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Do Early-Life Social, Behavioral, and Health Exposures Increase Later-Life Arthritis Incidence?

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Abstract

Objectives: This study investigates direct and indirect influences of childhood social, behavioral, and health exposures on later-life osteoarthritis and rheumatoid arthritis development.

Methods: Drawing from cumulative inequality theory and six waves of the Health and Retirement Study (2004–2014), we estimate structural equation modeling-based discrete-time survival analysis of the association between six childhood exposure domains and both osteoarthritis and rheumatoid arthritis incidence for men (n = 2720) and women (n = 2974). Using the delta method to test for mediation, we examine indirect effects via selected health-related risks and resources.

Results: Risky adolescent behavior is associated with rheumatoid arthritis incidence for women (h.O.R. = 1.883, 95% C.I. [1.016, 3.490]), whereas several types of childhood exposures are associated with later-life osteoarthritis development for both men and women. Experiencing two or more childhood socioeconomic disadvantages is indirectly associated with osteoarthritis (men: coef. = 0.024, 95% C.I. [0.003, 0.045]; women: coef. = 0.111, 95% C.I. [0.071, 0.150]) and rheumatoid arthritis (men: coef. = 0.037, 95% C.I. [0.000, 0.074]; women: coef. = 0.097, 95% C.I. [0.035, 0.159]) development through adult body mass index.

Discussion: Findings highlight the importance of childhood contexts in understanding the development of later-life osteoarthritis and rheumatoid arthritis.

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Declaration of Conflicting Interests

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Supplementary Material

Supplementary material for this article is available online.

Keywords

adverse childhood experiences; osteoarthritis; rheumatoid arthritis; body mass index; cumulative inequality theory

Over the past few decades, arthritis prevalence has grown to include nearly half of adults aged 65 and over (Barbour et al., 2017). Arthritis presents tremendous personal, social, and economic costs that are projected to worsen in our aging society (Hootman et al., 2016). As a common co-morbid condition, arthritis creates barriers to physical activity and raises the risk of disability and complications associated with heart disease and diabetes (Centers for Disease Control and Prevention [CDC], 2009; Peek & Coward, 2000). In terms of economic burden, it was estimated in 2013 that the combined earnings losses and medical expenses due to arthritis was $303.5 billion—about 1% of the US gross domestic product (Murphy et al., 2017).

Arthritis is characterized by painful inflammation in joints. There are different forms of the disease with the most prevalent types being osteoarthritis, which is a multifactorial degenerative disease that often occurs later in life (Glyn-Jones et al., 2015), and rheumatoid arthritis, which is a systemic, autoimmune disease that can occur at any age though it commonly develops in later life (Alpízar-Rodríguez & Finckh, 2017). Although specific causes are not well understood, risk factors common to both osteoarthritis and rheumatoid arthritis such as obesity and genetics have been identified along with greater prevalence among women (Alpízar-Rodríguez & Finckh, 2017; Glyn-Jones et al., 2015). Despite similarities between osteoarthritis and rheumatoid arthritis, their development and progression are substantially different. Risk factors specific to osteoarthritis include joint injury and trauma (Glyn-Jones et al., 2015), and those specific to rheumatoid arthritis include infection, smoking, and some nutritional factors such as high sodium intake and occupational exposure to chemicals (Alpízar-Rodríguez & Finckh, 2017).

Theoretical advancements, empirical evidence, and the widespread push towards prevention and early detection have built a convincing case for considering early-life exposures as antecedents of disease development. Indeed, evidence suggests that early-life events and experiences such as abuse and parental addictions are associated with adult arthritis (Fuller-Thomson et al., 2009; Spitzer et al., 2013). However, the current literature is limited in several ways as pointed out by a recent scoping review; studies often fail to distinguish between different types of arthritis and do not examine social pathways by which early-life exposures may be related to adult arthritis development (Schwetlik et al., 2021).

Given differences in the etiology and pathogenesis by arthritis type, it is possible that some types of childhood events and experiences (e.g., abuse and socioeconomic disadvantage) are associated with adult risk factors specific to osteoarthritis and/or rheumatoid arthritis, highlighting unique social pathways in the development of these diseases. Moreover, studies that do not examine arthritis types according to pathophysiology could give rise to misleading conclusions and misinformed interventions. To address these limitations, this study uses a nationally representative sample of adults over the age of 50 to investigate the direct and indirect associations between several types of childhood exposure domains

(i.e., socioeconomic disadvantage, risky parental behavior, infectious disease, chronic diseases, impairments, and risky adolescent behavior) and the incidence of osteoarthritis and rheumatoid arthritis among men and women in later life. In addition, we examine potential social/behavioral and health pathways that have been linked to childhood exposures and represent risk factors for osteoarthritis and/or rheumatoid arthritis (e.g., body mass index (BMI) and smoking).

Theoretical Framework

Processes of accumulation are central to the development and progression of several diseases. Exposures to risks or stressors over a long period of time may be important for the etiology of morbidity. For instance, continuous heavy stress on joints may eventually result in osteoarthritis (Glyn-Jones et al., 2015). The present study is guided by cumulative inequality (CI) theory, a middle-range theory that offers a framework for understanding the social processes by which early-life exposures shape later-life health (Ferraro & Shippee, 2009).

Cumulative inequality theory posits that childhood is pivotal to adult health. Before children have much autonomy of their own, their status is reflective of their parent's status, placing some individuals at an early disadvantage. For instance, experiencing socioeconomic disadvantage during childhood is associated with worse health outcomes, lower levels of school achievement, and maladaptive social functioning during childhood (Bradley & Corwyn, 2002). Adverse childhood conditions also have been associated with several unhealthy behaviors and lifestyles during adulthood such as smoking, alcohol abuse, and obesity (Felitti et al., 1998; Ferraro et al., 2016).

Children's positions in other social hierarchies also may influence their early-life experiences. For example, studies are beginning to reveal that the ways in which early-life exposures are associated with adult health vary by gender; health-related mediators such as smoking and education (Morton & Ferraro, 2020) as well as coping resources (Lee et al., 2021) differ in their importance for understanding how early-life events shape later-life health for men and women.

In conceptualizing childhood as a sensitive period, CI theory underscores the importance of considering several types, or domains, of exposures, as they potentially influence the process of CI in different ways (Ferraro & Morton, 2018). Indeed, research has demonstrated the value of examining unique domains of childhood exposures in lieu of a single summary score measuring multiple types of childhood experiences (e.g., Morton et al., 2012). Kopec and Sayre (2004) found that unique effects of hospitalization and being extremely scared during childhood raised the risk of arthritis in adulthood whereas a single cumulative score of several childhood traumas was not statistically significant in predicting adult arthritis. Disaggregating childhood experiences offers greater specificity and may reveal additional nuances by gender. For instance, Spitzer and colleagues (2013) found evidence that physical and emotional abuse during childhood were associated with rheumatoid arthritis among adult women, but not men. Therefore, the current study considers multiple

domains of childhood exposures to elucidate how childhood acts as a sensitive period for the development of arthritis in later life among men and women.

