because of other (unmeasured) factors have close adult relationships and lower mortality rates. It is possible, then, that social support reflects a broader set of other unmeasured protective influences and resources that collectively may be counteracting the mortality risk associated with early abuse. With that said, we considered multiple psychological resources, including purpose in life, control and mastery, and positive emotion, that in past research have been associated with lower mortality. There was no evidence to suggest these resources were responsible for social support's association with mortality risks. Moreover, animal studies that manipulate both early adversity and housing conditions in later phases of life show that the detrimental effects of early adversity can be reversed by environments enriched with more opportunities for social interactions and play in later stages of life[19,20]. These findings speak to the plausibility of our interpretation, though of course they do not by themselves prove it. Second, assessment of childhood abuse was based on retrospective self-reports. It is probable that some participants misreported their experiences of childhood abuse given concerns of social desirability and the fallibility of memory[43,44]. However, as long as it is random, misreporting is likely to bias results towards the null hypothesis. Moreover, evidence suggests that retrospective reports about the occurrence of major childhood traumas are generally accurate, even if details about their timing and nature are not[44]. Third, we were unable to test the biological pathways proposed above. Biological measures, including markers of inflammation, were assessed in a sub-project of MIDUS II nearly a decade after MIDUS I. However, these measures were obtained from only a small fraction ($n = 1{,}018$) of the respondents in our analyses, just 67 of whom (6.6%) have expired. When abuse exposure is considered, we end up with cell sizes much too small for valid survival analyses (for example, there is a total of 8 individuals in the MIDUS sample who endorsed severe physical abuse, had biological measures taken, and have expired). Testing biological mechanisms through which social support may exert its protective effects against mortality risk among those reporting childhood abuse will become more feasible as mortality increases in MIDUS. Lastly, timing of abuse was not assessed in MIDUS, but may have differential effects on biological processes thought to contribute to morbidity and mortality[21]. As such, it remains unknown whether social support in adulthood can dampen risk for mortality regardless of when abuse occurred in childhood and whether it operates through similar pathways. Relatedly, our analyses focused on a single outcome, overall mortality, and it thus remains unclear against which disease(s) social support can mitigate in the context of abuse.

Childhood abuse increases risk for a variety of adverse health outcomes across the lifecourse, including premature mortality[2,7]. Despite this general trend, there is a great deal of variability in the sequelae of abuse, which suggests the presence of intervening factors and processes that mitigate risks. The results of the current study highlight self-reported social support in adulthood as a protective factor that buffers against the excess mortality associated with childhood abuse. These findings suggest the possibility that adult social support could be used as leverage for interventions seeking to mitigate the adverse health consequences of childhood abuse, even though the exposure itself may have occurred many decades previously. Indeed, there is preliminary evidence to suggest that strengthening family relationships can offset some of health consequences of childhood socioeconomic disadvantage[45].

Methods

Participants and procedures. Data for the current analysis were drawn from the first wave of MIDUS. A sample of 7,108 non-institutionalized, English-speaking adults of ages 25 to 74 were recruited from a nationally representative, random-digit dialling sample in 1995–1996. Participants completed telephone interviews and mail-in self-administered questionnaires that included assessments of childhood abuse and current social support. Mortality data were then obtained through October 2015. Institutional review boards at the University of Wisconsin and Harvard Medical School approved all study procedures, and informed consent was obtained from all participants by telephone. The majority ($n = 6{,}325$; 89%) of the 7,108 participants in the first wave of MIDUS completed both the phone interview and the self-administered questionnaires. Of these, almost all ($n = 6{,}216$; 98%) completed measures of social support and of at least one category of childhood abuse. An additional 2.2% ($n = 138$) had missing data on demographic information and other covariates, leaving a final analytic sample ranging from 6,071 to 6,078. Power analyses indicated that these were appropriate sample sizes

for the present study. Given the proportion of participants who expired over the follow-up period (17.1%), we estimated that a sample of 5,346 was necessary to detect a moderate-sized interaction between social support and childhood abuse (that is, a hazard ratio of 0.80), with statistical power of 0.80.

