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Early childhood social disadvantage is associated with poor health behaviours in adulthood

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Abstract

Background—Individual health behaviours are considered important risk factors for cardiometabolic diseases. These behaviours may be socially patterned by early exposure to social disadvantage, but few studies have prospectively tested this hypothesis empirically.

Aim—We investigated whether childhood social disadvantage was associated with likelihood of engaging in less healthy behaviours 40 years later.

Subjects and Methods—Prospective data were analysed from the New England Family Study, a 2005–2007 adult follow-up of a cohort initiated in 1959–1966 ($n=565$). Childhood social environment (birth-age 7) was assessed using a cumulative index of socioeconomic and family stability factors. Logistic regression models evaluated associations between social disadvantage and each health-related behaviour and obesity in adulthood.

Results—Relative to low disadvantage, higher disadvantage was associated with 3.6-fold greater odds of smoking (95% CI: 1.9, 7.0), 4.8-fold greater odds (in women only) of excess alcohol consumption (95% CI: 1.6, 14.2), and 2.7-fold greater odds of obesity (95% CI: 1.3, 5.5), but was not associated with unhealthy diet or physical inactivity.

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Declaration of Interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of this article.

Conclusion—These findings suggest childhood social disadvantage may contribute to adult cardiometabolic disease by predisposing children to adopt certain unhealthy behaviours. If replicated, such findings may support intervention strategies that target social environmental factors and behavioural pathways that are established early in life.

Key Terms

Early life adversity; childhood socioeconomic status; body mass index; smoking; alcohol

Introduction

Behaviours such as smoking, unhealthy diet, and physical inactivity, are well known risk factors for cardiometabolic diseases, such as obesity, heart disease, and diabetes (Buttar et al., 2005, Franks, 2012). Excess alcohol consumption and smoking, after high blood pressure, are currently the leading contributors to the global burden of disease from young to mid adulthood (Lim et al., 2012). Because those with low socioeconomic status (SES) disproportionately engage in these harmful health behaviours (Stringhini et al., 2010), the blame for health disparities is frequently placed on poor health choices made by low-income individuals. However, many of these harmful health behaviours are likely not solely a result of individual choices arising *de novo* in adulthood, but in fact may be patterned by poverty and other related disadvantages in the early social environment.

A growing body of evidence supports the hypothesis that adverse early childhood experiences are likely to be associated with health risk behaviours in adulthood. Studies have considered childhood adversity with a range of behaviour-related outcomes including smoking, excess alcohol consumption, and obesity (Bellis et al., 2014, Boynton-Jarrett et al., 2012, Dube et al., 2010, Vander Weg, 2011, Davis et al., 2014). For example, in a large retrospective study in San Diego ($n=9215$), persons with four or more types of adverse childhood experiences, including various forms of abuse and household dysfunction, had higher risks for smoking, alcoholism, physical inactivity, and severe obesity in adulthood (Felitti et al., 1998). In a study of older women in Britain ($n=3523$), retrospectively assessed lower childhood SES was associated with lower fruit and vegetable consumption and lower physical activity levels, independent of adult SES (Watt et al., 2009). However, a major critique of these studies has been the use of retrospective data to examine the influence of the early environment; findings of these studies are susceptible to concerns about recall bias, such that individuals who are less healthy in adulthood are likely to recall greater adversity during childhood regardless of actual adversity level.

Among the four prospective studies that have examined associations of early adversity with adult health risk behaviours, measures of the early environment were narrowly defined. Many studies have focused solely on the role of low SES (as defined by income, parental educational status or occupation) in childhood in relation to adult health. For example, one study found that young adults who had low relative to high family SES as children had higher rates of cigarette smoking, but lower rates of alcohol or marijuana use (Patrick et al., 2012). A prospective cohort study demonstrated cumulative effects of low SES in childhood and in adulthood on smoking behaviours throughout the lifecourse (Gilman et al., 2003). In

the 1958 British Birth cohort study, childhood financial adversity was linked to a greater likelihood of heavy adult smoking and poor lung function in a large sample of 6,287 (Bartley et al., 2012). Additionally, a systematic review of 11 prospective population-based studies reported weak and inconsistent associations between lower childhood SES and excess alcohol use in adulthood (Wiles et al., 2007).