Cumulative inequality theory also draws attention to how risks and resources shape health. Chains of risk may develop whereby early risks lead to subsequent adverse experiences but, resources, agency, and perceptions have the potential to alter life-course trajectories (Ferraro & Shippee, 2009). Some studies report that early-life exposures are associated with higher arthritis prevalence but give scant attention to how resources may mediate the relationship (Badley et al., 2018; Blackwell et al., 2001; Brennan-Olsen et al., 2018; Fuller-Thompson et al., 2009). Perhaps there are direct effects of noxious childhood exposures on health, but there also may be adult resources that mitigate the risk. Given differences in how osteoarthritis and rheumatoid arthritis develop, it also is plausible that different risks and resources create distinct pathways in the development of each disease.

Beyond identifying risk factors directly associated with osteoarthritis or rheumatoid arthritis, a logical next step is to consider pathways by which risk factors, such as fewer years of schooling, influence disease occurrence (Felitti et al., 1998; Ferraro et al., 2016). As an example of one potential pathway, childhood socioeconomic disadvantage has been associated with obesity and lower educational attainment in adulthood (Ferraro et al., 2016). Educational attainment is associated with higher risk of obesity and employment in occupations that require repetitive movements, each of which may elevate risk of osteoarthritis via stress placed on joints over long periods of time (Canetti et al., 2020; Glyn-Jones et al., 2015).

Additionally, researchers argue that early-life stress and trauma may lead to a pro-inflammatory state in adulthood, partially contributing to the pathogenesis of autoimmune diseases such as rheumatoid arthritis (Spitzer et al., 2013). It has been hypothesized that certain childhood experiences such as abuse and poverty can raise the risk of rheumatoid arthritis directly via a dysregulated hypothalamic-pituitary-adrenal (HPA) axis (Kopec & Sayre, 2004; Spitzer et al., 2013; Von Korff et al., 2009). Childhood factors related to adult rheumatoid arthritis also may operate indirectly through pro-inflammatory health-related coping mechanisms such as smoking and obesity. Experiencing adversity during childhood increases the risk of smoking and obesity in adulthood, risk factors for rheumatoid arthritis (Felitti et al., 1998). We acknowledge the importance of pro-inflammatory mechanisms but focus on modifiable pathways using previously identified risk factors such as smoking and obesity as intermediary variables in this study.

Early Social Origins of Arthritis

Although modest in size, there is a body of research documenting the early origins of adult-onset arthritis. Most of these studies report that childhood exposures such as physical abuse, trauma, and low parental socioeconomic status (SES) are associated with greater risk of arthritis in adulthood (e.g., Fuller-Thomson et al., 2009; Spitzer et al., 2013; Springer et al., 2007). The link is established in studies in the US (Blackwell et al., 2001; Brennan-Olsen et al., 2018), Canada (Badley et al., 2018; Fuller-Thomson et al., 2009), Germany (Spitzer et al., 2013), Turkey (Bayram & Erol, 2014), and in cross-national studies (Scott et al.,

2011; Von Korff et al., 2009). The literature to date is impressive, but we believe that future research should address three noteworthy limitations.

First, studies use widely different definitions of the outcome. Although there are notable differences between osteoarthritis and rheumatoid arthritis, most prior studies do not make such a basic distinction. Of 11 studies identified, only one study focused exclusively on osteoarthritis (Fuller-Thomson et al., 2009) and only two addressed rheumatoid arthritis—relying on information from clinical samples (Bayram & Erol, 2014; Spitzer et al., 2013). It is somewhat surprising that the bulk of the literature uses an undifferentiated term for self-reported arthritis; many studies examine “arthritis or rheumatism” (e.g., Kopec & Sayre, 2004). This observation was spelled out in a recent review of chronic stress and arthritis, calling for “further research investigating relationships between chronic stress and the development of specific forms of arthritis” (Schwetlik et al., 2021).

Second, most studies focus on a single type of childhood exposure such as abuse. Whereas socioeconomic disadvantage and risky parental behaviors during childhood have been associated with arthritis in previous studies (e.g., Fuller-Thomson et al., 2009; Spitzer et al., 2013; Springer et al., 2007), other childhood exposures also may raise the risk of arthritis. For instance, poor childhood health can influence physical activity and/or educational attainment (possibly important for the development of osteoarthritis) or alter the phenotype via dysregulation of immune genes (potentially important for the development of rheumatoid arthritis) (Butts & Sterngerg, 2004). Simultaneously investigating multiple childhood exposures enables identification of which exposures are most consequential to adult health—a particularly useful strategy when investigating outcomes with unique etiologies. Perhaps some childhood exposures have similar influences on both osteoarthritis and rheumatoid arthritis. Alternatively, certain childhood exposures may be associated with osteoarthritis and rheumatoid arthritis in distinct ways.

Third, most studies rely on cross-sectional data to examine the link between childhood stressors and the *prevalence* of arthritis or rheumatism. Many do not include information about onset, which means that it is impossible to determine whether the “reach” of childhood stressors is short-lived or extends into later life. Some studies, however, have asked about age of onset to better understand life-course processes and found that arthritis risk was higher for persons who experienced childhood adversity and developed an anxiety disorder early in life (e.g., Von Korff et al., 2009). Nevertheless, those authors called for more studies to examine the early origins of arthritis development. Taking a longitudinal approach to examine the development of arthritis in later life also enables temporal ordering of our key variables—childhood conditions and mediators are assessed prior to arthritis onset.

An exemplar of a longitudinal study on the topic is a three-wave study of people 18 years or older in Canada, which spanned 4 years. Using the National Population Health Survey, the authors found that childhood trauma was associated with *incident* arthritis, even among older adults (Kopec & Sayre, 2004). Notably, this study suggests that the effects of childhood negative exposures are not exhausted in early or middle adulthood. Despite its important contribution, this study examined the outcome of “arthritis or rheumatism,”

making it difficult to discern if there are distinct life-course antecedents of osteoarthritis and rheumatoid arthritis. In short, we are unaware of any population-based longitudinal study of the early origins of both osteoarthritis and rheumatoid arthritis, and this article is designed to address this gap in the literature.

Methods

Data and Sample

This study used six waves of data (2004–2014) from the Health and Retirement Study (HRS), a national multi-stage area probability panel study of adults over the age of 50 (Health and Retirement Study HRS, 2018). The HRS conducts interviews every 2 years and new cohorts are added every 6 years, making the HRS the largest and most representative panel study of older adults in the contiguous US. The HRS collects information on a range of economic and health factors including wealth, employment, psychosocial factors, and anthropometric and biomarker data.

Since we examine the onset of arthritis, or new cases of arthritis among older adults, individuals who had arthritis at baseline (2004) were excluded (n = 10,687). To minimize recall bias, our analytic sample excluded respondents who used proxies for their childhood exposure indicators (n = 1780), had item-missing on more than two-thirds of the childhood exposure indicators (n = 1), or had cognition scores more than two standard deviations below the mean (n = 104), for an analytic sample size of 5694.

