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Bridget J. Goosby

University of Nebraska-Lincoln, bgoosby2@unl.edu

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Early Life Course Pathways of Adult Depression and Chronic Pain

Bridget J. Goosby¹

¹University of Nebraska–Lincoln, Lincoln, NE, USA

Abstract

Applying cumulative inequality theory, this study examines the extent to which childhood socioeconomic disadvantage and maternal depression increase the risk of major depression and chronic pain in U.S. working-aged adults. Further, I assess whether low socioeconomic status amplifies the risk of adult depression and/or pain. Using data from the 2003 National Comorbidity Survey Replication ($N=4339$), I find that socioeconomic disadvantage and maternal depression during youth increases the risk of adult depression and/or chronic pain. The probability of having chronic pain increases in magnitude over the life course for adults whose parents have lower educational attainment relative to those with more highly educated parents. Childhood socioeconomic circumstances are not completely explained by adulthood socioeconomic status indicators. These findings help illustrate the far-reaching influence of childhood context on adult physical and mental health.

Keywords

cumulative inequality; depression; intergenerational; life course; pain

Chronic pain and depression are two common health conditions experienced by adults in the United States. Over the lifetime, approximately 44 percent of U.S. adults experience chronic pain, and about 16 percent report one or more major depressive episodes, a condition with peak prevalence between ages 30 and 44 (Kessler et al. 2005). Moreover, major depression is associated with chronic pain risk (Gureje et al. 2008). On average, approximately 65 percent of patients with depression in clinical studies report pain, and when depression is present, they report pain of greater intensity (Bair et al. 2003). Growing clinical and gerontological literatures suggest that depression is strongly associated with chronic pain in aging adult populations (see Bierman 2011), which is particularly important because both depression and pain are tied to elevated levels of functional impairment (Gayman, Turner, and Cui 2008). With such high rates of these conditions, examining the role of early life course inequalities for the progression of depression and pain during the working ages is crucial.

Early childhood social conditions such as poverty and poor parental health leave offspring vulnerable to poor health conditions in later life (Hatch 2005; Power and Matthews 1997). In the case of chronic pain and depression, however, much of the prior sociological literature examining the social determinants of these conditions in conjunction with each other is limited to older populations or does not account for the presence of disadvantage earlier in

the life course. Identifying childhood social risks that lead to depression and pain in working-age adults will ultimately shed light on the pathways through which early life social circumstances lead to socioeconomic gradations in health during middle age. Indeed, there is a growing literature illustrating the implications of early poverty exposure for adult health and childhood exposure to maternal depression for subsequent adult depression levels. But whether childhood exposure to poverty and maternal depression share the same early social etiological pathways is yet to be understood.

Cumulative inequality (CI) theory, which posits that early exposure to stressful contexts predict adulthood disadvantage and amplifies differences as people age (Ferraro and Shippee 2009), offers a promising avenue for understanding the mechanisms of early life course risk associated with socioeconomic disparities in health. Accordingly, this study traces the links between early life course disadvantage and the risk of depression and chronic pain in middle age U.S. adults, either individually or jointly. Using data from the 2003 National Comorbidity Survey Replication (NCS-R), I identify salient childhood pathways through which adult health conditions emerge in middle age.

BACKGROUND

CI and Health

CI theory (Ferraro and Shippee 2009) combines concepts from the cumulative adversity/disadvantage (CAD; Dannefer 1987) and life course perspectives on aging (Ferraro 2007). CAD theory argues that socioeconomic gradations in health can be linked back to early life social status factors such as childhood poverty, gender, and race-ethnicity (Hatch 2005). For example, childhood socioeconomic status (SES) shapes access to resources and exposure to health risks (e.g., health care access, safe neighborhoods, quality health care), consequently leading to differential health outcomes over the life course. CI theory builds upon CAD theory, posited by Dannefer (1987) and O’Rand (2003), by explicitly integrating the intergenerational transmission of inequalities into life course aging and health processes.

CI theory is a “mid-range theory” that incorporates micro- and macro-sociological concepts to help connect prior empirical generalizations into a testable framework (Ferraro, Shippee, and Schafer 2009). Specifically, the CI axioms emphasize the importance of the intergenerational transmission of inequality as a mechanism through which social and biological processes in life course aging are differentiated by exposure to inequality. In this study, I test the following CI axioms, which are discussed in more detail below and depicted in Figure 1: (1a) childhood conditions are important for adult health trajectories, (1b) differentiation in health conditions may be shaped by intergenerational transmission, (2a) the consequences of disadvantage can be amplified over the life course, and (2b) inequality may diffuse across life course domains.

Axiom 1a: Childhood conditions and adult health—The first axiom states that childhood conditions are salient for adult health trajectories. With some evidence indicating that depression and pain share the same physiologic origins (Bair et al. 2003; Gureje et al. 2008), I hypothesize that *the presence of childhood socioeconomic disadvantage will increase the risk of adulthood pain and/or depression*. Depression and pain are depicted on the right side of Figure 1 and included together to indicate their interconnection with one another but are in separate inset boxes to show that they will be tested as separate and co-occurring dependent variables. The hypothesized (H1) pathway linking childhood SES to depression and pain is also shown. Prior findings suggest that early life circumstances such as childhood poverty play an important role in predicting health and economic viability in adulthood (Hamil-Luker and O’Rand 2007; Wagmiller, Lennon, and Kuang 2008). Adult chronic pain and depression are both conditions with markedly higher prevalence rates

among socioeconomically disadvantaged populations (Miech and Shanahan 2000; Poleshuck and Green 2008).

Stressful conditions beginning in early life may be contributors to the depression/pain relationship. Specifically, though occasional arousal of the body's physiologic response to stress is normal, chronic activation can disrupt neuroendocrine and immune function (e.g., via the hypothalamic-pituitary-adrenal, or HPA, axis; Ferraro and Shippee 2009). Clinical studies suggest that the interaction of these processes leave people susceptible to elevated responses to both painful stimuli (e.g., hyperalgesia) and depression (Blackburn-Munro 2004). Such studies report that chronic pain with depression is more likely to consist of severe headaches including migraines (Zwart et al. 2003), back and neck pain (Demyttenaere et al. 2007), arthritis (Kiecolt-Glaser and Glaser 2002), and other general complaints of pain (Bair et al. 2003). In the case of depression and pain, the stress of childhood hardship may play out physiologically through overactivity of the HPA axis in children and can accumulate over the life course, resulting in elevated susceptibility to mental and chronic physical health problems later in life (Repetti, Tay-lor, and Seeman 2002).