Other studies have focused on severe adversity, primarily on the role of physical, verbal, or sexual abuse in childhood, and the subsequent risk of obesity and poor health behaviours in adulthood (e.g. Boynton-Jarrett et al., 2012). These types of severe adverse experiences tend to be rarer than other forms of adversity such as parental divorce and low SES; therefore studies of their long-term effects raise the question of whether the effects of adversity on subsequent health-related behaviours are primarily due to experiencing the most severe forms of adversity or if effects may be evident across the range of adversity exposures.

Measuring the accumulation of multiple risk factors in the childhood environment is critical for capturing the full range of experiences that can influence a child's current and future health (Evans and Kim, 2010). Only a handful of prospective studies have considered a wider constellation of socioeconomic and family stability factors in the assessment of the child's total environment. In one study of rural New York children, researchers found that a cumulative index measuring SES (e.g. poverty, single parenthood, maternal education), physical risks (e.g. residential crowding, noise, and housing problems), and psychosocial stressors (e.g. family turmoil, family separation, exposure to violence) was associated with weight gain in adolescence (Evans et al., 2012). Another study using the 1958 British Birth Cohort examined a range of childhood adversities and found low parental aspirations, little parental interest in education, and 'hardly takes outings with father' were significantly associated with body mass index (BMI) and central obesity at age 45 (Thomas et al., 2008). However, no prospective studies to date have comprehensively considered effects of a range of adverse social exposures in childhood in relation to the set of behaviours and behaviour-related outcomes that comprise a healthy lifestyle in adulthood (Chiuve et al., 2014).

Prior research suggests that men and women may deal with stress differently, and thus may respond to early life adversity in different ways. Each sex may be socialized to have different stress coping strategies. For example, some studies report that although men engage in a number of maladaptive coping behaviours more frequently than women, such as consuming alcohol and unhealthy eating, they are also more likely to exercise in response to stress (Lindquist et al., 1997), while women tend to use social support and help-seeking behaviours (Tamres et al., 2002). Further, some studies have noted higher rates of riskier health behaviours among men relative to women (Courtenay et al., 2002). While few studies have examined this question explicitly, it may be that early life adversity contributes to this disparity, shaping men and women's health behaviours differently.

While the studies reviewed above demonstrate emerging evidence for associations between early life environments and adult health behaviours, more research is needed to discover the pathways through which these variables are linked. One likely pathway is through adult SES (Luo and Waite, 2005). Disadvantages early in life may set children on a path towards lower SES in adulthood, which may constrain opportunities for a healthy lifestyle (e.g. limited

material resources for purchasing healthy food or less safe neighborhoods for exercise). Alternatively, childhood disadvantage may have an effect on adult behaviours independent of adult SES, for example by shaping psychological or cognitive development of the children (Hackman et al., 2010), potentially leading them to make riskier health choices throughout their lives.

In earlier work using data from the New England Family Study, we created a multidimensional measure of childhood social disadvantage designed to capture a range of adverse early life experiences, including aspects of family stability and childhood SES (Non et al., 2014). Findings in the initial study suggested that early adversity was associated with higher cardiometabolic risk and greater likelihood of having a chronic disease (Non et al., 2014). We speculated that these associations might be due in part to the more harmful health behaviours practiced by those with high childhood social disadvantage.

In the current study, we use prospective data from participants in the New England Family Study to test the hypothesis that early childhood exposure to high levels of social disadvantage would be associated with a higher likelihood of engaging in unhealthy behaviours, including smoking, excess alcohol consumption, poor diet, low levels of physical activity, and a greater risk for obesity. Based on the literature of gendered coping strategies reviewed above, we further hypothesised that these associations may differ between men and women, such that men with high disadvantage may be more likely to smoke, drink, and eat less healthy diets than women. We also investigated the role of adult SES in these associations, as it is of considerable interest to determine if adult SES is on the pathway between childhood disadvantage and adult health behaviours.

