Childhood Socioeconomic Status and Host Resistance to Infectious Illness in Adulthood

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Objective: Low childhood socioeconomic status (SES) is a risk factor for adult morbidity and mortality primarily attributable to cardiovascular disease. Here, we examine whether childhood SES is associated with adult host resistance to infectious illness, and whether the effect is limited to a critical period of low SES exposure, can be undone by changes in childhood SES, and is explained by adult SES. **Methods:** Three hundred thirty-four healthy volunteers reported their own and their parents' level of education and the ages during their childhood when their parents owned their homes. Volunteers' current home ownership was recorded from real estate records. Subsequently, they were given nasal drops containing 1 of 2 rhinoviruses and were monitored in quarantine for infection and signs/symptoms of a common cold. **Results:** For both viruses, susceptibility to colds decreased with the number of childhood years during which their parents owned their home (odds ratios by tertiles adjusted for demographics, body mass, season, and prechallenge viral-specific immunity were 3.7 for fewest years, 2.6 and 1). This decreased risk was attributable to both lower risk of infection and lower risk of illness in infected subjects. Moreover, those whose parents did not own their home during their early life but did during adolescence were at the same increased risk as those whose parents never owned their home. These associations were independent of parent education level, adult education and home ownership, and personality characteristics. **Conclusions:** A marker of low income and wealth during early childhood is associated with decreased resistance to upper respiratory infections in adulthood. Higher risk is not ameliorated by higher SES during adolescence and is independent of adult SES. **Key words:** socioeconomic factors: child, socioeconomic factors: adult, social class, common cold, respiratory tract infections, disease resistance.

SES = socioeconomic status; **PES** = positive emotional style; **PSS** = Perceived Stress Scale; **CI** = confidence intervals; **OR** = odds ratios.

INTRODUCTION

Socioeconomic status (SES) during childhood as measured by living conditions, family income, and parental education and employment has been repeatedly associated with adult health status (1–4). Lower childhood SES has been reported to be a risk factor for mortality (5–8) primarily resulting from cardiovascular disease (9–11) but also from respiratory disease and stroke, and stomach and lung cancer (9). There is also evidence relating lower childhood SES to increased risk of adult cardiovascular disease (12,13), chronic bronchitis (14), and periodontal disease (13). An unresolved issue is whether there are critical periods during early childhood when SES exposure sets a health trajectory that cannot be undone by later upward or downward mobility (15,16). A related issue is whether the association between childhood SES and adult health occurs because early SES influences adult employment and social position (13). Previously reported associations between childhood SES and health have often (9,13,14,17–19) (but not always (14,20)) been independent of adult SES.

Lower childhood SES might influence adult health through childhood exposures to adverse physical or social environments, malnutrition, high fat diets, substance abuse, poor health practices (21), lack of access to health care, or chronic stress. Many of these factors are consistent with the literature's emphasis on low childhood SES and increased risk of cardiovascular disease and related mortality, but they are also consistent with the argument that childhood SES might influence the development of adult immunity and resistance to infectious disease (22). Infectious agents have recently been implicated in the development of a range of disease not traditionally thought to have infectious etiologies such as coronary artery disease (23), asthma (24), and some cancers (25). Hence, an association between childhood SES and adult host resistance would suggest a hypothetical pathway through which childhood experiences might influence a broad range of health outcomes. Here, we use a viral-challenge paradigm that controls for virus-specific immunity and exposure dose (26–30) to determine whether childhood SES is associated with adult susceptibility to infectious illness, and if so, whether the effect is limited to a critical period of low SES exposure, can be undone by changes in childhood SES and is explained by adult SES.

METHODS

Design

After assessment of adulthood and childhood SES, demographics, prechallenge immunity (virus-specific antibody levels), perceived stress and personality factors, volunteers were isolated in separate rooms on a single floor of a hotel, exposed to one of two rhinoviruses and then followed for 5 days to assess infection, and signs and symptoms of illness.

Subjects

Data were collected between 1997 and 2001. The subjects were 159 men and 175 women aged 18 to 54 years (mean = 28.8, SD = ±10.4) who responded to advertisements and were judged to be in good health. They were

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studied in 10 groups and were paid $800 for their participation. The study protocol was approved by the institutional review boards of the University of Pittsburgh School of Medicine and Carnegie Mellon University, and informed consent was obtained from each volunteer.