Much of the literature examining the social correlates shared by pain and depression, however, focus on contemporaneous adult SES, without accounting for childhood conditions. One major exception is studies examining the consequences of childhood abuse for the risk of adulthood pain, which demonstrate that child abuse and neglect elevate the risk of chronic pain in adulthood (Kopec and Sayre 2005). Such findings provide an important example illustrating the consequences of extreme cases of stress in the household. More generally, youth in chronically stressful environments due to economic hardship are at risk for a cascade of stressful circumstances (e.g., food insecurity, neighborhood violence, family conflict), which also elevate vulnerability to poor health in later life (Repetti et al. 2002). Despite the shortage of empirical literature, there is evidence that early life conditions can have important implications for the onset of depression and pain in adulthood.

Axiom 1b: Intergenerational transmission—Parent health is an important predictor of adult health (Power and Matthews 1997; Weissman et al. 2006) and a key pathway through which childhood disadvantage influences offspring health outcomes and life chances (Ensminger et al. 2003; Goosby 2007). I hypothesize, as illustrated in pathway H2 of Figure 1, that *maternal depression increases the risk of major depression and/or chronic pain in adulthood*. According to CI theory (Axiom 1b), family lineage in the form of both genetic transmission and shared environment plays a critical role in long-term offspring health outcomes (Ferraro and Shippee 2009). Behavioral genetic studies suggest that up to one half of the maternal and offspring depression relationship may be explained by genetic heritability (Kendler 1995; Rende et al. 1993). Children, however, are nested within families that share both home environments and genetic linkages, thereby exacerbating either the risk for poor mental and physical health outcomes or increased opportunities for better health (Freese and Shostak 2009).

Maternal mental health is also a key stressor in childhood associated with adult offspring well-being. Mothers in low-income households are especially vulnerable to psychological distress and disorders such as depression (Gilman et al. 2002; Kahn et al. 2000). Depression can impede a parent's ability to effectively nurture and supervise his or her offspring (Ensminger et al. 2003). Furthermore, mothers with depression are more likely to engage in harsh and inconsistent punishment of their children while exhibiting emotional withdrawal and pessimism about their children's future (Downey and Coyne 1990; Elder and Shanahan 2007). Consequently, maternal depression elevates the risk that offspring will experience developmental challenges coupled with declines in health (e.g., externalizing and

internalizing behavior problems and physiologic and neuroendocrine disruption; Repetti et al. 2002). Thus, maternal depression may contribute to the accumulated risk of experiencing long-term adult health problems, including chronic pain and depression.

Axiom 2a: Risk amplification—CI theory explicitly suggests that social disadvantages not only exacerbate risk of later life health conditions but also amplify health disparities over time. As shown in Figure 1 (H3), I hypothesize that *low childhood SES amplifies people's risk of experiencing depression and/or pain as they age*. That is, risk diverges over time as disadvantage accumulates or, as Ferraro and Shippee (2009:334) note, “age is an index of life changes and the accumulation of inequality.” Economic disadvantage in early life may leave people more vulnerable to risks including food insecurity, economic tenuousness, and stressful home environments over the life course (Repetti et al. 2002). Conversely, those from more highly educated and economically secure backgrounds may experience continuous advantages throughout the life course, including economic stability, access to quality health care, and healthier coping strategies for dealing with stress (Hatch 2005). These factors may thus contribute to adult socioeconomic differences in depression and pain.

Axiom 2b: Disadvantage across domains—According to Axiom 2b, inequality is not limited to only one domain but may diffuse across multiple domains. In this study, depicted in the H4 pathway of Figure 1, I posit that *childhood disadvantage not only elevates the risk of experiencing major depression and pain in adulthood but that adulthood SES is a key conduit through which adult health conditions emerge*. Economic and health inequalities can persist across generations; youth who experience childhood poverty are at higher risk of being poor in adulthood (Wagmiller et al. 2008) and have worse health relative to their non-poor counterparts, thus establishing a cycle that can be difficult to alter (Hamil-Luker and O’Rand 2007). Moreover, low-SES adults report higher rates of functional disability, depression, chronic pain, generally worse health, and earlier mortality than high-SES adults (Cohen et al. 2010; Lampe et al. 2003; Miech and Shanahan 2000; Power and Matthews 1997). Thus, adult SES may serve as an important connection linking childhood disadvantage to adult health (Hayward and Gorman 2004).

Economic hardship, however, may not completely attenuate the impact of childhood disadvantage on adult depression (Gilman et al. 2002) and/or pain (Jones, Power, and Macfarlane 2009). Walsemann, Gee, and Geronimus (2009), in their longitudinal examination of race differences in life course depressive symptom trajectories, found that the influence of offspring family background (i.e., parental education and occupation) was independently associated with the offspring’s depressive symptom trajectories through adulthood, even after accounting for adult offspring SES characteristics including educational attainment and family income (Walsemann et al. 2009). Consequently, they argue that adult economic disadvantage may not accurately capture life course accumulation of disadvantage and that “family background [...] experiences may continue to resonate into adulthood” (Walsemann et al. 2009:95). Adults from low-SES backgrounds also have a twofold higher risk of diagnosed major depression relative to adults from high-SES backgrounds even after accounting for their own adult SES (Gilman et al. 2002, 2003). Taken together this literature reinforces the importance of childhood circumstances for adult health in both indirect and direct ways.

DATA AND METHODS

Data

I use data from the National Comorbidity Survey Replication (NCS-R) for the present analysis. The NCS-R is a nationally representative survey of U.S. adults aged 18 and older.