Methods

Sample Population

This study analysed data from offspring of participants enrolled in the New England (Boston and Providence) sites of the Collaborative Perinatal Project (CPP). Figure 1 displays a flow chart describing how the study sample was selected. The CPP was a nation-wide longitudinal study of children born to women pregnant in 1959–1966, initially conducted to study neurological and neurosensory defects and the relationship of pregnancy and perinatal factors with subsequent child health (Niswander and Gordon, 1972). Detailed prospective data were collected from mothers on their medical history and the social environment of the child at the time of enrollment and again when children were seven years old. The New England Family Study (NEFS) comprises a series of follow-up studies of the Boston and Providence CPP offspring in adulthood. Participants in the current analysis were identified through their participation in the Brown-Harvard Transdisciplinary Tobacco Use Research Center (TTURC, $n=1,674$) between 2005–2007 (Gilman et al., 2008) and its subsequent reinterview study on educational attainment and health (“EdHealth”) that included assessments of cardiovascular biomarkers and risk behaviours (Loucks et al, 2012). Of relevance to the current analysis is the design and participation rate of EdHealth, which sought to enroll 914 TTURC participants with representation across levels of education and from all racial/ethnic groups in the cohort, of which 898 were eligible (i.e. living and not incarcerated) and invited to participate, and in which 618 participated (69% response rate)

(Loucks et al., 2012). At the adult follow-up, in their early forties, the 618 participants of EdHealth completed a 3-hour in-person interview that included detailed assessment of adult SES, psychological functioning, behaviours, literacy, and physical health among other factors.

For this current study, we excluded participants missing data on childhood social disadvantage factors ($n=49$) or missing data on demographic covariates ($n=7$), resulting in a final sample size of $n=564$ with complete data on childhood social disadvantage. The 54 participants missing critical childhood data did not differ meaningfully from those without any missing data in regards to age, sex, ethnicity, smoking, alcohol consumption, physical activity, diet, obesity, or BMI. Complete case analysis was performed for each health outcome, resulting in varied sample sizes across the analyses: smoking ($n=555$), excess drinking ($n=450$), unhealthy diet ($n=542$), exercise ($n=392$), and obesity/BMI ($n=506$). The final analytic sample includes some siblings, which cluster into 323 to 423 families, depending on the health outcome analysed. Participants in the study self-identified as “white,” “black,” “Asian,” “Pacific Islander,” or “Native American,” which for our analyses were categorised as “white” or “non-white.” These self-designated categories will be used throughout the manuscript because they were used in the initial survey that was not conducted by the authors of this paper. Institutional review approval was obtained from the Harvard School of Public Health (Boston, MA) and Brown University (Providence, RI).

Childhood Social Disadvantage

We constructed an index of childhood social disadvantage based on 10 family stability and socioeconomic exposures assessed via questionnaires given to participants’ mothers between pregnancy and age 7 years. This index was constructed as a sum of these 10 binary (present/absent) factors measured. If any of the disadvantage factors occurred at either time point, the factor was counted as one point in the social disadvantage index. The family stability factors included living away from home for more than 3 months between birth and age 7, sibling death between birth and age 7, moving more than 3 times between birth and age 7, change in parental marital status between birth and age 7, and whether a single mother was raising the child at age 7. The socioeconomic measures included manual occupation of the main household wage earner either at the participant’s birth or at age 7 years, having a father who was unemployed for more than 6 months in the past year when the participant was 7 years of age, crowded housing density (>1.5 people/ room) at age 7 years, household income less than or equal to the poverty threshold at birth or age 7 years, and highest parental educational level of less than high school at the participant’s birth. The index was then categorised into the following 3 groups: least disadvantaged (0–2 adverse exposures), moderately disadvantaged (3–4 adverse exposures), and most disadvantaged (≥ 5 adverse exposures). Following our earlier work with this sample, to test the two domains within the disadvantage score, we also divided the total score into two separate subscales of family stability and childhood SES, which were each analysed in sensitivity analyses (Non et al., 2014).

Health Behaviours

Each health behaviour was considered separately as a binary variable, defined by a previously defined cutoff point according to whether behaviours meet recommended levels for a healthy cardiovascular lifestyle based on recommendations from the American Heart Association (AHA), and other researchers (e.g. , 2014a, 2015). All health behaviours were self-reported at adult follow-up.

Smoking and Alcohol Consumption—Smoking status was assessed by the question, “Do you smoke cigarettes now?” and those who reported any amount of smoking were classified as current smokers (yes/no). Alcohol consumption was assessed with two questions, “In the past 12 months, how often did you have at least one drink?” and “On days that you drank in the past 12 months, about how many drinks did you usually have per day?” Those who responded that they never drank any alcohol in the past year or ≤ 2 drinks/day were categorised as not heavy drinkers, while those who stated they had at least one drink in the past year, and when they did drink, usually >2 drinks/day, were categorised as heavy drinkers, based on clinical recommendations for healthy levels of alcohol consumption (Bradley et al., 1993, 2015).