Experimental Plan

Volunteers underwent medical screenings and were excluded if they had a history of psychiatric illness, major nasal or otological surgery, asthma or cardiovascular disease, had abnormal urinalysis, CBC or blood enzymes, or were pregnant, currently lactating, seropositive for HIV, or on regular medication. Specific serum antibody titer to the challenge virus, demographics, weight, and height were assessed at screening and SES, perceived stress, and personality characteristics were assessed during the 6 weeks between screening and virus exposure.

During the first 24 hours of isolation (before viral exposure), volunteers had a nasal examination and a nasal lavage. Baseline symptoms, nasal mucociliary clearance, and nasal mucus production were assessed. Volunteers were excluded at that point if they had signs or symptoms of a cold. If a viral pathogen was isolated from the nasal lavage, the data from the subjects were excluded from analysis.

Then, subjects were given nasal drops containing 100 to 300 $TCID_{50}$ of 1 of 2 rhinovirus types (RV39 [$N = 228$] or RV23 [$N = 106$]). The isolation continued for 5 additional days. On each day, volunteers reported their respiratory symptoms, were assessed for nasal mucociliary clearance and nasal mucus production, and nasal lavage samples were collected for virus culture. Approximately 28 days after virus exposure, blood was collected for serological testing. All investigators were blinded to SES, psychological, and biological measures.

Childhood SES

We assessed 2 markers of childhood SES: parental education and parental home ownership during childhood. Volunteers scored both father's and mother's education on a 9-point scale ranging from "didn't finish high school" to "earned Ph.D., MD or other higher degree." For analysis, each score was assigned the number of years of education it represented (eg, completed high school = 12 years). (Categorical analyses yielded the same results).

Home ownership is associated with both greater assets and income (31) and with better health (32). For women, it is thought to be a more sensitive measure of SES than husband's occupation or income (32). Because adults are generally able to recall whether their parents owned or rented their homes when they were growing up, parental home ownership provides a good retrospective indicator of economic circumstance over the course of childhood and adolescence. Volunteers indicated (yes, no, or don't know) whether their parents owned their own home (including having a mortgage) during each of the subject's first 18 years of life. We calculated the total number of years that were marked yes.

Current SES

Volunteers scored their education level on the same scale used for parent education. Data were coded in the same manner. We also checked public real estate records to determine whether subjects owned their current homes.

Standard Control Variables

In the analyses, we controlled for virus type (RV23 [$N = 106$] or RV39 [$N = 228$]), prechallenge homotypic antibody titer (≤ 4 [$N = 209$] or ≥ 8 [$N = 125$]), age (18–21 [$N = 124$], 22–32 [$N = 96$], 33–54 [$N = 114$]), body mass index (weight [kilograms]/height [meters]^2, mean = 26.32), race (white [$N = 226$], other [$N = 108$]), sex [159 men and 175 women], and month of exposure (March [$N = 68$], May [$N = 108$], July [$N = 28$], September [$N = 65$], or December [$N = 65$]).

Viral Cultures and Antibody Response

Virus-specific neutralizing antibody titer was measured in serum collected before and 28-days after virus exposure (33) and results were expressed as reciprocals of the final dilution of serum. Daily nasal lavage samples were frozen at −80°C and later cultured for rhinovirus using standard techniques (33).

Signs and Symptoms

On each day of isolation, subjects rated on a scale from 0 (none) to 4 (very severe) the severity over the previous 24 hours of 8 symptoms (congestion, runny nose, sneezing, cough, sore throat, malaise, headache, and chills) (27). Daily mucus production was assessed by collecting used tissues in sealed plastic bags (28). The bags were weighed and the weight of the tissues and bags was subtracted. Nasal mucociliary clearance function was assessed as the time required for dye administered into the anterior area of the nose to reach the nasopharynx (28).

Baseline-adjusted daily scores for symptoms, mucus weights, and nasal clearance were each calculated by subtracting the appropriate baseline score from each of the 5 postexposure daily scores. Negative adjusted scores were reassigned a value of 0. These adjustments remove any effect of basal levels of signs and symptoms on postexposure response. Total scores for symptoms, mucus weight, and nasal clearance were calculated by summing the respective, adjusted daily scores over the 5 days.