Face-to-face household surveys were conducted with 9,282 English-speaking adults between 2001 and 2003 in the coterminous United States based on a multistage clustered area probability sample design (Kessler et al. 2004). The response rate for the full sample was 70.9 percent. The survey consisted of two parts. Part I was administered to all respondents in the study, with data collected on demographic characteristics and diagnostic assessments. Part II was administered to a subsample of 5,692 respondents, which included all respondents who screened positive for at least one Part I disorder as well as a probability subsample of remaining respondents. The remainder of the Part II survey portion included additional questions about psychological disorders and their correlates. Analyses here were performed using the Part II portion of the sample, which was weighted to adjust for differential probabilities of selection (i.e., undersampling of respondents with no psychological disorders).

For the purpose of this study, the sample was further restricted to adults ages 25 to 64 to cover the distribution of working-age adults only. Multiple imputation by chained equations in Stata 12 (the *ice* command; Royston 2004, 2005) was employed to handle missing data (Little and Rubin 2002). The sample included only cases that were not missing on the dependent variables, and analyses were replicated across 10 imputation data sets. The results were combined to produce final estimates using Rubin's rules (see Schafer 1999). The final sample size for the analysis was 4,339. As noted previously, the data were obtained using a multistage probability sample to adjust for multiple levels of nesting within standard metropolitan statistical areas and primary sampling units. In this sample there were 42 standard metropolitan statistical areas and 84 primary sampling units. To account for the nonindependence of observations due to clustering at the household level, all analyses are conducted in Stata 12 using the built-in survey procedures, which adjusted for the complex survey design (StataCorp 2007).

Dependent Variables—Meta-analytic study results show that anywhere from 15 percent to 100 percent of depressed patients also report experiencing pain (Bair et al. 2003). Major depressive disorder is manifested as a combination of symptoms including sadness, trouble sleeping, and lack of engagement in pleasurable activities, which can be so severe that they may interfere with daily life (Department of Health and Human Services 2011). In this study, major depression is measured using the dichotomous item *12-month major depressive episode*, assessed using the *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition (American Psychiatric Association 2000). Those who experienced the condition were coded as 1 and those who did not were coded as 0.

Chronic pain is a dichotomous measure created from a battery of questions in the NCS-R about chronic physical health problems, which combines a lifetime measure of arthritis and other pain conditions reported during the past 12 months. For arthritis, respondents were asked, (1) "Have you ever had *arthritis/rheumatism*?" For the remaining pain conditions respondents were asked, (2) "Did you still have *back or neck problems* or receive any treatment for them at any time during the past 12 months?" (3) "*frequent or severe headaches*?" and (4) "*any other chronic pain*?" Of the respondents, 25 percent reported having *arthritis/rheumatism*. Of those who reported having any of the other chronic health conditions over the past 12 months, the weighted proportions for respondents reporting each individual outcome were .67, .57, and .70, respectively. The individual items were condensed into a dichotomous measure where 0=*no pain* and 1=*answering "yes" to one or more chronic conditions*. For the purpose of testing Hypotheses 1 through 4, the two dichotomous measures for depressive symptoms and chronic pain were combined into a four-level categorical variable. The four categories are (1) *no depression or chronic pain*, (2) *depression only*, (3) *chronic pain only*, and (4) *depression and chronic pain*.

Independent Variables

Childhood family SES: Several indicators of childhood SES and severity of family economic hardship are included in the analysis. *Parental education* is calculated by measuring the average number of years of education among parents in the household. *Childhood AFDC* (i.e., *Aid to Families with Dependent Children*) receipt is measured using a dichotomous measure asking if the family received government assistance for six months or more during childhood and/or adolescence (0=*no*; 1=*yes*). Finally, to capture the severity of economic hardship in the household during childhood, *went hungry during childhood* was measured with the following question: “How often did your parents or caregivers make you go hungry or not prepare regular meals?” (0=*never* to 3=*often*). To test whether the association between childhood disadvantage and the experience of adult depression and/or pain is amplified with age, interactions were created for each of the childhood SES indicators with age.¹ To make the interpretation of the age intercept more meaningful, age was mean centered at 42. In addition, the interaction variables were transformed (original variable/10) such that the reported coefficients indicate a unit increase of age equals 10 years rather than 1, to provide a clearer interpretation of the coefficients.

Maternal mental health and parenting: The primary measure of the respondents’ *mothers’ depression* was measured using a dichotomous item asking, “During the years you were growing up, did your mother or the woman who raised you ever have periods lasting 2 weeks or more where she was sad or depressed most of the time?” (0=*no*; 1=*yes*).² Maternal closeness and parenting behavior are also controlled for. *Maternal closeness* was measured by asking, “How emotionally close were you with her while you were growing up—very close, somewhat, not very, or not at all?” Parenting behavior was measured using *frequency of physical punishment*. The item was created from a measure asking the following question: “How often did your mother parent/guardian push, grab, shove, throw something, slap, or hit you?” Responses were coded 1=*never*; 2=*rarely*; 3=*sometimes*; 4=*often*.

Adulthood characteristics: Adult SES is assessed using the following measures: *employment status*, *household income*, *level of education*, and *government assistance in adulthood*. *Employment status* is measured using a set of three dichotomous variables: (1) *employed (the reference category)*, (2) *unemployed (seeking work)*, and (3) *not in the labor force*. *Household income* was measured as the log of respondents’ current household income. Respondents’ highest level of *education* was measured using a continuous measure in years of education (range: 4 years to 17+ years). *Adulthood government assistance* was asked as follows: “Have you ever received public assistance or welfare since turning age 18—such as Aid to Families with Dependent Children, General Assistance, or Temporary Assistance for Needy Families?” (0=*no*; 1=*yes*).

Demographic controls: Demographic controls in the models include age, marital status, race-ethnicity, and sex. Respondents’ *age* is measured using the continuous measure in years of age. *Marital status* is categorized as dummy variables for married, never married, and divorced or separated, with married as the reference category. Respondents’ *race and ethnicity* was measured with white as the reference category and dummy variables for black, Hispanic, and other. Given the small number of individuals in the Asian and other groups, the two categories were combined. *Sex* was measured as *male*=0 and *female*=1.