Physical Activity—Physical activity was assessed with two questions: “In a usual week, do you do vigorous activities for at least 10 minutes at a time?” Vigorous activities were defined as activities that can cause large increases in breathing or heart rate (e.g. running, walking quickly up a hill, lifting heavy boxes, doing heavy yard work). The same question was asked about moderate activities, which were defined as activities that cause small increases in breathing or heart rate (e.g. carrying groceries or children, working in a garden, walking while pushing a baby stroller). For this study, we summed the minutes of vigorous and moderate physical activities in a usual week, categorised into high physical activity if ≥ 30 min/day, versus inadequate physical activity, if <30 min/day, based on AHA recommended levels of activity to maintain cardiovascular health (2014a).

Diet—Diet was assessed by asking “how often, on average, have you eaten this food during the past year (including foods eaten at home and away from home)?” Following prior work on the Mediterranean and other healthy diet scores, (Chiuve et al., 2014, Fung et al., 2009), healthy foods included in our diet score were: fruits, vegetables, eggs, chicken or turkey, fish, dark bread, and nuts. Unhealthy foods included fried food, ice cream, fruit juice, processed meats (e.g. sausage, salami, bacon), hot dog/hamburger, white bread, beef sandwiches, beef/pork/lamb, non-diet soda, coffee, bakery items (e.g. cookies, brownies, cake), butter, pasta, potatoes (baked, boiled or mashed), and shrimp/lobster/scallops. Responses were chosen from options which we ranked from 0–8: Never or <1 /month, 1–3/month, 1/week, 2–4/week, 5–6/week, and ≥ 6 /week. We summed the frequency ranks for healthy foods and for unhealthy foods and then calculated a total food score (unhealthy foods–healthy foods), following work done in a prior study (Chiuve et al., 2014). Those who scored in the top 40% of this diet score were then classified as healthy diet, and the lower 60% were classified as unhealthy, also following prior studies (Chiuve et al., 2014).

Obesity and BMI—Data on height and weight were collected via in-person anthropometric measurements. BMI was defined as an individual's weight (kg) divided by height (m²). Participants were categorised as obese (yes/no) if BMI ≥ 30, according to the standard cutoff point for obesity defined by the American Heart Association (2014b). We also considered body mass index (BMI) as a continuous measure so as not to lose information in categorizing it into groups.

Adult SES Measures—Two measures of adult SES were obtained, educational attainment and household income. Educational attainment was self-reported as years of education achieved (ranging from 0 to 21, median=12). Household income was assessed via the question, “How much did your household earn, before taxes and other deductions, during the past 12 months? Please include all income from your job(s), your family member's job(s), money from government programs, child support, interest and dividends, money from rental property, etc.” Response options included categories, 1 = “Less than \$5000, 2 = \$5,000–\$11,999, 3 = \$12,000–\$15,999, 4 = \$16,000–\$24,999, 5 = \$25,000–\$34,999, 6 = \$35,000–\$49,999, 7 = \$50,000–\$74,999, 8 = \$75,000–\$99,999, and 9 = \$100,000.

Data analysis—We used logistic regression models to analyse the association between each binary health behaviour assessed in middle-adulthood and the three childhood social disadvantage categories (high, medium, and low, using low as the reference). In minimally adjusted models, we adjusted for age (mean-centered), ethnicity (“white” or “non-white”), sex, and study site (Boston or Providence). A linear regression model was used to examine associations between childhood social disadvantage and (log)BMI. Therefore we present the estimates of the percent increase for the (geometric) mean of BMI for high or medium disadvantage groups relative to the low disadvantage group. In a second set of models (labeled fully adjusted models), adult SES variables of educational attainment and household income were added, as they are hypothesised to be on the pathway from high childhood disadvantage towards worse adult health behaviours. For all outcomes, we consider possible effect modification by sex, and if the interaction was significant, we present the results from the stratified model. To account for relatedness among siblings, we included a random intercept for each family in the linear regression models of log(BMI). However in logistic models with random effects, the procedure used to produce confidence intervals (CIs) was unreliable. Since models with and without random effects yielded similar effect estimates, for the logistic models we present results without the random effects for relatedness. All data analyses were performed using R, v3.2.1.