Infections and Colds

Volunteers were considered to have a clinical cold if they *both* were infected and met an objective illness criterion. Infection was defined as recovery of the challenge virus on any of the 5 postchallenge days or a 4-fold or more increase in virus-specific neutralizing antibody titer (preexposure to 28-days after exposure) (29,30). The illness criterion was a total adjusted mucus weight of at least 10 g *or* a total adjusted mucociliary clearance time of at least 35 minutes (29). The mean total adjusted respiratory symptom score was 20.8 (SD = ±18.2) for those with vs. 7.5 (SD = ±10.0) for those without colds ($t(332) = -8.5$, $p < .001$).

Personality and Stress Measures

We measured a series of personality factors for 2 reasons. First, in previous papers we reported that greater extraversion and agreeableness (34) and greater positive emotional style (PES) (35) were associated with less risk of experimental colds in this sample. By including these variables as controls, we could assess whether associations of childhood SES and colds were attributable to these previously reported associations. Second, we wanted to include personality factors that might bias retrospective reports of childhood SES. Such factors include neuroticism, openness to experience, and conscientiousness (36).

We assessed the "Big 5" characteristics (extraversion, agreeableness, conscientiousness, neuroticism, and openness) thought to represent the basic structure of personality. The scale included 40 adjectives, 8 for each factor (29,34,37). We used the average of the scores from 2 assessments. We also measured PES, which is known to influence evaluative self-reports (35). Volunteers were phone interviewed on 3 evenings per week for 2 weeks during the month before viral challenge and interviewed on the evening of the first day (baseline) of isolation. They were asked how accurately (0 = not at all accurate to 4 = extremely accurate) each of 9 adjectives (lively, full-of-pep, energetic, happy, pleased, and cheerful, at ease, calm, relaxed) described how they felt during the last day (38,39). The scores for all 7 days were summed to form a measure of PES.

Finally, we have found in previous studies that psychological stress predicts susceptibility to respiratory illness (30,40). We thought it was possible that early childhood experiences influence adult stress levels, which in turn would influence susceptibility to disease. Perceived stress was assessed with the 4-item version of the Perceived Stress Scale (PSS) (41). The PSS assesses how unpredictable, uncontrollable, and overloading respondents find their lives.

Statistical Analyses

Scores for body mass, mucus weight, and mucociliary clearance were log transformed (base 10) to better approximate normal distributions. Stepwise logistic regression was used to predict presence/absence of infection and of a cold (42). Initially, SES variables were treated as continuous variables. There,

the regression coefficient, its standard error, and probability level are reported. When there was a significant association between a continuous SES variable and an outcome, we present odds ratios (OR) and 95% confidence intervals (CI) for SES variables categorized by tertile. In all analyses, the standard control variables were entered into the first step of the regression equation and the SES measures entered in the second step. To determine whether the association between SES measures and susceptibility to colds was substantially modified after controlling for other variables (perceived stress, personality, adulthood SES), these were added in the first step of the regression analyses along with standard controls. Interaction terms were entered in a third step.

RESULTS

Sixteen percent (52/334) of the volunteers did not know whether their parents owned their home during 1 or more of the 18 years. The incidence of colds for these volunteers (23.1%) was not different from that (26.2%) for those with complete information. The analyses were done on those 282 volunteers with complete data.

In an analysis in which all the standard control variables were entered together, 2 were independently associated with colds. Those with prechallenge antibody titers ≤4 were more likely to develop colds than those with titers ≥8 (OR = 2.6 [CI 1.38, 4.90]) and those exposed to RV39 were more likely (OR = 3.6 [CI 1.76, 7.37]) to develop colds than those exposed to RV23.

The number of years that parents owned their home was associated in a dose–response manner with a decreasing incidence of clinical colds (b = −0.07 [±0.02] for the continuous variable, $p < .01$; adjusted OR = 3.8 [CI 1.75, 8.46] for lowest number of years, 2.6 [CI 1.28, 5.47], and 1; Table 1).

A series of analyses addressed whether subject or parent education could account for the association of parental home ownership and colds. Father and mother education ($r = 0.25$ and 0.24, respectively, $p < .001$) and subject education ($r = 0.33$, $p < .001$) were all correlated with years of parental home ownership. However, none of these variables predicted colds nor did adding all three to the equation described above reduce the relationship between parental home ownership and risk of colds (b = −0.09 [±0.03], $p < .001$). There were also no interactions between parental home ownership and parent or subject education.