¹Exploratory analyses assessing nonlinear specifications of age by testing interactions using age² found no evidence of nonlinear relationships between age and the childhood socioeconomic status measures.

²For the purpose of this study, I focus on maternal depression and parenting because mothers are traditionally the primary caregivers in the home.

Analytic Design

As mentioned previously, a four-level categorical variable (i.e., no conditions, depression only, pain only, or both conditions) was used to examine the relationship between childhood circumstances and major depression, chronic pain, or both conditions. Hypotheses 1 through 4 were tested using a series of multinomial logistic regression models (Long 1997). The results are reported for the multinomial models with “no depression and no chronic pain” as the reference category. The regression coefficients are exponentiated and are thus presented as relative risk ratios (RRR) in Tables 2–4. The outcome category in Table 2 is the risk of experiencing depression only; Table 3 reports the risk of experiencing pain only, and Table 4 reports the risk of experiencing both pain and depression. The multivariate analysis includes a series of models testing the CI axioms by first introducing childhood SES characteristics controlling for background measures (M1; H1), followed by the introduction of maternal depression (M2; H2) and parenting variables. Next, I introduce the *cumulative inequality* age interactions (M3; H3). Finally, the models account for the effects of adulthood SES (M4; H4).

RESULTS

Descriptive Results

Table 1 provides the weighted descriptive statistics for the analytic sample. Major depression and chronic pain are comorbid outcomes for roughly 5 percent of adults in the sample, while approximately 53 percent of the sample reported no experience with major depression or chronic pain. While just 3 percent of the sample reported having had only major depression in the past 12 months, 39 percent reported chronic pain.³ The post hoc chi-square test results confirm that major depression and chronic pain are significantly associated with one another ($\chi^2=59.1$; $df=1$; $p=.000$).

Among the childhood SES characteristics, average parental education is 11.6 years, and just over 10 percent of the sample reported receiving government assistance in the form of AFDC during childhood. Most of the sample reported never experiencing childhood hunger (93 percent), and approximately 25 percent of the participants reported having mothers who had bouts of depression lasting two or more weeks. The majority of the participants reported feeling close to their mothers (62 percent). About 90 percent said their mothers never physically punished them, 5 percent reported rarely receiving physical punishment, and the remainder reported punishment sometimes (4 percent) and often (2 percent). Regarding adulthood characteristics, 74 percent of the sample was employed and approximately 59 percent had greater than 12 years of education (30 percent have 13–15 years, and 29 percent have 16 or more years). Finally, around 20 percent of the sample reported receiving welfare assistance as adults.

Multivariate Results

Depression only—Model 1 of Table 2 shows that, of the indicators of childhood SES, only the experience of hunger is associated with a higher risk of adulthood depression compared to individuals having no conditions, net of controls (RRR=1.51; $p<.01$). Childhood hunger remains significant after maternal depression and parenting factors are introduced in Model 2. Model 2 also shows that having a mother who experienced depression multiplies a respondent’s relative risk (RR) of having depression by 1.9 relative to no conditions compared to persons whose mothers did not experience depression. This relationship remains robust and persists throughout the analysis. Model 3 includes the CI

³The lifetime distribution of specific chronic pain conditions (i.e., “Ever having pain”) are as follows: back and neck=.30; frequent or severe headaches=.25; other chronic pain=.10.

interactions; there is modest evidence of childhood AFDC receipt amplifying the risk of adulthood depression as people age (RRR=1.48; $p<.10$), but this effect is attenuated in Model 4 with the inclusion of adult SES measures. Finally, in Model 4, of the adulthood SES indicators, not being in the labor force is associated with an elevated risk of experiencing major depression (RRR=1.63; $p<.01$) relative to no symptoms. In sum, the results show some support for CI theory, indicating that certain early life socioeconomic conditions and maternal mental health are associated with the risk of major depression in adulthood.

Chronic Pain Only—Table 3 shows the chronic pain only versus no conditions results. Unlike the depression only results in Table 1, all indicators of childhood SES exhibit significant associations with the RR of adulthood chronic pain relative to no conditions. Model 1 shows that higher parental education is associated with a lower RR of experiencing chronic pain (RRR=.97; $p<.05$) versus no symptoms while childhood AFDC receipt and going hungry increased the RR of chronic pain relative to no symptoms by factors of 1.35 and 1.30, respectively. These relationships remain significant until the inclusion of adulthood SES measures. Further, maternal depression multiplies the RR of pain relative to no symptoms by 1.45 compared to those who did not report having a depressed mother. Similar to the depression analysis above, this relationship is robust and persistent even after the inclusion of maternal punishment and closeness (M2). Moreover, individuals who reported being close to their mothers had a lower risk of experiencing chronic pain relative to no symptoms (RRR=.91; $p<.05$).

Model 3 provides evidence that the impact of parental education on the risk of experiencing chronic pain versus no symptoms in adulthood is moderated by age. Figure 2 graphically depicts this interaction and shows results that are largely consistent with the CI hypothesis of amplified inequality. For simplicity, parental education is represented using the following categories: parents with 10 years of education, parents with 12 years of education, and parents with 16 years of education. The results unexpectedly suggest that among younger working-aged adults the predicted probability of reporting chronic pain is higher for individuals whose parents have 16 years of education relative to individuals whose parents have less education. Around the mean age of 42, the RRs converge for all educational categories. Subsequently, the probability of individuals experiencing pain becomes higher for adults whose parents have fewer years of education, supporting the CI hypothesis. Post hoc analyses, however, show that differences across groups prior to age 42 were not statistically significant, but the divergence in patterns between groups by age 59 are statistically significant at the $p<.05$ level.⁴ Put simply, differences by parental education in the probability of individuals' experiencing chronic pain begin during middle adulthood and culminate into marked differences by older adulthood. This relationship is not attenuated by adult socioeconomic factors.