Osteoarthritis and Rheumatoid Arthritis

Arthritis was measured as first diagnosis at each wave after the 2004 interview until 2014 with the question “have you ever had, or has a doctor ever told you that you have arthritis or rheumatism?” A follow-up question then asks about the type of arthritis the person has. We assessed arthritis with two measures: (1) osteoarthritis or arthritis due to injury and (2) rheumatoid arthritis, lupus, or gout (lupus is an autoimmune disease; by the HRS, gout and lupus are combined). To analyze incidence, separate indicators for each type of arthritis and each wave were created so that if the respondent experienced a new diagnosis at that wave, they are coded as 1, no new or past experience of arthritis was coded 0.

Childhood Exposures

Guided by theory, prior research, and psychometric analysis, we used the available data to create six domains of childhood exposures occurring before age 18 (Felitti et al., 1998; Ferraro & Morton, 2018; Ferraro & Shippee, 2009; Smith et al., 2019). For each domain, dichotomous childhood exposure indicators were summed and top coded at 2, except for the risky adolescent behavior domain, which was top coded at 1 due to small cell sizes. We dichotomized the four ordinal indicators to represent adversity or misfortune (e.g., fair/poor health and poor family finances) (Smith et al., 2019). Given item-missing data on indicators that make up the domains, we retained as much information as possible by coding the domain as 2+ if two or more exposures were reported in that domain regardless of missing on other indicators, an approach used by Smith and colleagues (2019). For the domain with the most item-missing (i.e., socioeconomic disadvantage), this technique reduced

missingness from 37% to 30%. The remainder of the missing data were handled using full-information maximum likelihood which is conceptually similar to multiple imputation and produces similar results (Schlomer et al., 2010).

The socioeconomic disadvantage domain includes father had an unskilled manual labor occupation, father with less than 8 years of education (mother's education is used if father's is missing), moved due to finances, and a rating of family finances as poor. Smoking, substance abuse and physical abuse make up the risky parental behavior domain. Using available data, the childhood infectious disease domain consists of measles, mumps, and chicken pox. The childhood chronic disease domain includes asthma, diabetes, respiratory disorders, allergic conditions, heart trouble, ear problems, epilepsy or seizures, headaches or migraines, stomach problems, high blood pressure, and self-rated health (poor or fair is coded as 1 and 0 otherwise). The childhood impairment domain consists of vision problems, speech problems, learning problems, severe head injury, and disability. Substance abuse, trouble with the police, depression, and other psychological issues (described as "any other emotional or psychological problems") make up the risky adolescent behavior domain.

Adult Exposures

Given strong evidence linking negative childhood conditions to education, BMI, and smoking, as well as evidence suggesting each of these are risk factors for osteoarthritis, rheumatoid arthritis, or both, we focused on these three adult factors as key intermediate variables. Education was measured as the number of years of schooling with a range of 0–17. Body mass index was measured as kg/m^2 calculated from self-reported height and weight. The number of pack-years smoked was created by multiplying the number of packs a respondent smokes daily and the number of years they have smoked; those who have never smoked are coded 0. These variables were measured in 2004 to establish temporal ordering, ensuring that our potential mediators are precursors to the development of arthritis.

Covariates

All covariates were taken from baseline interviews. Race and ethnicity was categorized as non-Hispanic Black, non-Hispanic White (reference), and Hispanic adults. Other races were excluded because there were too few cases for meaningful analysis ($n = 149$). Among adult SES variables, the cube root of total household wealth in thousands of dollars was used to adjust for skewness (Tukey, 1977), and manual occupation was coded 1 if the longest job held was a manual labor occupation. Models adjust for health insurance type: private insurance along with any other type (reference); Medicaid only; Medicare only; and no insurance. Marital status was measured with the categories: married or partnered (reference); divorced or separated; widowed; and never married.

In addition to BMI and smoking, three other health-related covariates were included. Heavy alcohol consumption was coded 1 for women who report drinking 4+ and men who drink 5+ drinks per day when they drink, 0 otherwise (Dawson, 2011); no additional information on what constitutes a drink is available. Physical activity was coded 1 if the respondent reported exercising at least once per week, 0 otherwise. Depressive symptoms were measured as a sum of the 8-item version of the CES-D scale (Karim et al., 2015).

Analytic Plan

We used discrete-time survival analysis to examine the probability of experiencing the onset of osteoarthritis and rheumatoid arthritis during follow-up as well as selected life-course pathways that potentially result in arthritis. A discrete-time model is preferred since information on arthritis is collected every 2 years in the HRS, yet specific dates of diagnosis are unknown. Instead of examining existing cases of arthritis, this method enabled us to observe new cases of arthritis that occurred among older adults (i.e., those at risk of experiencing arthritis over the study period), offering some insight to how far-reaching early-life exposures may be. A relatively recent approach has advanced discrete-time survival analysis in a structural equation modeling framework by integrating a latent variable model (Muthén & Masyn, 2005; Raykov et al., 2017). Among the benefits of this approach is the ease of exploring mediational relationships, along with the ability to incorporate measurement error (Muthén & Masyn, 2005; Pratschke et al., 2016). By combining discrete-time survival analyses and mediation methods in a structural equation framework, we can observe potential pathways that connect early-life exposures and the probability of developing arthritis later in life (Muthén & Masyn, 2005).

In these models, the latent variables represent the propensity to develop osteoarthritis and rheumatoid arthritis during follow-up, as measured by survival indicators at each follow-up wave, described previously (we reference terms used by Muthén and Masyn (2005), but our results and their interpretation do not imply causation). This is equivalent to modeling the effect of each explanatory variable on each of the five discrete-time incidence indicators, with each of these indicators constrained to be equal (Pratschke et al., 2016). Therefore, the latent variable reflects the underlying probability of developing arthritis over the observation period. This approach has been used to model other discrete health outcomes in the HRS (Raykov et al., 2017) as well as examine mediational pathways (Fairchild et al., 2015). Preliminary logistic regressions that pooled the five waves of data produced similar conclusions.

Using Mplus version 7, we estimated two sets of equations. First, we examined the extent to which childhood exposures are associated with the incidence of rheumatoid and osteoarthritis at each follow-up. Second, we investigated the extent to which select adult health-related and lifestyle factors, including smoking, BMI, and education mediate the associations between childhood exposures and arthritis incidence using a maximum likelihood robust estimator to produce standard errors for direct and indirect effects via the delta method (Muthén, 2011). Considering evidence suggesting potential sex differences in the etiology and pathogenesis of arthritis (e.g., influence of hormones, Alpízar-Rodríguez & Finckh, 2017; Glyn-Jones et al., 2015), in which childhood exposures are associated with arthritis (e.g., Spitzer et al., 2013), and in several adult health-related risks and resources (Read & Gorman, 2010), models were stratified by sex. Differences in coefficients for men and women were examined using a Wald χ^2 test. Item-missing data were handled using full-information maximum likelihood, and models were adjusted for the complex survey design to obtain robust standard errors.