Results

Among the 564 individuals analysed in this study, the average age at adult follow-up was 42.5 (range 38–47), 60% were women, and 78% identified as “white.” When considering the distribution of childhood social disadvantage across the sample, 69% were classified as low, 22% were classified as moderate, and 9% were classified as high. Characteristics of the sample across all three levels of childhood social disadvantage are shown in Table 1. Those with high relative to low childhood disadvantage were significantly more likely to be ethnic minorities, and as adults, were more likely to have earned less than a high school degree, (or achieved fewer years of education) and more likely to have a lower household income in

adulthood. Based on tests of proportions, they were also significantly more likely to engage in less healthy behaviours, including smoking, and more likely to be obese and have a higher BMI (Figure 2).

Childhood Social Disadvantage and Smoking

Results from logistic regressions of health risk behaviours are presented in Table 2. For participants with the highest level of childhood social disadvantage, there was a 3.61-fold (95% CI: 1.86, 7.03) higher odds of being a current cigarette smoker as adults than those with low childhood social disadvantage. Those with moderate child disadvantage also had a higher (OR=1.99, 95% CI: 1.22, 3.24) odds of smoking. Adjusting for adult SES variables (which were both inversely associated with smoking) attenuated the effect of childhood social disadvantage by 35%, though it remained statistically significant.

Childhood Social Disadvantage and Excess Drinking

There was a significant interaction between sex and childhood social disadvantage in the model predicting excess drinking ($p<0.01$). Stratified models indicate that high childhood disadvantage was associated with 4.80-fold higher odds of excess drinking in females (95% CI: 1.63, 14.15), but was not associated with drinking in males (OR: 0.63, 95%CI: 0.16, 2.30) (Table 2). In fully adjusted models, both adult SES variables were significantly associated with excess drinking but after adjustment for adult SES, the effect size of high childhood disadvantage on excess drinking among females was virtually unchanged (i.e., a 6% attenuation).

Childhood Social Disadvantage and Unhealthy Diet

Unhealthy diet in adulthood was not related to childhood disadvantage (Table 2). Adult SES variables were associated with unhealthy diet in the expected direction, but adding them to the model with childhood disadvantage did not uncover an association between childhood disadvantage and unhealthy diet.

Childhood Social Disadvantage and Inadequate Physical Activity

There was no association between childhood social disadvantage and physical activity below recommended levels in adulthood. Only ethnicity was significantly associated with this variable, such that those who were “non-white” relative to “white” were twice as likely to have inadequate physical activity. In fully adjusted models, higher educational attainment was significantly associated with inadequate physical activity (OR: 1.14, 95% CI: 1.02, 1.28) (Table 2), which was largely driven by the fact that men with low education had more active jobs in this dataset.

Childhood Social Disadvantage, Obesity, and BMI

There was a 2.68-fold higher odds of obesity (95% CI: 1.34, 5.45) among participants in the highest level of childhood social disadvantage. This association remained significant in models adjusted for adult SES (Table 2). We then analysed BMI as a continuous variable. Relative to the (geometric mean) BMI of those in the low childhood disadvantage category, the BMI estimated for those who experienced high childhood disadvantage was 16% higher

(95% CI: 1.07, 1.26) and the BMI for those who experienced moderate disadvantage was 5% higher (95% CI: 0.99, 1.10). In fully adjusted models, adult SES variables were not significantly associated with BMI, but the effect of high childhood social disadvantage was essentially unchanged. We also tested the effect of adding BMI at age 7 to the minimally adjusted model, and found it to be associated with a 5% increase in adult BMI (95% CI: 1.04, 1.07), but the effect of childhood disadvantage on adult BMI remained unchanged, suggesting that the association between childhood disadvantage on adult BMI could not be explained by differential growth patterns according to disadvantage during childhood.

Sensitivity Analyses

When we analysed the subscales of the childhood disadvantage index, both family stability (FS) and childhood SES (CSES) were associated with smoking (FS OR: 1.23, 95% CI: 1.00, 1.50; CSES OR: 1.50, 95% CI: 1.26, 1.78), excess drinking (in females only) (FS OR: 1.54, 95% CI: 1.10, 2.17; CSES OR: 1.34, 95% CI: 1.02, 1.75), obesity (FS OR: 1.31, 95% CI: 1.07, 1.61; CSES OR: 1.18, 95% CI: 1.00, 1.40), and BMI (FS OR 1.04, 95% CI: 1.01, 1.06; CSES OR: 1.03, 95% CI: 1.01, 1.05). Neither subscale was associated with unhealthy diet or inadequate physical activity. Interactions between ethnicity and childhood disadvantage were also tested, but no significant interactions with ethnicity were identified in any models.