In the final model, Model 4, adulthood income, education, and welfare receipt are all significantly and independently associated with the risk of individuals experiencing chronic pain relative to no conditions at all (M4). Both adult income and education, as they increase, lower the RR of adults experiencing chronic pain relative to no symptoms (RRR=.86 and .95, respectively). Respondents who reported receiving welfare in adulthood have a RR of experiencing pain relative to no symptoms that is 1.4 times larger. The inclusion of these measures attenuates the childhood poverty and pain relationship but does not attenuate the parental education by age interaction. In sum, these results show that both childhood

⁴Analyses were conducted adjusting the mean age included in the age*parental education to assess when group differences by age became statistically significant after age 42. In the full analytic models where mean age was adjusted to 59 and multiplied by parental education, the interaction was significant at $p<.05$.

circumstances and adulthood SES are important predictors of adult chronic pain. Further, there is evidence that the risk for experiencing chronic pain is amplified with older age among adults whose parents have lower educational attainment.

Chronic Pain and Depression—Table 4 reports the results assessing the childhood and adulthood factors associated with the risk of individuals experiencing both chronic pain and depression versus no symptoms. Among the childhood SES measures in Model 1, as parental education increases, the risk of adults experiencing depression and chronic pain decreases (RRR=.96; $p<.10$), while childhood AFDC receipt and going hungry during childhood are associated with a higher RR of experiencing both chronic pain and major depression relative to no symptoms (RRR=1.70 and 1.74, respectively; $p<.01$). As in the previous models, maternal depression is salient for the RR of experiencing chronic pain and depression. Specifically, respondents who reported having a depressed mother had a 1.84 times higher RR of reporting both depression and pain relative to no conditions at all. As in the prior analysis, this association persists across models.

In Model 3, there was no evidence that the effects of childhood disadvantage are amplified with age. In the final model (M4), adult SES characteristics are included. Here, a modest amplification effect of childhood hunger by age emerges with the introduction of adult SES measures. Labor force participation, income, and welfare receipt are all associated with the risk of individuals experiencing both pain and depression versus no conditions but do not attenuate the role of childhood hunger or maternal depression for elevating the risk of experiencing chronic pain and depression. Respondents who were unemployed exhibited 1.73 ($p<.05$) times higher RR of experiencing both pain and depression relative to no symptoms, while not being in the labor force was associated with a 2.64 times higher RR of experiencing both pain and depression ($p<.001$) versus no symptoms. Adults who received welfare since they were 18 were at a 1.69 times higher RR of experiencing both conditions versus no conditions. Taken together, the results provide little evidence of risk amplification for people with low childhood SES backgrounds. They provide support, however, for CI theory by demonstrating the importance of adulthood SES conditions as a conduit of risk for the influence of childhood SES factors on adult comorbid depression and chronic pain.

DISCUSSION

This study provides evidence that both childhood disadvantage and maternal mental health are important early life course circumstances that elevate the risk of U.S. adults' experiencing depression, chronic pain, or both conditions. The results support the axioms posited by CI theory that childhood disadvantage not only shapes later life health outcomes but that the health inequalities associated with early life conditions are amplified as people age. By focusing on the working-aged adult population, this study was able to document how early life course circumstances affect key health outcomes that are correlated with conditions such as functional disability as people age into retirement.

The results show support for Hypothesis 1, that childhood disadvantage increases the risk of individuals experiencing depression and/or pain versus no symptoms. Axiom 1a of the CI theory notes that childhood circumstances are salient for adulthood outcomes (Ferraro and Shippee 2009). In this study, childhood hunger is the most robust SES predictor of adult depression, while all childhood SES conditions were significant predictors of experiencing both adult pain and depression versus no conditions. Food insecurity and hunger are particularly harmful for children's mental and physical health; children who experience hunger or food insecurity report higher rates of chronic illness and internalizing behavior problems (Weinreb et al. 2002). Moreover, in families where food insecurity is present, mothers tend to also report severe depression, physical health complaints, and high rates of

posttraumatic stress disorder (Siefert et al. 2004; Wehler et al. 2004). Therefore, respondents who report the experience of childhood hunger in this study may represent people who faced more severe levels of childhood poverty, thus explaining the strong significant relationships between experiencing hunger and the higher RR of reporting both chronic pain and major depression.

Similarly, maternal depression is a robust predictor of adult mental and physical health, supporting Hypothesis 2. According to CI Axiom 1b, physical and social environments both play key roles in shaping health inequalities over the life course. Biological processes may tie depression to rates of morbidity/mortality through immune dys-regulation, or physiological responses of the immune system to stressors. The reactivity of the immune system, in turn, leads to an increased susceptibility to physical illness and poor mental health (Kiecolt-Glaser and Glaser 2002). Specifically, individuals with parents who suffer from depression or other health conditions are more vulnerable to negative reactivity in risky family environments (Taylor, Repetti, and Seeman 1997). These data do not provide the means to test physiologic responses to stressful life conditions or to measure the extent to which genes contribute to the propensity for individuals to experience major lifetime depression. In this study, however, 4 percent of adults diagnosed with major depression and 11 percent of people with chronic pain in the past 12 months reported having a depressed mother. In addition, 13 percent of respondents who experienced chronic pain in their lifetime and 7 percent who experienced major lifetime depression had depressed mothers. While these rates were low, their associations were nontrivial.

Whether the risk of experiencing depression and/or pain is amplified with age for people who had lower SES in childhood (H3) received some support. Specifically, childhood hunger moderately amplified the risk of experiencing pain individually and depression and pain simultaneously, but the most robust effect was for the role of parental education in elevating the risk for chronic pain. Unexpectedly, higher parental educational status was related to a modestly higher risk of reporting chronic pain in early adulthood, perhaps suggesting that younger adults from more highly educated backgrounds are more likely to identify and seek treatment for their pain symptoms—symptoms that may not be associated with high stress or strenuous work conditions. Moreover, young adults with more highly educated parents may perceive experiencing pain earlier in adulthood as atypical in healthy early life course aging and thus may be more inclined to seek treatment and have the resources to do so.