Supplementary analysis.—To verify the robustness of results herein, we conducted a series of sensitivity analyses. First, in bivariate analyses we examine how left-censored respondents (i.e., those with arthritis at baseline) differ from those in the analytic sample. Those who had arthritis at baseline were more likely to be women, older, and non-Hispanic Black and White; have lower SES (education, wealth, worked manual occupations); report unhealthy lifestyles (smokers, higher BMI, less physically active); and have two or more childhood SES disadvantages, chronic diseases, and infectious diseases. Thus, interpretation of results should consider the robustness of the analytic sample.

Next, we examined a categorical specification of BMI to investigate a potential non-linear association between BMI and arthritis but there was no evidence of nonlinearity. Therefore, a continuous measure of BMI is used. Then, we examined the potential of sample selection. Although we removed 104 respondents at risk of arthritis with low cognition scores to reduce the possibility of recall bias, we re-estimated our models to include these individuals. Most conclusions were similar to the analyses presented, but two differences in the osteoarthritis models emerged. However, upon further inspection, we found that people with low cognition at baseline had significantly more item-missing data and differed from people without low cognition in distinct ways that may also bias results, such as more depressive symptoms among women with low cognition (Vuolo et al., 2014). Since cognition can interfere with recollection (Vuolo et al., 2014) and these individuals in our sample had significant nonrandom missing data, we decided to take a more conservative approach to exclude these respondents as is consistent with prior research attempting to reduce recall bias (Smith et al., 2019).

Lastly, we re-estimated models that examine rheumatoid arthritis incidence only given the differences between autoimmune diseases (i.e., rheumatoid arthritis and lupus) and gout, a form of arthritis caused by the accumulation of urate crystals. Our estimates are similar to estimates presented herein that predict developing rheumatoid arthritis, gout, or lupus with two exceptions possibly due to smaller cell sizes. Results from supplementary analyses are available upon request.

Results

Means (or proportions), standard deviations, and the range of all variables for men and women are shown in Table 1. Of those who did not have arthritis in 2004, 25% of men and 32% of women reported being diagnosed with osteoarthritis during the follow-up period. Roughly 8% of men and women reported developing rheumatoid arthritis between 2004 and 2014.

Osteoarthritis Incidence

Direct effects.—Table 2 displays the regression results for osteoarthritis incidence separately for men and women. In the male subsample (first two columns), among childhood exposures, one impairment (h.O.R. = 1.392, $p < .01$, 95% C.I. [1.092, 1.775]) and one or more risky adolescent behaviors (h.O.R. = 1.384, $p < .05$, 95% C.I. [1.026, 1.866]) compared to no exposures in each domain were directly associated with significantly higher hazard odds of osteoarthritis during the study period in fully adjusted models (we

use the terms hazard odds ratio and hazard odds following Masyn (2009) who describes the coefficients as the change in the logit hazard probability for one unit increase in the independent variable). Among adult risks and resources, higher BMI (h.O.R. = 1.038, $p <$ .001, 95% C.I. [1.021, 1.053]) and heavy drinking (h.O.R. = 1.419, $p < .05$, 95% C.I. [1.028, 1.958]) were associated with higher hazard odds of osteoarthritis.

In the female sample, age was associated with higher hazard odds of osteoarthritis (h.O.R. = 1.015, $p < .05$, 95% C.I. [1.001, 1.028]) and Black women had *lower* hazard odds of osteoarthritis than White women (h.O.R. = 0.676, $p < .01$, 95% C.I. [0.529, 0.862]). Among childhood exposures, two or more chronic diseases (h.O.R. = 1.328, $p < .05$, 95% C.I. [1.045, 1.687]), one infectious disease (h.O.R. = 1.548, $p < .05$, 95% C.I. [1.047, 2.289]), and one or more risky adolescent behaviors (h.O.R. = 1.411, $p < .05$, 95% C.I. [1.006, 1.980]) were associated with higher hazard odds of osteoarthritis compared to no exposures in the respective domains. Education (h.O.R. = 1.034, $p < .05$, 95% C.I. [1.005, 1.064]), higher BMI (h.O.R. = 1.053, $p < .001$, 95% C.I. [1.037, 1.069]), and depressive symptoms (h.O.R. = 1.080, $p < .001$, 95% C.I. [1.036, 1.125]) were associated with higher hazard odds of osteoarthritis during the study period for women. Comparing coefficients across models with the Wald χ^2 test, we found that the associations between experiencing one infectious disease ($\chi^2 = 9.90$, $p < .01$) and osteoarthritis was significantly different for men and women.

Indirect effects.—Next, indirect associations were examined; education, BMI, and smoking were tested as intermediaries between each childhood exposure domain and osteoarthritis. Results reveal that experiencing two or more SES disadvantages during childhood compared to no exposure to SES disadvantage was associated with higher adult BMI (coef. = 0.662, $p < .01$, 95% C.I. [0.179, 1.144]), and BMI was associated with higher osteoarthritis hazard odds among men (coef. = 0.037, $p < .001$, 95% C.I. [0.021, 0.052]). Although there was no significant direct effect of childhood SES on osteoarthritis, there was a significant indirect effect through adult BMI (indirect coef. = 0.024, $p < .05$, 95% C.I. [0.003, 0.045]) as displayed in Figure 1a.

Among women, one and two or more childhood SES disadvantages compared to none were associated with higher adult BMI (respectively: coef. = 1.267, $p < .001$, 95% C.I. [0.689, 1.846]; coef. = 2.125, $p < .001$, 95% C.I. [1.549, 2.701]) and lower educational attainment (respectively: coef. = −1.018, $p < .001$, 95% C.I. [−1.239, −0.797]; coef. = −2.584, $p <$ .001, 95% C.I. [−2.901, −2.267]) (see Figure 1a). Similar to men, there was not a significant direct effect of childhood SES on osteoarthritis. However, there were significant indirect effects through BMI (1 SES indirect coef. = 0.066, $p < .01$, 95% C.I. [0.031, 0.101]; 2+ SES indirect coef. = 0.111, $p < 0.001$, 95% C.I. [0.071, 0.150]) and education (1 SES indirect coef. = −0.033, $p < .05$, 95% C.I. [−0.064, −0.002]; 2+ SES indirect coef. = −0.083, $p < .005$, 95% C.I. [−0.159, −0.007]). Socioeconomic disadvantage during childhood was associated with higher adult BMI and less education in adulthood—and adult BMI and education each were related to higher hazard odds of osteoarthritis among women. The direct effect of one and two or more childhood SES disadvantages on BMI (1 SES: $\chi^2 =$ 6.008, $p < .05$; 2+ SES: $\chi^2 = 20.853$, $p < .001$) and the indirect effects of childhood SES on adult osteoarthritis risk via BMI differed for men and women (1 SES: $\chi^2 = 8.164$, $p < .01$;

2+ SES: $\chi^2 = 17.414$, $p < .001$). Smoking did not mediate the effects of childhood exposures on adult osteoarthritis for men or women.