Measures. *Early abuse.* Questions on the childhood abuse scale used in MIDUS I were drawn from the revised Conflict Tactics Scale[46] and probed three categories of childhood abuse: severe physical abuse, moderate physical abuse and emotional abuse. Each category was assessed with a single item on a 4-point scale (1 = often, 4 = never/does not apply). For severe physical abuse, participants indicated whether someone "kicked, bit, or hit you with a fist or tried to hit you with something/beat you up/choked you/burned or scalded you". Moderate physical abuse included whether someone "pushed, grabbed or shoved you/slapped you/threw something at you", and emotional abuse included whether someone "insulted or swore at you/sulked or refused to talk to you/stomped out of the room/did or said something to spite you/threatened to hit you/smashed or kicked something in anger". Items were reverse coded such that higher scores reflected more frequent abuse.

Each item was asked separately for mother, father, brothers, sisters and anybody else. However, in line with previous research, we focused on abuse from participants' mother and father given that the most common perpetrators of childhood abuse are parents and that abuse by parents may be the most egregious violation of trust[47,48]. As such, abuse scores were based on responses to a total of six probes (emotional, moderate physical and severe physical, for both mother and father). As in our previous analyses, abuse was coded as present if it happened frequently—that is, when participants endorsed one of the items as happening at least some of the time[7].

Social support. MIDUS used 12 items from previous research to assess social support from family (excluding spouse or partner), friends and spouse or partner[49,50]. Four items were asked for each source, and included "how much do they care about you", "how much do they understand the way you feel about things", "how much can you rely on them for help if you have a serious problem" and "how much can you open up to them if you need to talk about your worries". Participants responded to each item on a 4-point scale (1 = a lot, 4 = not at all). Responses were reverse coded, such that higher scores indicated greater levels of support, and averaged within each source of support. The scales had strong internal consistency in the present sample, as indicated by Cronbach's α (family $\alpha = 0.85$; friends $\alpha = 0.89$; spouse/partner $\alpha = 0.87$). We focused on overall support, and therefore averaged values across sources (overall $\alpha = 0.90$).

Mortality. Data on mortality were collected using several methods, including National Death Index reports, tracing that included mortality closeout interviews, and longitudinal sample maintenance, until October 2015. Survival times for decedents reflected the number of years between the date when MIDUS I self-administered questionnaires were returned and the date of death. Due to confidentiality purposes, only month and year of death were documented; consequently, the day for all deaths was set to the 15th day of each month. Survival times for participants who were still living reflected the length of follow-up censored at 31 October 2015.

Covariates. Statistical models included a panel of covariates that are known contributors to premature mortality, and could plausibly confound its association with abuse or support. These variables included sociodemographic characteristics (age, gender, race/ethnicity and education level), health behaviours (smoking and alcohol consumption), and major medical conditions (history of heart disease, cancer and depression). Participants reported their gender (0 = male, 1 = female), their date of birth from which age was computed, the highest level of education completed (coded as less than a high school diploma, high school degree, some college, college degree or some graduate school, or master's or professional degree) and their race, which was dummy coded into variables reflecting African American or other with European Americans as the reference group. Single items with binary responses (yes/no) assessed whether participants ever smoked cigarettes regularly (at least a few cigarettes every day), ever consumed at least one alcoholic drink three or more days a week, ever had heart trouble (heart attack, coronary artery disease, heart failure, valve disease, hole in heart, angina, hypertension, arrhythmia, heart murmur, or other) suspected or confirmed by a doctor, or ever had cancer (breast, cervical, colon, lung, lymphoma/leukaemia, ovarian, prostate, skin, uterine or other). Lastly, participants completed questions assessing depressed mood, anhedonia and related symptoms in the previous 12 months from the World Health Organization's Composite International Diagnostic Interview[51]. Based on criteria specified in the third edition of the American Psychiatric Association Diagnostic and Statistical Manual of Mental Disorders (DSM-III-R)[52], depression was coded as being present or absent. Criteria for major depression included having depressed mood or anhedonia most of the day, almost every day, and at least four other symptoms, including loss of interest, energy or appetite, trouble sleeping or concentrating, and having feelings of low self-worth or suicidal thoughts, for a period of at least two weeks[53].