For adults with less educated parents, the social etiology of chronic pain may differ. Adults from families with less educated parents are more likely to engage in risky behaviors such as smoking, which is associated with chronic pain (Thomas et al. 1999). Moreover, adults from lower-SES backgrounds are also more likely to engage in more strenuous and repetitive work earlier, which is associated with early chronic health condition onset in middle age (Power and Matthews 1997). Consequently the pattern illustrated in Figure 2 may reflect accumulated risk of not only poor childhood conditions but the stressful life conditions associated with low SES in adulthood that leave individuals vulnerable to experiencing chronic pain in middle age.

This evidence of risk amplification supports existing literature showing that years of education is a robust predictor of chronic illness (Dupre 2007) and mortality (Brown et al. 2012; Montez, Hummer, and Hayward 2012). Educational disparities in health and mortality have grown substantially over the past 20 years, and years of education, regardless of credentialing (e.g., diploma, college degree) are salient factors for health outcomes (Montez et al. 2012). Adults with 12 or fewer years of education may live with structural constraints that, for example, limit their ability to live in safe neighborhoods and hold occupations that

provide healthy, autonomous work environments. Furthermore, such occupations may limit access to benefits and health care, thus regulating their risk of engaging in unhealthy lifestyle behaviors (Glass and McAtee 2006) and exacerbating their risk of experiencing chronic health conditions. Offspring are embedded in their parents' life course, thus creating related opportunities and risks for illness over their life course. Moreover, the reproduction of health across generations may in part be based on changing norms around healthy life course aging that could vary by educational attainment and SES. Consequently, offspring health trajectories are informed by shared family context during childhood, thereby creating a key pathway for class replication and perpetuation of poor health in adulthood (Walsemann et al. 2009).

Whether adulthood economic conditions would attenuate the relationship between childhood disadvantage and adult pain and depression postulated in Hypothesis 4 was partially supported. As posited in Axiom 2b of CI theory, the results show that indicators of adult socioeconomic conditions also independently predict the risk of reporting chronic pain and/or depression in adulthood. Accounting for adult SES slightly attenuated the associations between childhood hunger and the risk of experiencing adult pain and depression versus no symptoms independently. For the comorbid depression and pain outcome, adulthood economic conditions reduced parental education and childhood AFDC receipt to nonsignificance, while hunger was slightly reduced in magnitude; it remained a robust childhood circumstance that elevates the risk of experiencing chronic pain and depression. Prior literature explicitly assessing the role of childhood conditions for the occurrence of widespread chronic pain in adulthood demonstrates that both childhood economic conditions along with adult working conditions and SES are powerful predictors of chronic pain in adulthood (Jones et al. 2009). The continued salience of childhood conditions for adult health, even after accounting for adult socioeconomic conditions, may reflect the accumulation of behavioral, environmental, and physiological risk associated with poor SES over the life course. Power and colleagues (1997, 2007) report that adults with lower SES backgrounds engage in health-damaging behaviors earlier in adulthood, have less secure employment, and have more psychosocial job strain.

Certain adult SES conditions were more salient for the risk of experiencing different constellations of pain and depression. For depression only, not being in the labor force is associated with the risk of experiencing major depression relative to no conditions, although causal order cannot be assessed. Having major depression may lead to leaving the labor force, suggesting that such individuals may be too impaired to participate in regular employment. Conversely, having to leave the labor force may, in fact, exacerbate the risk of experiencing major depression. Similarly, people who received welfare after the age of 18 are more likely to report chronic pain, which supports a significant body of literature reporting that welfare recipients are more likely to suffer from chronic health conditions (Yoshimama, Hammock, and Horrocks 2006) relative to those who do not. Moreover, higher income and educational attainment are protective and associated with a lower risk of experiencing or being treated for chronic pain.

Comorbid chronic pain and depression represent health conditions that can be debilitating when severe. Unemployed adults and those receiving welfare may find it more difficult to obtain and keep employment, particularly when dealing with ongoing, reciprocating conditions. Clinical studies report that people experiencing depression also are more likely to anticipate experiencing more severe pain, thus exacerbating their already existing depression (Bair et al. 2003). Together, these conditions increase the risk of being unable to consistently maintain employment. Consequently, people who are not in the labor force may signify the most severely impaired group with their RR of having both conditions versus no conditions 2.64 times larger than that of a working-aged adult who is employed.

This study is not without important limitations, and the results should be viewed with a measure of caution. In this sample, respondents were asked to report on their parents' SES, hardship experiences during childhood and adolescence, and whether or not their mothers experienced depression. The reliability and validity of the results can be compromised in the following ways: First, in this sample, some adults reporting on prior conditions may also be experiencing pain and/or depression at the time of reporting.⁵ Prior research suggests that depressed adults may report more childhood adversity compared to nondepressed adults, although Schraedley, Turner, and Gotlib (2002) found that depressed adults who reported parent psychopathology did not differ based on severity of their depression. In addition, asking adults to report on certain childhood socioeconomic conditions may also be underreported, in part, due to the fact that adults may not have been aware of their parents' receipt of welfare (Hardt and Rutter 2004). Furthermore, reporting on events such as hunger and maternal depression may be limited to the most severe conditions and those that happened when offspring were older. Infantile amnesia is a common condition where adults are unable to recall events that happened in the first three or so years of life (Hardt and Rutter 2004). Consequently, early childhood circumstances may not be accurately reported without prospective data.

In addition, there is growing evidence that the timing, duration, and severity of childhood conditions also have implications for health over the life course (Ferraro and Shippee 2009). In this study, the variation in timing and duration of economic disadvantage and maternal depression cannot be assessed. Not accounting for variations in childhood exposure to poverty and its reciprocal relationship with parental health may miss nuances in differential exposure and their contribution to the onset of health conditions. As the duration and severity of poverty increase during childhood, the life chances and health of offspring decline (Kuh and Ben-Shlomo 2004). Furthermore, there is evidence that poverty timing and exposure have differential impacts on both parents' health outcomes and the economic outcomes of their offspring in adulthood (Wagmiller et al. 2008). Finally, this study cannot account for childhood health conditions, which may also confound the findings in this study.⁶

Despite these shortcomings and the lack of prospective data that can address childhood circumstances and onset of depression and pain, the findings support the growing literature demonstrating that early life conditions are instrumental in shaping health outcomes in working-aged adults. Though the literature examining the links between childhood poverty and adult health is more extensive, few studies address the consequences of maternal depression for adult offspring mental and physical health. Pain and depression are conditions that elevate the risk of functional impairment in adults and share similar early childhood socioeconomic origins. Understanding the degree to which these conditions are shaped by the intergenerational transmission of inequality may help shed light on the role of early childhood circumstances for the onset and progression of chronic health conditions across the life course.