Rheumatoid Arthritis Incidence

Direct effects.—Table 3 displays the discrete-time models predicting onset of rheumatoid arthritis from 2004–2014 separately for men and women. Age was associated with elevated rheumatoid arthritis incidence among men (h.O.R. = 1.028, $p < .05$, 95% C.I. [1.006, 1.050]). None of the childhood exposure domains were related directly to rheumatoid arthritis for men. Among health behaviors and lifestyle factors, BMI was associated with increased hazard odds of rheumatoid arthritis (h.O.R. = 1.058, $p < .01$, 95% C.I. [1.021, 1.095]).

Parallel results for women are shown in Table 3. Black women had a notably higher probability of developing rheumatoid arthritis during the study period compared to White women (h.O.R. = 2.030, $p < .01$, 95% C.I. [1.313, 3.136]). Moreover, women who report one or more risky adolescent behaviors had hazard odds of rheumatoid arthritis almost twice of those who reported no risky adolescent behaviors (h.O.R. = 1.883, $p < .05$, 95% C.I. [1.016, 3.490]); this finding was significantly different for men and women ($\chi^2 = 5.84$, $p < .05$; no association was found for men). Higher BMI (h.O.R. = 1.046, $p < .01$, 95% C.I. [1.016, 1.076]), depressive symptoms (h.O.R. = 1.087, $p < .05$, 95% C.I. [1.018, 1.160]), and heavy drinking (h.O.R. = 2.467, $p < .05$, 95% C.I. [1.137, 5.355]) were associated with higher hazard odds of rheumatoid arthritis among women.

Indirect effects.—Figure 1b illustrates indirect effects of childhood exposures on rheumatoid arthritis through adult BMI. Among men, experiencing two or more SES disadvantages during childhood compared to none was associated with higher adult BMI in adulthood (coef. = 0.666, $p < .01$, 95% C.I. [0.187, 1.146]), and BMI was associated with elevated hazard odds of rheumatoid arthritis (coef. = 0.056, $p < .01$, 95% C.I. [0.021, 0.090]). Among women, one and two or more SES disadvantages compared to none were associated with higher BMI (respectively: coef. = 1.323, $p < .001$, 95% C.I. [0.739, 1.906]; coef. = 2.270, $p < .001$, 95% C.I. [1.683, 2.856]), and BMI was associated with higher hazard odds of rheumatoid arthritis (coef. = 0.043, $p < .01$, 95% C.I. [0.013, 0.072]). Thus, the indirect effects through BMI were significant for both men (2+ SES indirect coef. = 0.037, $p < .05$, 95% C.I. [0.000, 0.074]) and women (1 SES indirect coef. = 0.056, $p < .05$, 95% C.I. [0.01, 0.103]; 2+ SES indirect coef. = 0.097, $p < .01$, 95% C.I. [0.035, 0.159]). The direct effects of two or more SES disadvantages on BMI ($\chi^2 = 21.86$, $p < .001$) and indirect effect of two or more SES disadvantages through BMI was different for men and women ($\chi^2 = 3.933$, $p < .05$). Neither education nor smoking mediated the effects of childhood exposures on adult rheumatoid arthritis for men or women.

Discussion

This study advances understanding of the epidemiology of arthritis in several ways. We examine osteoarthritis and rheumatoid arthritis as separate diseases, give close attention to childhood context by examining several domains of childhood exposures, illustrate how early exposures are indirectly associated with later-life osteoarthritis and rheumatoid arthritis

though adult risk factors, and examine arthritis *incidence* to gain insight to the reach of negative childhood exposures into later life. Different types of childhood exposures were directly related to either osteoarthritis or rheumatoid arthritis, however, the analysis revealed that childhood SES disadvantage was indirectly associated with elevated odds for both types. Moreover, the analyses for osteoarthritis and rheumatoid arthritis were on a sample of older adults who were arthritis-free in 2004, suggesting that childhood exposures have a long-range impact, directly and indirectly, on arthritis development in later life.

Although the older sample precludes us from observing arthritis development prior to age 50, our findings may be interpreted as the longitudinal progression of arthritis development in later life associated with early-life exposures. To examine arthritis incidence in later life and maintain temporal order, we excluded respondents who had arthritis at baseline resulting in a fairly robust analytic sample. In addition, late-onset rheumatoid arthritis (often defined as onset after the age of 60) may be a more severe form of the disease; it has been associated with greater disease activity, radiographic changes, inflammatory activity over time, and functional impairment (Innala et al., 2014). Therefore, associations between childhood exposures and arthritis incidence in this sample suggest that these early-life exposures may leave an indelible imprint that manifests decades later.

Unlike any prior study of which we are aware, the analyses presented herein reveal that *BMI acted as a mediator between childhood SES disadvantage and osteoarthritis as well as rheumatoid arthritis in later life*—and this was consistent for men and women. There are several ways in which childhood SES may influence adult BMI including sedentary behavior and dietary habits (Vazquez & Cubbin, 2020). Subsequently, a higher BMI raises the risk of osteoarthritis as well as rheumatoid arthritis by, respectively, putting stress on the joints and causing systemic inflammation (Felson et al., 2000; Festa et al., 2001). Although childhood SES disadvantage is an antecedent of several diseases in later life, this study found indirect effects, rather than direct effects, for osteoarthritis and rheumatoid arthritis through higher BMI in adulthood.

An indirect association with no statistically significant direct association between childhood exposures and later-life health is conceptually consistent within the life-course framework which posits that early-life experiences may have enduring consequences by affecting subsequent transitions (Elder, 1998). These pathways may be described by the chains of risk model whereby exposures are linked and lead to subsequent exposures (Kuh et al., 2003). Moreover, given that childhood socioeconomic disadvantage is indirectly associated with health, we can draw from fundamental cause theory, which describes the effects of SES on a wide range of risk factors and outcomes (Link & Phelan, 1995). Childhood exposure to the chronic stress of socioeconomic disadvantage may lead to heightened risk of arthritis via biological mechanisms such as *predicted adaptive responses* that heighten immune response and inflammation, leading to dysregulation of the HPA axis as outlined by the biological embedding model (Miller et al., 2011). Taken together, childhood SES influences resources available to prevent disease and risk factors associated with developing disease through multiple pathways.

The findings herein, along with evidence suggesting that health beliefs and behaviors are developed during early life, suggest that prevention and intervention efforts (i.e., healthy eating and physical activity) should begin early and target those who are socioeconomically disadvantaged (Patrick & Nicklas, 2005). Some evidence indicates that interventions that require parental engagement such as reducing screen time may be effective in reducing obesity among socioeconomically disadvantaged children (Vazquez & Cubbin, 2020). However, benefits of new interventions are often first experienced by those with higher SES, so it has been argued that policies such as mandating nutritional guidelines in childcare settings would perhaps be more ideal (Vazquez & Cubbin, 2020). It is likely that prevention and intervention efforts at multiple levels of action are needed to reduce the negative effects of socioeconomic disadvantage that accumulate across the life course.