Alternative explanations. To determine whether social support simply reflected other protective factors associated with morbidity and mortality, we created a psychological resources composite that included measures of positive affect, sense of control over one's life, and purpose in life. For positive affect, participants indicated on a 5-point scale (1 = none of the time, 5 = all of the time) how much of the time during the past 30 days they felt cheerful, in good spirits, extremely happy, calm and peaceful, satisfied, and full of life ($\alpha = 0.91$). Sense of control was assessed along two dimensions: personal mastery (one's sense of efficacy in pursuing and achieving goals) and perceived constraints (beliefs that obstacles are beyond one's control). Mastery was assessed with four items from the Pearlin Mastery Scale[54] and perceived constraints were assessed with 8 additional items. Participants responded on a 7-point scale (1 = strongly agree, 7 = strongly disagree). Example items include "what happens to me in the future mostly depends on me" and "what happens in my life is often beyond my control". Positively worded items were reversed coded and responses across items were averaged ($\alpha = 0.85$). Lastly, purpose in life was assessed with three items from the Ryff Scales of Psychological Well-Being[55]. Using a 7-point scale (1 = strongly disagree, 7 = strongly agree) participants indicated the extent to which they agreed with the following: "some people wander aimless through life, but I am not one of them"; "I live life one day at a time and don't really think about the future"; and "I sometimes feel as if I've done all there is to do in life" ($\alpha = 0.35$). A principal components analysis of the measures in the resources composite yielded a single component that explained 57.5% of the variance, with loadings ranging from 0.52 to 0.65.

Analytic approach. A series of Cox proportional hazard models with standardized continuous variables were estimated using Stata 14. We first determined whether the proportional hazards assumption of Cox models was violated by formally testing non-zero slopes between time and Schoenfeld residuals of predictor and covariate variables[56]. The proportionality assumption was not upheld for age ($P = 0.001$) and cancer ($P < 0.001$). As such, age by time and cancer by time interactions were included as time-varying covariates in all models. Next, we conducted primary analyses. Demographic characteristics (age, age × time, gender, race and educational attainment) were entered in the first step, medical history (heart problems, cancer, cancer × time, and depression) in the second step, and health behaviours (smoking and alcohol use) in the third step. Main effects of self-reported childhood abuse and self-reported social support were entered in the fourth and fifth steps, respectively. Lastly, a product term reflecting the interaction between participant-reported childhood abuse and social support was added in the final step. To facilitate interpretation of significant interaction effects, we stratified the sample according to self-reported presence of childhood abuse and estimated the link between self-reported social support and mortality risk.

Less than 3% of data were missing for each of the variables included in analyses. Because estimates are not likely to be biased when missing data occurs at a rate less than 10%[57], imputation was deemed unnecessary. In performing analyses for the present study, we have complied with all relevant ethical principles.

Life Sciences Reporting Summary. Further information on experimental design is available in the Life Sciences Reporting Summary.

Code availability. Computer code supporting the present study's findings are available from the corresponding author upon reasonable request.

Data availability. The data on which the present study is based are publicly available online from the Inter-university Consortium for Political and Social Research at http://www.icpsr.umich.edu/icpsrweb/ICPSR/series/203.

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Author contributions

J.J.C. and G.E.M. developed the study concept. J.J.C. conducted data analyses and interpretation under the supervision of G.E.M. J.J.C. and G.E.M. drafted the manuscript, and E.C. provided critical revisions. All authors approved the final version of the manuscript.