In order to distinguish if the children's health behaviours were influenced by parental behaviours, rather than just disadvantage itself, we also tested models of excess drinking that included a retrospectively reported measure of family drinking behaviour, which represented if a family member (including mother, father, sibling, or child) ever had a period where he/she drank a lot (yes/no). Family drinking behaviour was significantly associated with excess drinking (OR: 2.71, 95% CI: 1.69, 4.46, $p < 0.001$), but the effect of a high level of childhood disadvantage was unchanged.

Discussion

In this prospective study following participants from birth through to middle adulthood, our results indicate that high levels of childhood social disadvantage are associated with worse profiles of adult health-related behaviours more than 40 years later, including cigarette smoking and excess drinking, but not with unhealthy diet or inadequate physical activity. We also found associations with obesity and higher BMI, which are partly a product of these health-related behaviours. Significant associations persisted after accounting for individual characteristics (age, sex, ethnicity, study site), and also after accounting for adult attained SES, which attenuated associations somewhat in models predicting smoking and drinking behaviour. These results support the hypothesis that early life social disadvantage predisposes children to adopt some unhealthy behaviours in adulthood and to have a higher BMI, over and above the influence of adulthood SES.

Our findings are consistent with a cumulative risk model for health, which suggests that exposure to multiple stressors increases the likelihood of harmful effects on child behaviour and other health outcomes (Appleyard et al., 2005, Forehand et al., 1998). Our findings extend this model to suggest that cumulative risk in childhood can also have far-reaching

consequences on health behaviours into adulthood. In particular, our use of an additive index allows for a more comprehensive assessment of early life social disadvantage than prior studies which have been limited by their assessment to individual factors, such as childhood SES or child abuse (Patrick 2012, Boynton Jarrett 2012). While examination of individual risk factors can be useful to identify components of the total environment in order to target interventions, an additive formula for social disadvantage has the benefit of demonstrating the cumulative contribution of each additional risk factor (Evans et al 2010, Appleyard 2005). According to cumulative risk theory, it is the convergence of risk factors more than any one particular risk that causes dysfunction by overwhelming one's adaptive abilities (Flouri and Kallis, 2007). There are also methodological benefits to using a cumulative measure of disadvantage, including that aggregate measures a) are more stable than any individual variable; b) capture the natural covariance of risk factors; and c) increase power to detect effects due to reduced measurement error and preserved degrees of freedom (Burchinal et al., 2000, Flouri and Kallis, 2007).

Results of our study align with the few existing prospective studies that examine relationships between adult health behaviours and early childhood environments, though prior studies primarily focused on childhood SES. For example, our finding that higher childhood disadvantage was significantly associated with adult smoking is similar to the findings of two prior prospective studies in which children with lower childhood SES had higher risk of adult smoking (Bartley et al., 2012, Patrick et al., 2012).

Associations we identified with alcohol consumption add to an existing and often conflicted literature on childhood SES and drinking (Wiles et al., 2007). While some studies in the Dunedin birth cohort in New Zealand indicated that low childhood SES is associated with more alcohol consumption or dependence later in life (Droomers et al., 2003, Melchior et al., 2007), other studies in Finland and the US showed high early life SES to be associated with higher alcohol consumption in young adulthood (Pietila et al., 1995, Patrick et al., 2012). In women, we found high childhood disadvantage was significantly associated with higher likelihood of excess drinking, but a non-significant result of lower likelihood of excess drinking among men with high disadvantage. Our study is not the first to identify differences across sexes, though others have found different sex-specific effects. In the British birth cohort, men with fathers of low social class at time of birth were more likely to be excess users of alcohol at age 33, though no associations were identified for women (Power and Matthews, 1997). Differences across these studies could result from different approaches to measuring childhood disadvantage and alcohol consumption as well as differences in length of follow-up. It may be that childhood social disadvantage is only one of many interactive factors, such as genetics and neighborhood environments, that contribute to determining adult levels of alcohol consumption and a greater understanding of the web of contributing factors is needed.