Additional analyses addressed whether current home ownership could account for the association of parental home ownership and colds. Current home ownership (only 8% of the sample owned their own homes) was not associated with either parental home ownership or with colds. Moreover, adding it to the equation did not reduce the association between parental home ownership and risk of colds ($N = 263$ with available data on current home ownership; before adding current home ownership b = −0.06 [±0.02], $p < .008$, after adding current home ownership b = −0.06 [±0.02*J*, $p < .008$).

To test whether SES at a particular time in childhood was more important, we calculated the number of years parents owned their homes when the volunteers were aged 1 to 9 and 10 to 18 years and conducted separate analyses (with standard controls) for each of these variables. Greater risk of developing a cold was associated with fewer years of parental home ownership at 1 to 9 (b = −0.15 [±0.04], $p < .001$; adjusted OR = 3.4 [CI 1.64, 7.24] for lowest number of years, 2.5 [CI 1.18, 5.12], and 1) and at 10 to 18 (b = −0.08 [±0.04], $p < .05$; adjusted OR = 1.82 [CI 0.98, 3.37] for lowest number of years, 0.95 [CI 0.11, 8.41], and 1). However, when the 2 measures were simultaneously entered into the same equation with standard controls, only the 1 to 9 variable was significant (b = −0.20 [±0.06], $p < .002$; adjusted OR = 3.6 [CI 1.23, 10.75] for lowest number of years, 2.5 [CI 1.16, 5.31], and 1). Hence, home ownership during early childhood was associated with colds independently of the association of home ownership during adolescence with colds.

We conducted further analyses of the age issue in order to attempt to refine the point in childhood that parental home ownership mattered most. To do this, we created 9 variables, each representing a 2-year span (1–2 years of age; 3–4, 5–6, etc.). We scored each variable between 0 and 2, with 0 = home not owned either year, 1 = home owned one of the two years, and 2 = home owned both years. We then conducted 9 separate logistic regressions, each containing the standard control variables and 1 of the 2-year span variables. Figure 1 plots the adjusted regression coefficients for each of the 2-year span variables ranging from infancy through adolescence. As apparent from the figure, the effect sizes are inversely proportional to age at which their parents owned their homes. The

TABLE 1. Percentages of Clinical Colds Presented by Number of Years Parents Owned Their Home, Preexposure Antibody Titer, and Virus Type[a]

Prechallenge Ab titer	Virus type	Years parents owned home		
		Low	Middle	High
≤4	RV23	27.3 ($n = 22$)	15.3 ($n = 12$)	14.3 ($n = 24$)
	RV39	54.5 ($n = 26$)	37.7 ($n = 36$)	30.8 ($n = 52$)
	Total	42.0 ($n = 48$)	32.1 ($n = 48$)	25.6 ($n = 76$)
≥8	RV23	13.9 ($n = 12$)	12.3 ($n = 10$)	6.4 ($n = 13$)
	RV39	25.9 ($n = 22$)	20.8 ($n = 23$)	16.0 ($n = 30$)
	Total	21.6 ($n = 34$)	18.2 ($n = 33$)	13.1 ($n = 43$)
Grand total		33.6 ($n = 82$)	26.8 ($n = 81$)	20.8 ($n = 119$)

[a] Percentages of clinical colds have been adjusted for standard control variables. The categorization of low, middle, and high scores on Years Parents Owned Home is based on whether the number of years fell in the lowest (0–6 years), middle (7–17 years), or highest (18 years) tertile.

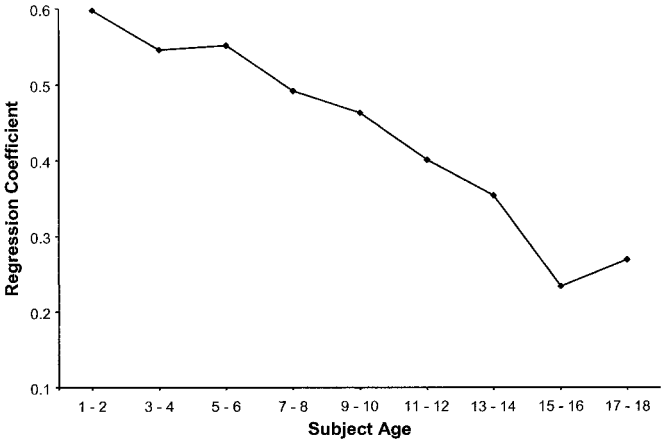

Figure 1. Adjusted effect size (regression coefficient) for the associations between parental home ownership and adult susceptibility to colds at different ages (2-year ranges) during childhood and adolescence.