Competing interests

The authors declare no competing interests.

Additional information

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Experimental design

1. Sample size

Describe how sample size was determined.

Sample size calculations prior to data collection were not performed, as the current study was based on secondary data analyses. However, as reported in the manuscript, post-hoc power analyses conducted for the current analysis confirmed that the sample size was sufficient.

2. Data exclusions

Describe any data exclusions.

Individuals with missing data on any of the variables of interest were excluded from analyses, as reported in the manuscript.

3. Replication

Describe whether the experimental findings were reliably reproduced.

The current study is an observational study and replication with another sample was not attempted.

4. Randomization

Describe how samples/organisms/participants were allocated into experimental groups.

Because the current study did not employ an experimental design, randomization did not apply.

5. Blinding

Describe whether the investigators were blinded to group allocation during data collection and/or analysis.

Because the current study did not employ an experimental design, investigators were not blind to group allocation.

Note: all studies involving animals and/or human research participants must disclose whether blinding and randomization were used.

6. Statistical parameters

For all figures and tables that use statistical methods, confirm that the following items are present in relevant figure legends (or in the Methods section if additional space is needed).

n/a	Confirmed
☐	☒ The exact sample size (*n*) for each experimental group/condition, given as a discrete number and unit of measurement (animals, litters, cultures, etc.)
☐	☒ A description of how samples were collected, noting whether measurements were taken from distinct samples or whether the same sample was measured repeatedly
☒	☐ A statement indicating how many times each experiment was replicated
☐	☒ The statistical test(s) used and whether they are one- or two-sided (note: only common tests should be described solely by name; more complex techniques should be described in the Methods section)
☐	☒ A description of any assumptions or corrections, such as an adjustment for multiple comparisons
☐	☒ The test results (e.g. *P* values) given as exact values whenever possible and with confidence intervals noted
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See the web collection on statistics for biologists for further resources and guidance.

Software

Policy information about availability of computer code

7. Software

Describe the software used to analyze the data in this study.	Stata 14 was used to perform all data analyses.

For manuscripts utilizing custom algorithms or software that are central to the paper but not yet described in the published literature, software must be made available to editors and reviewers upon request. We strongly encourage code deposition in a community repository (e.g. GitHub). *Nature Methods* guidance for providing algorithms and software for publication provides further information on this topic.

Materials and reagents

Policy information about availability of materials

8. Materials availability

Indicate whether there are restrictions on availability of unique materials or if these materials are only available for distribution by a for-profit company.	No unique materials were used.

9. Antibodies

Describe the antibodies used and how they were validated for use in the system under study (i.e. assay and species).	No antibodies were used.

10. Eukaryotic cell lines

a. State the source of each eukaryotic cell line used.	No eukaryotic cell lines were used.
b. Describe the method of cell line authentication used.	No eukaryotic cell lines were used.
c. Report whether the cell lines were tested for mycoplasma contamination.	No eukaryotic cell lines were used.
d. If any of the cell lines used are listed in the database of commonly misidentified cell lines maintained by ICLAC, provide a scientific rationale for their use.	No misidentified cell lines were used.

Animals and human research participants

Policy information about studies involving animals; when reporting animal research, follow the ARRIVE guidelines

11. Description of research animals

Provide details on animals and/or animal-derived materials used in the study.	No animals were used.

Policy information about studies involving human research participants

12. Description of human research participants

Describe the covariate-relevant population characteristics of the human research participants.	The average age of participants was 46.78 (SD = 12.90). Approximately half (52.5%) of participants were female. The vast majority were of European descent (90.8%) and reported obtaining a high school or higher degree (90.2%). Nearly a third (32.6%) reported a history of heart disease or cancer, and/or depression in the past year, and many had regularly smoked (51.2%) or consumed alcohol (41.6%) in their lifetime.