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⁵Accounting for contemporaneous medical treatment for depression did not change the major results in additional analyses. Results are available upon request.

⁶Additional analyses were implemented controlling for self-rated health. The major findings were unchanged, but these analyses are available upon request.

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Biography

Bridget J. Goosby is an assistant professor of sociology at the University of Nebraska–Lincoln. Her research examines how interactions between social and biological processes influence the development of mental and physical health disparities over the life course and across generations. Her research has appeared in *Social Forces*, *Social Science Research*, *Society and Mental Health*, *Health and Place*, and other venues.

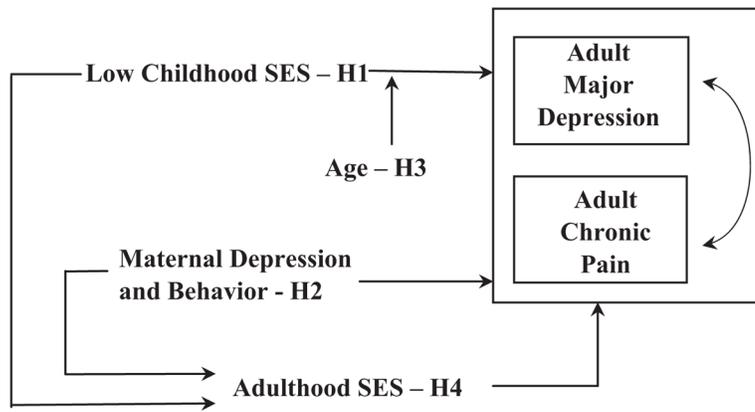


Figure 1. Cumulative Inequality Pathways of Adult Major Depression and Chronic Pain.

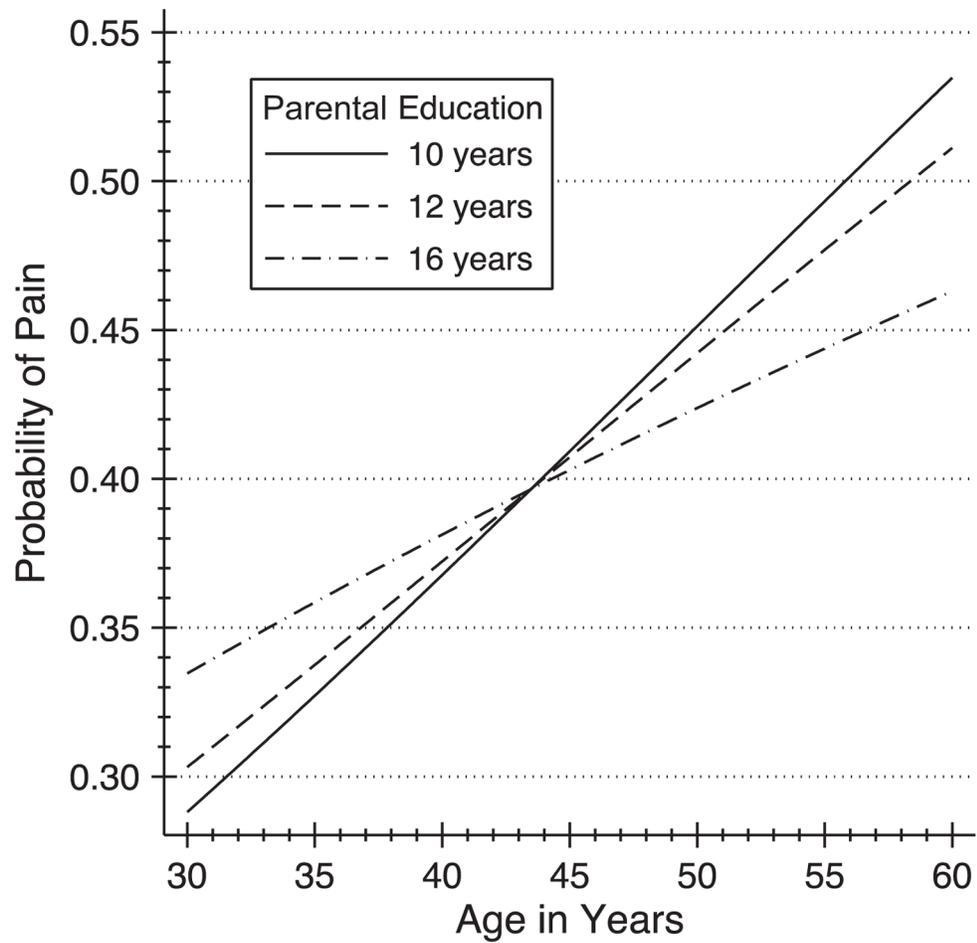


Figure 2. Moderating Effects of Age and Parental Educational Attainment on the Predicted Probability of Offspring Adult Chronic Pain.

Table 1

Weighted Proportions of Analysis Sample (N=4936).

Variable	Proportion^a
<i>Adulthood depression and chronic conditions[*]</i>	
No depression/no pain	.53
Depression only	.03
Chronic pain only	.39
Depression and chronic pain	.05
<i>Childhood SES</i>	
Parental education ^a	11.60
Family AFDC receipt	10.37
Went hungry	
Never	.93
Rarely	.03
Sometimes	.03
Often	.01
<i>Maternal mental health and behavior</i>	
Maternal depression	.25
Mother physical punishment	
Never	.89
Rarely	.05
Sometimes	.04
Often	.02
Closeness to mother	
Very	.62
Somewhat	.26
A little	.09
Not at all	.03
<i>Adult SES</i>	
Employed	.74
Unemployed	.04
Not in labor market	.22
Highest education	
12 yrs	.41
13–15	.30
16+	.29
Income (ln) ^a	10.74
Welfare receipt since 18	.20
<i>Respondent demographic characteristics</i>	
Female	.58
Race	

Variable	Proportion ^a
Black ^b	.13
Latino	.09
Other	.05
Divorced/Separated ^c	.21
Never married	.16
Age (years) ^a	42.32

Note: Descriptives are reported on nonimputed sample. SES = socioeconomic status; AFDC = Aid to Families with Dependent Children.