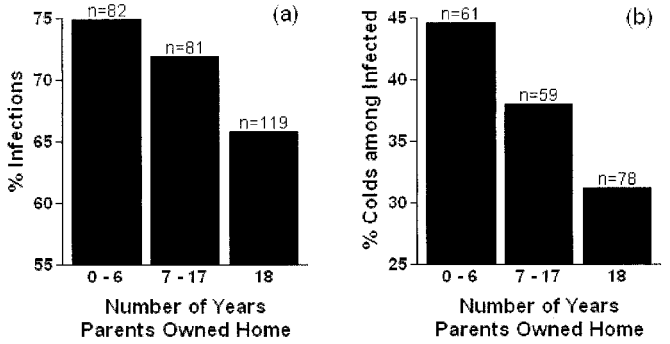

Figure 2. Adjusted (for standard controls) incidences of infection (a) and clinical illness among infected volunteers (b) as a function of years of parental home ownership (tertiled) when the subjects were aged 0 to 18 years.

coefficients in the figure were all significant ($p < .05$) except for 15–16, and 17–18 years of age. Then we fit a stepwise logistic regression, forcing in the standard control variables in the first step and allowing the 9 2-year span variables to step in. Only the 1- to 2-year variable entered the equation (b = −0.60 [±0.17], $p < .001$), suggesting not only that this is the largest effect, but also that once this infancy variable is entered, additional information does not improve the prediction.

We also conducted an analysis to more carefully examine whether *change* in parental home ownership between childhood and adolescence affected susceptibility. Median splits were used to create groups low and high for years of parental home ownership at 1–9 and at 10–18 years. We then created 4 categories representing patterns of change between childhood and adolescence: low at 1–9/low at 10–18 ($N = 96$), low/high ($N = 46$), high/low ($N = 9$), and high/high ($N = 131$). Analyses using these categories resulted in odds ratios of 3.2 for low/low [CI 1.54, 6.85], 3.2 for low/high [CI 1.39, 7.29], 1.2 for high/low [CI 0.21, 6.93], and 1 for high/high. Reanalysis dropping the high/low group (small N) yielded similar results (3.4 [CI of 1.62–7.31] for low/low; 3.3 [CI 1.41, 7.55] for low/high; and 1 for high/high).

The association of parental home ownership with cold incidence could reflect a decreased risk of infection and/or a decreased risk of clinical illness in infected persons (29,30). Years of parental home ownership was associated with lesser likelihood of both infection (b = −0.08 [±0.03], $p < .005$; OR = 3.3 [CI 1.4, 7.9], 1.6 [CI 0.8, 3.5], 1, Figure 2a) and clinical colds in infected subjects (b = −0.05 [±0.02, $p < .04$; OR = 3.1 [CI 1.4, 7.3], 2.5 [CI 1.2, 5.4], 1, $N = 198$; Figure 2b).

In previous papers, we reported that greater extraversion and agreeableness (37) and greater PES (34) were associated with less risk of experimental colds in this sample. To ensure that parental home ownership was not a marker of these or other dispositional characteristics, we reanalyzed the data set adding the Big 5 factors and PES to our standard control variables. Even with this very conservative analysis, parental home ownership still predicted cold incidence (b = −0.06 [±0.02], $p < .01$; OR = 3.5 [CI 1.6, 7.9], 2.4 [CI 1.1, 5.0], 1). Finally, in previous studies we have found that psychological stress predicted risk of colds (30,40). To be sure that parental home ownership was not a marker of current (adult) levels of perceived stress, we reanalyzed the data adding the PSS to the equation. Parental home ownership still predicted cold incidence (b = −0.07 [±0.02], $p < .003$; OR = 3.9 [CI 1.8, 8.8], 2.8 [CI 1.3, 5.8], 1).