We did not find significant associations with unhealthy diet or inadequate physical activity in our sample. One possibility is that participants may have over reported the frequency of healthy eating and physical activity, which is often observed in self-report measures related to healthy lifestyle (Ferrari et al., 2002). Over reporting healthy behaviours on these measures could lead to more inaccuracy than in measures of current cigarette smoking,

which did not require recall of frequency, or in the measure of BMI, which was objectively obtained. The possibility of over reporting is also supported by the fact that these diet and exercise measures were not associated with cardiometabolic risk factors or chronic disease in our earlier work with the New England Family Study (Non et al., 2014) despite the fact that these are well-established risk factors for cardiometabolic disease (Lim et al., 2012). These measures may be analysed more extensively in future studies that use exercise monitoring or food logging devices, with lower risk for recall bias or false reporting.

Our significant findings of associations of childhood disadvantage with obesity and adult BMI are consistent with findings from the 1958 British Birth Cohort which demonstrated a number of individual child adversities, such as physical or emotional neglect, to be associated with higher BMI and central obesity in mid-adulthood (Thomas et al., 2008), but they did not examine a cumulative measure of adversity. Our results may be more comparable to the one study that showed a comprehensive measure of childhood social exposures to be associated with weight gain in adolescence (Evans et al., 2012). We show a similar result of increased BMI, but our study extends this finding over 4 decades later into adulthood.

Our findings do not strongly support the hypothesis that early adversity contributes to health behaviours differently in men versus women, as no significant sex interactions were found except in the model of excess drinking. It is likely that our sample size is not sufficient to adequately address this question, due to the low number of men in the high childhood social disadvantage category ($n=18$). While men have higher rates of worse health behaviours than women, further research is needed to determine if adverse early life exposures associate with male and female health behaviours differently.

Potential Pathways

One possible pathway through which childhood social disadvantage could affect adult health behaviours is by shaping adult SES. Educational attainment and household income were both significantly inversely associated with all of the poor health behaviours in our dataset (with the exception of physical activity). In models of smoking and drinking, adult SES factors attenuated, but did not remove the significant effect of childhood disadvantage. Thus associations of childhood disadvantage with adult health behaviours are partly, though not entirely, accounted for by a pathway of adult SES.

Another potential explanation for the observed associations comes from the evolutionary perspective of the life history tradeoff theory, which posits that lifetime strategies to prioritise current risks over delayed rewards, may be shaped by resource scarcity and unpredictability in childhood (Griskevicius et al., 2011). Specifically, children with high social disadvantage may be receiving cues about their expected longevity early in life, shaping a riskier strategy that values the rewards of the present over long term planning for the future. Additionally, recent experimental studies have suggested a link between poverty and cognitive ability, potentially because the cognitive or self-regulatory resources of the poor may be consumed by the mental burden of poverty (Mani et al., 2013). In our sample, as in many other studies (Turkheimer et al., 2003, Marcus Jenkins et al., 2013), children with higher social disadvantage also have a lower childhood IQ, supporting the idea that the

mental burden of adversity may begin in childhood. Finally, children may simply be adopting cultural or social norms of health behaviours they observe in their families (Savage et al. 2007, Power and Parsons 2000).

A number of limitations of this study are important to consider. A larger sample size may have allowed increased detection of effects, particularly to detect sex interactions, as our sample may have been underpowered to detect associations in men. Additionally, accuracy of health risk behaviours (other than BMI) may be limited by self-reporting, with the potential for overreporting healthy behaviours, and the fact that they were measured only at one time point in mid-adulthood. Generalisability is potentially limited to urban centres in New England in the early 1960s. Finally, the cohort design did not allow for a formal test of mediation by adult SES, given that adult health behaviours were measured at the same time as adult SES. Further, we note that BMI is partly a product of lifestyle variables such as diet and exercise that may affect weight gain, but since these variables were measured simultaneously, we could not provide a model that tests mediation of BMI by these health behaviours. However, our study is strengthened by the use of a prospective population-based birth cohort with extensive and well-characterised measures of the child social environment that are not affected by recall bias. This sample also uniquely allowed for detailed assessment of a wide range of health behaviours in middle adulthood, along with other relevant childhood and adult covariates. Finally, while our study does not include more severe forms of child adversity, such as abuse or neglect, our results support that less severe but more common exposures are relevant to lifetime trajectories of health behaviours.

In conclusion, our findings suggest that childhood social disadvantage may contribute to the establishment of trajectories of unhealthy behaviours including smoking, excess drinking, and the behaviour-related outcomes of obesity and higher BMI. Replication of these findings is essential to determine if results hold in other populations and to establish the proximate pathways that may link these factors. If replicated, these findings support research into prevention or intervention strategies that target social environmental factors and the behavioural pathways that are established relatively early in life.

