ORIGINAL ARTICLE

Good Relationships With Parents During Childhood as Buffers of the Association Between Childhood Disadvantage and Adult Susceptibility to the Common Cold

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ABSTRACT

Objective: Children reared by parents of low socioeconomic status (SES) go on to have elevated rates of physical health problems and premature mortality. However, many children reared in low-SES families remain healthy throughout the life-span. Here, secondary analyses of archival data tested the hypothesis that a positive relationship with parents during childhood acts as a buffer of the increased risk of adult susceptibility to infectious illness associated with low childhood SES.

Methods: One hundred seventy-six healthy adults reported their childhood SES and the quality of their relationships with their parents during childhood. Relationship quality was defined as parental care, love and support, lack of conflict with parents, and family cohesiveness. Afterward, participants were exposed to a respiratory virus and monitored in quarantine for 5 days for the development of a "common cold" as indicated by infection and objective markers of illness.

Results: The increased risk of developing a cold associated with being reared in a low SES household was attenuated by a positive relationship with parents during childhood (b(SE) = 0.08 (0.03), p = .010). This buffering of disease risk held up across the four components of relationship quality (p values < .05). The association was independent of adult SES, demographics, prechallenge immunity to the virus, current levels of neuroticism and stress, parental divorce during childhood, and number of siblings (p values < .05).

Conclusions: Individuals with positive relationships with their parents during childhood are buffered from the increased risk of adult susceptibility to an infectious disease associated with low childhood SES.

Key words: childhood socioeconomic status, parent-child relationship, common cold, immunocompetence.

INTRODUCTION

Children reared by parents of low socioeconomic status (SES) go on to have elevated rates of morbidity from infectious and cardiovascular diseases throughout the life-span, as well as greater risk of premature mortality (reviews by Refs. (1,2)). These associations are independent of traditional risk factors such as poor health practices and lack of access to medical resources. In most cases, they also withstand adjustment for adult SES.

Although the association between childhood SES and adult health is well established, many children reared in low-SES families remain healthy across the life course (3). Identifying sources of this resilience is important for basic scientists interested in stress and coping, as well as for practitioners and policymakers seeking intervention targets for reducing health disparities. One source of resilience that may buffer individuals from the physical risks associated with low childhood SES is the quality of the parent-child relationship during childhood and adolescence. In fact, there is increasing evidence that better relationships with one's parents may reduce the detrimental effects of childhood adversity on concurrent physiology (e.g., Refs. (4–9)). Less is known about whether these benefits extend to physical health problems, particularly those that do not appear until later in life (10). Data from the Midlife in the United States Study (MIDUS) begin to suggest that they do. Specifically, children from low-SES families were more likely to develop metabolic syndrome—a set of risk factors for cardiovascular disease and type 2 diabetes—as adults, but this risk was attenuated in those who reported having had a nurturing relationship with their mothers, but not by nurturing relationships with their fathers (10).

Here we use an archival database (Pittsburgh Cold Study 3 [PCS3]) to examine whether the quality of parent-child relationships buffers against the increased risk of adult susceptibility to an objectively defined disease state that has been associated with

CI = confidence interval, **FES** = family cohesion subscale of Family Environment Scale, **MIDUS** = Midlife in the United States Study, **OR** = odds ratio, **PBI** = Parental Bonding Instrument, **PCS3** = Pittsburgh Cold Study 3, **PPCR** = Positive Parent-Child Relationship composite scale, **PRS** = Parental Relationship Scale, **PSS** = Perceived Stress Scale, **RFQ** = Risky Families Questionnaire, **SES** = socioeconomic status


Podcast

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low childhood SES, the common cold (11,12). In PCS3, healthy volunteers reported on childhood SES and completed four scales that assessed different aspects of childhood relationships with parents: parental care, love and support, getting along with parents, and family cohesiveness. After these assessments, volunteers were exposed to a virus that causes the common cold and followed in quarantine to determine whether they developed a clinical illness (viral infection plus objective signs of illness). Previous analysis found lower childhood SES to be associated with greater disease susceptibility (12). Here, in a secondary analysis of these data, we test whether the increase in adult disease susceptibility associated with childhood SES is buffered by components of positive childhood relationships with parents.