DISCUSSION

The risk in adults for developing a common cold when exposed to a rhinovirus decreased with the number of years their parents owned their homes during the subject's childhood. This relation was independent of prechallenge immunity, age, body mass, race, sex, virus type, and month of exposure. The effect was substantial with an adjusted odds ratio of developing a cold of 3.8 when comparing the lowest to highest tertile of years of parental home ownership. Moreover, the risk of colds increased in a dose–response fashion with decreasing years of home ownership and occurred in both those with and without prechallenge antibody to the virus and across both virus types (see Table 1).

Similar to previous findings for cardiovascular and periodontal health (14), upward mobility in adolescence did not moderate the increased cold risk of low, early childhood SES. Moreover, exposure during infancy (1–2 years) was able to account for the entire effect of parental home ownership. These results support an early exposure hypothesis and are consistent with the hypothesis that there is a critical period confined to in utero exposure or to exposure in infancy (43). However, the data supporting the infant exposure hypothesis is subject to an alternative explanation. Parental home ownership during infancy (1–2 years of age) is strongly associated with the total number of years of parental home ownership ($r = 0.77$ for years of home ownership between 1 and 18 years of age and $r = 0.88$ between 1 and 9 years of age, $p < .001$ in both cases), which as noted earlier is a strong predictor of colds. If early exposure is key, SES related early environmental influences on childhood respiratory function such as air pollution, lead exposure, and damp housing (9,44) or more

general factors affecting the development of immunocompetence (22) such as nutrition, health care, or chronic childhood stress could mediate the observed association.

We also found that fewer years of parental home ownership was associated with 2 independent processes involved in the development of clinical illness: greater infection and greater illness expression. This is consistent with the argument that early economic experiences influence both the development of immune processes involved in host resistance to infection and disrupts the regulation of inflammatory mediators thought to be responsible for the expression of illness (22,40).

In contrast to home ownership, parent education was not associated with adult resistance to colds. It is possible that parent income and assets (as indicated by home ownership) are of primary importance in setting early childhood health trajectories. Certainly income and wealth would be more closely tied to several of the proposed pathways (eg, adverse social and physical exposures, malnutrition, high fat diets, health care, or chronic stress) than education. However, our assessment methods were not focused on capturing information regarding educational quality, which may better reflect SES. Moreover, we assessed parent education at the time of the study. It is possible that this assessment is inflated, because parents may have acquired additional education after the subjects' early childhood.

The lack of association between adult SES markers (volunteer education and current home ownership) and colds may to some degree reflect the insensitivity of these standard measures in this type of sample. For example, highly educated people who participate in a study requiring multiple visits to the hospital followed by 6 days of isolation are often unemployed or underemployed in occupations not commensurate with their education. In this case, level of education may not be a sensitive marker of either social status or income. The mean age of the sample (29 years) and the possibility that those with higher incomes probably would not volunteer to participate may both contribute to why only 8% of our participants were found to own their own homes and consequently why owning one's own home did not predict disease susceptibility. With these caveats in mind, it is still important that the risk associated with parental home ownership during childhood was not attributable to the subjects' adult social position as assessed in this study.

Retrospective reports of childhood experience can be flawed. However, home ownership seemed to be a salient issue for our subjects, who showed little trouble answering the questions. Moreover, controls for personality characteristics known to bias memories (view things more positively or negatively) did not influence the association between parental home ownership and colds. Although home ownership is a relatively insensitive measure of economic circumstances in places with highly inflated housing costs like New York City, San Francisco, or London, our subjects were for the most part raised in the Mid-Atlantic states, where housing costs have remained relatively affordable and home ownership is a reasonably sensitive indicator of economic circumstances.

We viewed home ownership as a marker of wealth and income and consequently of both access to health-promoting resources (eg, health care, nutrition) and of the ability to avoid the social and physical environmental risks associated with fewer economic resources (eg, chemical exposures, violence). However, parental home ownership might be correlated with other health-related factors such as number of children in a family, crowding, and exposure to day-care/child-care. Also, parents who did not own their homes are likely to have moved more frequently and to be separated, divorced, or single parents, all of which contribute to less stable environments. Although none of these variables have (as yet) been associated with adult host resistance to infectious illness, they are correlates of childhood economic circumstances that may explain or contribute to the association we found.

In summary, our data are consistent with childhood SES playing a role in susceptibility to cold viruses in adulthood. Because infectious agents have been implicated in the development of a wide range of diseases, this association between childhood SES and adult resistance to infection suggests a hypothetical pathway through which childhood experiences might influence multiple health outcomes.

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