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Figure 1.
Flow chart of included and excluded study participants.
TTURC=Transdisciplinary Tobacco Use Research Center

Figure 2. Prevalence (%) of health risk behaviours across levels of childhood social disadvantage. P-values represent pairwise tests of proportion across social disadvantage groups.

Table 1

Characteristics of EdHealth sample by childhood social disadvantage (n=564).

Variables	High Social Disadvantage (n=50)	Moderate Social Disadvantage (n=124)	Low Social Disadvantage (n=390)	P-value *
Mean age, years (SD)	42.8 (1.9)	42.2 (1.8)	42.5 (1.8)	0.059
Sex No. (%)				0.894
Female	32 (64%)	75 (60.5%)	237 (60.8%)	
Male	18 (36%)	49 (39.5%)	153 (39.2%)	
Ethnicity No. (%)				<0.001
“White”	30 (60.0%)	76 (61.3%)	334 (85.6%)	
Other	20 (40.0%)	48 (38.7%)	56 (14.4%)	
Education No. (%)				<0.001
No HS/GED Degree	15 (30%)	13 (10.5%)	12 (3.1%)	
At least HS/GED degree	35 (70%)	111 (89.5%)	376 (96.9%)	
Years of education	11.9 (2.2)	12.6 (1.7)	14.1 (2.7)	
Household Income No. (%)				<0.001
<\$25,000	9 (20.0%)	29 (25.0%)	25 (6.6%)	
\$25,000–\$49,999	11 (24.4%)	30 (25.9%)	46 (12.2%)	
\$50,000–\$74,999	10 (22.2%)	28 (24.1%)	84 (22.2%)	
\$75,000–\$99,999	7 (15.6%)	11 (9.5%)	87 (23.0%)	
100,000	8 (17.8%)	18 (15.5%)	136 (36%)	

* P-values derived from chi square test (for categorical variables) or ANOVA (for continuous variables).

Table 2
Odds of poor health behavior in adulthood by childhood social disadvantage levels in total sample

Characteristic	Models					
	Smoking n=555	Excess Drinking n=450	Unhealthy Diet n=542	Inadequate Physical Activity n=392	Obesity n=506	
	OR (95%CI) P-value	OR (95%CI) P-value	OR (95%CI) P-value	OR (95%CI) P-value	OR (95%CI) P-value	
Social Disadvantage (minimally adjusted)						
Low (reference)	1.0	1.0	1.0	1.0	1.0	
Moderate	1.99 (1.22, 3.24)	2.36 ^a (1.06, 5.21)	1.05 (0.66, 1.67)	0.59 (0.25, 1.29)	1.20 (0.74, 1.93)	
High	0.006	0.034	0.242	0.205	0.460	
	3.61 (1.86, 7.03)	4.80 ^a (1.63, 14.15)	1.54 (0.76, 3.24)	1.25 (0.42, 3.29)	2.68 (1.34, 5.45)	
	<0.001	0.004	0.242	0.666	0.006	
Social disadvantage (fully adjusted)						
Low (reference)	1.0	1.0	1.0	1.0	1.0	
Moderate	1.27 (0.72, 2.20)	1.26 ^a (0.48, 3.12)	0.69 (0.41, 1.15)	0.73 (0.29, 1.67)	1.00 (0.60, 1.68)	
High	0.406	0.629	0.157	0.475	0.988	
	2.34 (1.08, 5.03)	4.71 ^a (1.42, 15.71)	1.22 (0.54, 2.88)	1.69 (0.54, 4.73)	2.34 (1.11, 5.04)	
	0.029	0.011	0.632	0.338	0.027	

The minimally adjusted model is adjusted for age, sex, ethnicity, and study site, while the fully adjusted model is additionally adjusted for adult educational attainment and household income. In the models of excess drinking, a sex by social disadvantage interaction was significant.

^aThe OR estimate shown applies to women; for men, the OR estimates for high and moderate disadvantage in the minimally adjusted model are 0.63 (95%CI: 0.16, 2.30, p=0.489), and 0.69 (0.30, 95%CI: 1.54, p=0.371), respectively; for men in fully adjusted models, the OR estimates for high and moderate disadvantage are 0.65 (95%CI: 0.15, 2.85, p=0.565), and 0.42 (95%CI: 0.16, 1.04, p=0.067), respectively.