^aMean reported.

^bWhite is the reference category.

^cMarried is the reference category.

* Wald statistic for depression/pain group membership: $\chi^2=81.4$; $df=42$; $p=.000$.

Table 2

Relative Risk Ratios from Multinomial Logit Models: Depression Only versus No Conditions among Working-Age Adults (N=4339).

	M1	M2	M3	M4
<i>Childhood SES</i>				
Parental education	.98	.97	.97	.98
Childhood AFDC	.95	.82	1.03	.93
Went hungry	1.51**	1.32*	1.35*	1.32 [†]
<i>Maternal factors</i>				
Maternal depression		1.91***	1.95***	1.92***
Physical punishment		.99	.99	.99
Close to mother		.89	.88	.89
<i>Adulthood SES</i>				
Unemployed				.88
Not in labor force				1.63**
Income (ln)				.89
Education				1.00
Welfare receipt since 18				1.43
<i>Cumulative inequality</i>				
Parental Education * Age			.98	.99
Childhood AFDC * Age			1.48 [†]	1.45
Childhood Hunger * Age			1.14	1.17
<i>Controls</i>				
Age ^a	.82**	.82**	.91	.79
Female	1.41**	1.35*	1.37*	1.26*
African American	.84	.93	.93	.81
Hispanic	.96	1.01	1.04	1.01
Other race	.71	.71	.71	.67
Never married	2.10***	2.11***	2.06***	1.83***
Divorced/Separated	1.98***	1.89***	1.89***	1.68**

Note: All models are adjusted for nonindependence of observations due to clustering. SES = socioeconomic status; AFDC = Aid to Families with Dependent Children.

^aAge is mean centered.

[†]p<.10.

*p<.05.

**p<.01.

***p<.001.

Table 3

Relative Risk Ratios from Multinomial Logit Models: Pain Only versus No Conditions among Working-Age Adults (N=4339).

	M1	M2	M3	M4
<i>Childhood SES</i>				
Parental education	.97*	.97*	.97*	1.00
Childhood AFDC	1.35**	1.23 [†]	1.29*	1.16
Went hungry	1.30***	1.19*	1.21*	1.16 [†]
<i>Maternal factors</i>				
Maternal depression		1.45***	1.42***	1.42***
Physical punishment		1.05	1.05	1.06
Close to mother		.91*	.91*	.92 [†]
<i>Adulthood SES</i>				
Unemployed				1.10
Not in labor force				1.16
Income (ln)				.86***
Education				.95***
Welfare receipt since 18				1.40***
<i>Cumulative inequality</i>				
Parental Education * Age			.97**	.97**
Childhood AFDC * Age			1.01	1.00
Childhood Hunger * Age			1.14	1.16 [†]
<i>Controls</i>				
Age ^a	1.43***	1.44***	1.77***	1.68***
Female	1.55***	1.50***	1.50***	1.42***
African American	.76**	.80*	.79**	.70***
Hispanic	.79 [†]	.81	.83	.78 [†]
Other race	.99	.99	.99	.95
Never married	.93	.93	.91	.81
Divorced/Separated	1.28*	1.25*	1.25**	1.09

Note: All models are adjusted for nonindependence of observations due to clustering. SES = socioeconomic status; AFDC = Aid to Families with Dependent Children; (ln) = natural logarithm of income.

^aAge is mean centered.

[†]p<.10.

*p<.05.

**p<.01.

***p<.001.

Table 4

Relative Risk Ratios from Multinomial Logit Models: Major Depression and Chronic Pain versus No Conditions among Working-Age Adults (N=4339).

	M1	M2	M3	M4
<i>Childhood SES</i>				
Parental education	.96 [†]	.96 [*]	.96 ^{**}	.99
Childhood AFDC	1.70 ^{***}	1.49 ^{**}	1.57 ^{**}	1.27
Went hungry	1.74 ^{***}	1.53 ^{***}	1.55 ^{***}	1.46 ^{***}
<i>Maternal factors</i>				
Maternal depression		1.84 ^{***}	1.85 ^{***}	1.86 ^{***}
Physical punishment		1.04	1.04	1.04
Close to mother		.90	.90	.90
<i>Adulthood SES</i>				
Unemployed				1.73 [*]
Not in labor force				2.64 ^{***}
Income (ln)				.87 [*]
Education				.96
Welfare receipt since 18				1.69 ^{***}
<i>Cumulative inequality</i>				
Parental Education * Age			.99	1.00
Childhood AFDC * Age			1.10	1.08
Childhood Hunger * Age			1.17	1.20 [†]
<i>Controls</i>				
Age ^a	1.13 ^{**}	1.14 ^{**}	1.04	.94
Female	2.22 ^{***}	2.11 ^{***}	2.13 ^{***}	1.82 ^{***}
African American	.52 ^{***}	.58 ^{**}	.58 ^{***}	.46 ^{***}
Hispanic	.50 ^{***}	.53 ^{**}	.54 ^{**}	.50 ^{**}
Other race	1.40	1.39	1.39	1.24
Never married	1.72 ^{***}	1.73 ^{***}	1.71 ^{***}	1.51 ^{**}
Divorced/Separated	2.68 ^{***}	2.58 ^{***}	2.58 ^{***}	2.26 ^{***}

Note: All models are adjusted for nonindependence of observations due to clustering. SES = socioeconomic status; AFDC = Aid to Families with Dependent Children.

^a Age is mean centered.

[†] p<.10.

^{*} p<.05.

^{**} p<.01.

^{***} p<.001.