This study also found that respondent's *education mediated the relationship between childhood SES disadvantage and osteoarthritis among women*. One and two or more childhood SES disadvantages were associated with fewer years of education, and education was positively associated with osteoarthritis, for an overall negative indirect effect. Even though we adjusted for medical insurance, the surprising association between education and higher odds of osteoarthritis may suggest that women with fewer years of education—especially women who were not socially mobile—may be less likely to get diagnosed with osteoarthritis. Seeing a physician more regularly, which is common among highly educated women, may elevate the likelihood that those women are diagnosed with osteoarthritis. Nonetheless, this finding should be interpreted with caution given that there was no significant difference between men and women, and the effects were not significant for men. It is also possible that the higher odds of osteoarthritis for women are an artifact of the older (50+), at-risk sample. It would be helpful if future studies included younger adults to confirm or refute the relationship between education and osteoarthritis among women identified herein.

In addition to childhood exposures indirectly influencing arthritis, several childhood exposures also were directly related to arthritis among older adults. Even after excluding the individuals with arthritis in 2004, having two or more chronic diseases and at least one infectious disease during childhood predicted osteoarthritis onset for women over the next 10 years (2004–2014). The extent to which childhood health is an exogenous factor is unclear (Blackwell et al., 2001). If chronic or severe, poor childhood health, among other early-life exposures, may alter the structure and function of the developing body (e.g., heightened immune response and pro-inflammatory state and dysregulation of the HPA axis), heightening the risk of arthritis directly, or it may be related to a cascade of negative events and experiences that have cumulative effects on the body over time. In a study examining several childhood exposures and adult health outcomes, Blackwell and colleagues (2001) reported that non-infectious diseases during childhood are associated with arthritis and/or rheumatism among older adults, however, the *p*-value they used to determine significance in a sample of 654 was 0.10. By using a larger sample and a more conservative criterion level, the present study strengthens these earlier findings.

Among men, experiencing an impairment during childhood or risky adolescent behavior predicted osteoarthritis incidence. Supplementary analyses revealed that head injury and

trouble with the police were the indicators driving the associations for the impairment and risky adolescent domains, respectively. It may be that childhood head injuries were linked to sports activity for men, which is a risk factor for osteoarthritis (Vigdorchik et al., 2017). It is also plausible that risky behavior by adolescents (i.e., trouble with the police) increases stress, altering the HPA axis as suggested by Fuller-Thomson et al. (2009). Unfortunately, the HRS does not provide more detail about these early-life events and experiences.

Our finding that *risky adolescent behaviors are associated with higher hazard odds of rheumatoid arthritis for women*, but not men, is consistent with evidence from prior studies suggesting that emotional abuse and neglect increase women's odds of rheumatoid arthritis (respectively, Bayram & Erol, 2014; Spitzer et al., 2013). Emotional neglect and abuse are not probed in the HRS. However, the measures that make up the risky adolescent behavior domain comprise internalizing and externalizing behaviors, suggestive of an adolescent attempting to deal with these types of adversities. Some authors propose that the HPA axis may be a pathway by which childhood exposures influence arthritis (osteoarthritis, Fuller-Thomson et al., 2009; rheumatoid arthritis, Spitzer et al., 2013), whereas others suggest that negative childhood exposures may lead to unhealthy coping behaviors associated with rheumatoid arthritis (Bayram & Erol, 2014). In any case, this finding should be interpreted with caution given that the association did not reach statistical significance in all sensitivity analyses.

Several types of childhood exposures were related to osteoarthritis directly, whereas only one type was associated with rheumatoid arthritis and for women only. This may be due to the differing etiologies. Osteoarthritis is a degenerative joint disease most notable for affecting cartilage whereas rheumatoid arthritis, also a degenerative disease, is immunological. Perhaps the childhood exposures examined in this study influence the accumulation of minor mechanical shocks—routine daily activities that are associated with repetitive movement or stress on joints, and that are not easily operationalizable. However, altering the body's biological processes may require a more severe shock. This study examined direct and indirect associations with early-life exposures and osteoarthritis through a social and behavioral lens; the pathogenesis of rheumatoid arthritis may merit greater attention through a biological lens.

One somewhat unexpected finding was that for men and women, manual occupation was not associated with osteoarthritis. In comparing characteristics of those who had arthritis at baseline in 2004 to those in our analytic sample (no known arthritis at baseline) with ANOVA and χ^2 tests, we found that more respondents had manual occupations at baseline compared to those who did not have arthritis at baseline. Thus, perhaps manual occupations are associated with earlier onset arthritis.

There are some limitations of this study that must be recognized. First, retrospective childhood measures are used, so there is the potential of recall bias. Vuolo and colleagues (2014) investigated the issue of older adults changing responses to childhood questions and recommend controlling for adult memory, psychological issues, self-rated health, and SES in retrospective studies. Other studies that examine the reliability and validity of adult retrospective reports of childhood conditions show that under-reporting adversities is more

common than over-reporting (Hardt & Rutter, 2004). This scenario would likely lead to underestimating the relationship between childhood exposures and adult arthritis; however, caution is warranted in interpreting results. To address potential recall bias, the present study adjusted analyses for SES and excluded individuals who had low cognition scores at baseline. Second, the HRS offers a good deal of information on childhood health, yet very little about childhood abuse. Several researchers have found that abuse during childhood is linked to adult arthritis (Bayram & Erol, 2014; Fuller-Thomson et al., 2009; Spitzer et al., 2013). Although the risky parental behavior domain included one global measure of physical abuse, there were no measures of other forms of abuse or neglect.

Third, similar to other U.S. population health surveys, the HRS measures arthritis based on respondents' self-report of physician diagnoses. There are no additional measures to further classify arthritis based on common classification criteria. Therefore, it is likely that some respondents mis-classified their diagnosis, and this could potentially explain the relatively high proportion of the sample who developed rheumatoid arthritis over the study period.

Despite these limitations, this research offers important insight into the early origins of adult health literature. Few studies examine osteoarthritis and rheumatoid arthritis as separate diseases or investigate potential pathways by which early-life exposures influence the development of osteoarthritis and rheumatoid arthritis. Findings from this study indicate that early-life exposures are associated with an increased risk of osteoarthritis and rheumatoid arthritis decades later.

This study found that experiencing SES disadvantage during childhood is a risk factor for adult-onset arthritis via the pathway of higher BMI during adulthood. Given the strong link between negative childhood exposures and adult obesity, a life-course approach could help strengthen intervention strategies by targeting children in at-risk conditions. Our results suggest that maintaining a healthy BMI during adulthood may help prevent arthritis, especially for people from socioeconomically disadvantaged backgrounds. In addition, several childhood exposures were directly associated with osteoarthritis and rheumatoid arthritis development. Reducing disease burden early in life and directing efforts to modify risky adolescent behavior could potentially have long-term health benefits. The findings herein challenge us to shift our focus from mid-life interventions to prevention across the life course in order to reduce arthritis onset during later life.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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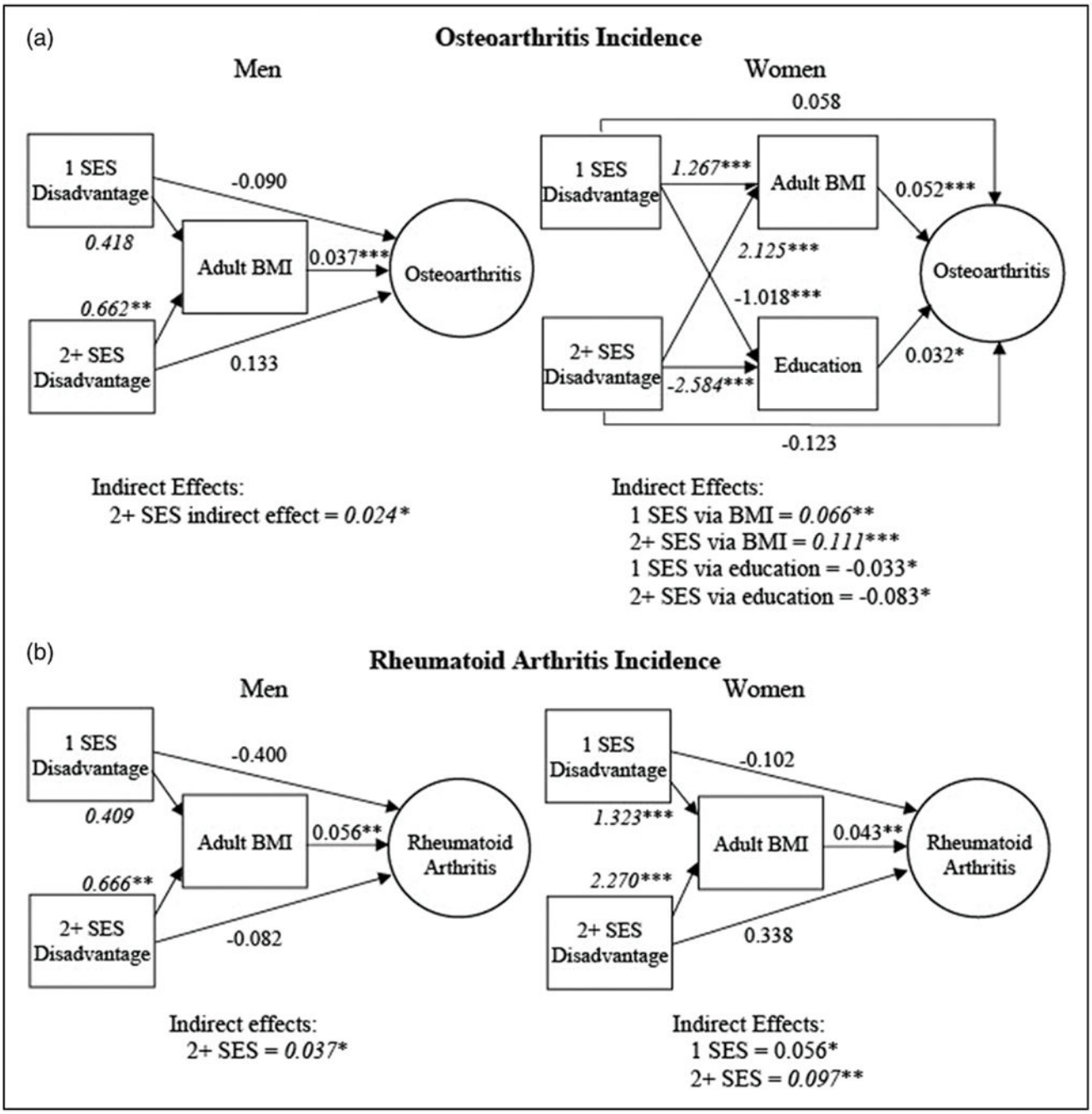

Figure 1.

Mediational Effects of Adult BMI on Childhood SES Disadvantage and Arthritis Incidence. (a). Direct and Indirect Effects of Childhood SES Disadvantage on Osteoarthritis Incidence among Men and Women. (b). Direct and Indirect Effects of Childhood SES Disadvantage on Rheumatoid Arthritis Incidence among Men and Women. Note. Unstandardized coefficients are shown; based on models fully adjusted for covariates listed in Tables 2 and 3; no SES disadvantage is the reference group; italics indicate a significant difference between men and women at the 0.05 level of significance (Wald χ^2); rectangles represent observed variables

and circles represent the latent propensity of event occurrence (these simplified models do not show the individual survival indicators). *p<.05; **p<.01; p***<.001. Note. BMI = body mass index; SES = socioeconomic status.

Table 1.

Descriptive Statistics from the Health and Retirement Study, 2004–2014.

	Men (n = 2720)		Women (n = 2974)	
	Range	**Mean (S.D.[a])**	**Range**	**Mean (S.D.)**
Osteoarthritis incidence	0, 1	0.247[b]	0, 1	0.324
Rheumatoid arthritis incidence	0, 1	0.078	0, 1	0.070
Race/Ethnicity				
Non-Hispanic Black	0, 1	0.119	0, 1	0.136
Non-Hispanic White	0, 1	0.779	0, 1	0.762
Hispanic	0, 1	0.101	0, 1	0.102
Age (years)	50–95	63.390 (9.270)	50–95	63.074 (9.567)
Childhood exposures				
SES disadvantage[c]	0–2	1.239 (0.832)	0–2	1.1758 (0.839)
Risky parental behavior	0–2	0.892 (0.654)	0–2	0.865 (0.668)
Chronic diseases	0–2	0.382 (0.633)	0–2	0.438 (0.692)
Infectious diseases	0–2	1.670 (0.646)	0–2	1.755 (0.536)
Impairments	0–2	0.213 (0.463)	0–2	0.156 (0.411)
Risky adolescent behaviors	0, 1	0.121	0, 1	0.043
Adult risks and resources				
Education (years)	0–17	13.270 (3.269)	0–17	12.933 (2.847)
Wealth[d]	−13.095–31.224	6.391 (3.774)	−7.453–31.224	6.087 (3.499)
Manual occupation	0, 1	0.468	0, 1	0.230
Private health insurance	0, 1	0.466	0, 1	0.499
Medicaid only	0, 1	0.009	0, 1	0.008
Medicare only	0, 1	0.441	0, 1	0.401
No insurance	0, 1	0.084	0, 1	0.092
Married	0, 1	0.812	0, 1	0.629
Divorced	0, 1	0.103	0, 1	0.149
Widowed	0, 1	0.046	0, 1	0.188
Never married	0, 1	0.039	0, 1	0.034

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	Men (n = 2720)		Women (n = 2974)	
	Range	**Mean (S.D.[a])**	**Range**	**Mean (S.D.)**
Body mass index (kg/m^2)	17.2–57.4	27.519 (4.388)	14.7–55.5	26.577 (5.397)
Smoking (pack-years)	0–205	18.603 (26.762)	0–152	9.594 (17.798)
Heavy drinking	0, 1	0.050	0, 1	0.017
Physical activity	0, 1	0.391	0, 1	0.326
Depressive symptoms	0–8	0.869 (1.512)	0–8	1.134 (1.782)

[a] S.D. = standard deviation.

[b] italics indicate a significant chi-square, t-test, or analysis of variance (ANOVA) test in difference between men and women at the 0.05 level of significance.

[c] SES is socioeconomic status.

[d] cube root of wealth in $1,000s.

Table 2.

Structural Equation Modeling-Based Discrete-Time Survival Models of Childhood Exposures Predicting Osteoarthritis Incidence among Men (n = 2720) and Women (n = 2974), 2004–2014.

	Men		Women	
	h.O.R[a]	95% C.I[b]	h.O.R	95% C.I
Age (years)	1.014	0.999–1.029	1.015 *	1.001–1.028
Race/Ethnicity (ref. = Non-Hispanic White)				
Non-Hispanic Black	0.779	0.605–1.003	0.676 **	0.529–0.862
Hispanic	0.797	0.588–1.080	0.859	0.670–1.101
Childhood exposures (ref. = 0 exposure[c])				
1 SES disadvantage[d]	0.914	0.691–1.209	1.058	0.867–1.275
2+ SES disadvantages	1.143	0.874–1.469	0.878	0.704–1.096
1 risky parental behavior	0.956	0.806–1.134	1.065	0.881–1.288
2+ risky parental behaviors	1.040	0.811–1.332	1.215	0.964–1.533
1 chronic disease	1.217	0.665–1.553	1.122	0.899–1.401
2+ chronic diseases	1.194	0.824–1.728	1.328 *	1.045–1.687
1 infectious disease	0.798	0.596–1.067	1.548 *	1.047–2.289
2+ infectious diseases	1.185	0.874–1.608	1.401	0.925–2.121
1 impairment	1.392 **	1.092–1.775	1.020	0.843–1.234
2+ impairments	1.578	0.980–2.537	1.097	0.589–2.042
1+ risky adolescent behavior	1.384 *	1.026–1.866	1.411 *	1.006–1.980
Adult risks and resources				
Education (years)	0.998	0.961–1.037	1.034 *	1.005–1.064
Wealth[e]	0.994	0.969–1.020	1.008	0.983–1.033
Manual occupation	0.971	0.770–1.213	1.025	0.831–1.266
Body mass index (kg/m2)	1.038 ***	1.021–1.053	1.053 ***	1.037–1.069
Pack-years	1.001	0.998–1.005	1.000	0.996–1.004
Heavy drinking	1.419 *	1.028–1.958	1.349	0.786–2.314
Physical activity	1.082	0.938–1.247	1.077	0.946–1.226

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	Men		Women	
	h.O.R[a]	**95% C.I**[b]	**h.O.R**	**95% C.I**
Depressive symptoms	1.016	0.958–1.078	1.080***	1.036.125
R^2	0.043		0.062	
Log-likelihood	−61,448.8		−64,823.9	
BIC	126,606.6		133,398.7	

Note. Italics indicate a significant difference between men and women at the 0.05 level of significance (Wald χ^2). Models also adjust for health insurance status and marital status; full tables are available in the online Supplementary Material.

*$p < .05$;

**$p < .01$;

***$p <. 001$.

[a]h.O.R. is hazard odds ratio.

[b]C.I. is confidence interval.

[c]The reference category for each domain is experiencing no exposure in that domain, coded as 0.

[d]SES is socioeconomic status.

[e]cube root of wealth in $1,000s.

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Table 3.

Structural Equation Modeling-Based Discrete-Time Survival Models of Childhood Exposures Predicting Rheumatoid Arthritis Incidence among Men (n = 2720) and Women (n = 2974), 2004–2014.

	Men		Women	
	h.O.R[a]	95% C.I[b]	h.O.R.	95% C.I.
Age (years)	1.028 *	1.006–1.050	1.014	0.986–1.043
Race/Ethnicity (ref. = Non-Hispanic White)				
Non-Hispanic Black	1.467	0.972–2.212	2.030 **	1.313–3.136
Hispanic	1.446	0.937–2.232	1.471	0.864–2.507
Childhood exposures (ref. = 0 exposure[c])				
1 SES disadvantage[d]	0.670	0.391–1.148	0.890	0.481–1.647
2+ SES disadvantages	0.921	0.609–1.394	1.365	0.733–2.542
1 risky parental behavior	0.914	0.628–1.332	0.745	0.530–1.046
2+ risky parental behaviors	0.995	0.652–1.519	0.968	0.628–1.490
1 chronic disease	1.102	0.800–1.517	1.119	0.766–1.634
2+ chronic diseases	1.319	0.777–2.237	1.076	0.717–1.613
1 infectious disease	1.170	0.645–2.125	1.261	0.569–2.790
2+ infectious diseases	1.292	0.784–2.125	1.339	0.638–2.809
1 impairment	0.891	0.567–1.402	1.234	0.788–1.929
2+ impairments	0.847	0.348–2.059	0.751	0.252–2.239
1+ risky adolescent behavior	*0.737*	0.415–1.309	1.883 *	1.016–3.490
Adult risks and resources				
Education (years)	0.988	0.933–1.045	0.998	0.931–1.071
Wealth[e]	0.968	0.931–1.003	0.965	0.914–1.017
Manual occupation	0.851	0.592–1.226	1.052	0.689–1.606
Body mass index (kg/m2)	1.058 **	1.021–1.095	1.046 **	1.016–1.076
Pack-years	0.997	0.991–1.003	1.001	0.992–1.009
Heavy drinking	0.891	0.465–1.711	2.467 *	1.137–5.355
Physical activity	0.955	0.752–1.212	0.391	0.645–1.284

	Men		Women	
	h.O.R[a]	**95% C.I**[b]	**h.O.R.**	**95% C.I.**
Depressive symptoms	1.084	0.995–1.182	1.087 *	1.018–1.160
R^2	0.074		0.114	
Log-likelihood	−60,041.7		−62,691.4	
BIC	123,792.5		129,133.7	

Note. Italics indicate a significant difference between men and women at the 0.05 level of significance (Wald χ^2). Models also adjust for health insurance status and marital status; full tables are available in the online Supplementary Material.

[a] h.O.R. is hazard odds ratio.

[b] C.I. is confidence interval.

[c] The reference category for each domain is experiencing no exposure in that domain, coded as 0.

[d] SES is socioeconomic status.

[e] cube root of wealth in $1,000s.

* $p < .05$;

** $p < .01$;

*** $p < .001$.