Data are also available from this study to examine whether any reported buffering effects could be attributed to adult levels of stress and neuroticism, to childhood experiences of parental divorce, or to living with several siblings. Moreover, one of the four scales we used in this study to assess the quality of childhood relationships with parents distinguishes between relationships with mothers and fathers, allowing a preliminary view of whether quality of relationship effects may be modified by source.

METHODS

Design

The temporal sequence of study activities is displayed in Figure 1. Healthy adult participants were asked to report both whether their parents owned the family home during each year of their childhood and adolescence (i.e., ages 1–18 years) and to complete four different scales that assessed their relationships with their caregivers during childhood. They were subsequently exposed to a virus that causes the common cold and followed in quarantine to assess whether they developed a clinical illness.

Participants

Participants were drawn from 212 volunteers aged 18 to 55 years selected for good health from greater Pittsburgh, Pennsylvania. The participants were recruited by newspaper and posted advertisements, and each was paid $1000. The data were collected between 2007 and 2011. The study was approved by the institutional review boards of both Carnegie Mellon University and the University of Pittsburgh, and all participants provided signed informed consent forms. All procedures, questionnaires, and the data used in analyses in this article are available online: PCS3 on www.commoncoldproject.com. Only participants with complete parental home ownership data for ages 1 through 18 years (n = 176; 42% women) were included in the present analyses. Excluded participants did not differ from the participants examined here (p values > .15) on most demographic characteristics, rates of becoming infected with the challenge virus, rates of developing a clinical cold, or any of the four individual measures or the aggregate measure assessing their relationship with parents/family during childhood. The two groups did, however, differ on racial/ethnic distribution, with excluded participants being less likely to self-identify as white (52.8% versus 69.9%, $\chi^2(1) = 3.96$, $p = .047$).

Procedures

Volunteers were included for participation if they were in "good general health" as determined by medical history, physical examination, and clinical profiles via urinalysis, complete blood count, and analysis of blood chemistry and were not currently taking any medications (see Ref. (13) and www.commoncoldproject.com). Baseline immunity to the challenge virus (viral specific antibody titers), demographics, and anthropometrics were also assessed at screening. Volunteers with a viral specific antibody level >4 at the medical screening were excluded to maximize infection rate (14).

Qualifying volunteers completed a demographic questionnaire and a measure of perceived stress. They were then interviewed by telephone on 14 consecutive evenings about their daily social interactions (data not included here) and then isolated in a local hotel for a 6-day period. During the first 24 hours of quarantine (before viral exposure), the volunteers completed the childhood SES and psychosocial questionnaires, received baseline nasal examination and nasal wash, and were assessed for baseline symptoms, nasal mucociliary clearance, and nasal mucus production (see the Measures section). Participants were dismissed from the study if they displayed signs or symptoms of a cold on that day, and data for participants in whom a virus was isolated from the nasal lavage fluids collected on that day were excluded from all analyses. Participants were then given nasal drops containing approximately 100 50% tissue culture infectious dose per milliliter of rhinovirus 39, a virus that causes a common cold. (A placebo condition was not included because colds are caused by viruses and participants receiving placebos in this paradigm do not develop colds (15).) The quarantine continued for 5 days. On each day, volunteers were assessed for nasal mucociliary clearance and nasal mucus production, and nasal wash samples were collected for virus culture. Approximately 28 days after virus exposure, blood was collected for serological testing. Investigators were blinded to all predictor variables at all points of the trial.

Measures

Standard Control Variables

Self-reported age, sex, and race (selected from white, black, Native American, Asian/Pacific Islander, Hispanic/Latino, or other, and coded as white and nonwhite because of small numbers reporting nonblack minority categories), body mass index (weight in kilograms/[height in meters]^2), a marker of participant's current (adult) SES (educational attainment [in years]), season of the year (winter, spring, summer), and baseline immunity to the challenge virus (neutralizing specific antibody titer as assessed from a blood sample collected 2–3 days before exposure to the virus) were included in all models adjusting for covariates.

Childhood SES

Home ownership is a widely used marker of childhood SES and is associated with greater assets and income and with better health (13,16,17). 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 (11,18).

Data on parental home ownership were collected by retrospective self-report questionnaire during the baseline day in quarantine. For each year of their childhood and adolescence (i.e., ages 1 through 18 years), participants were asked whether their parents owned their family's home. Response alternatives included *yes*, *no*, and *I do not know*. We calculated the total number of years of home ownership from ages 1 through 18 with *I do not know* treated as missing data.

Parental Relationships

We created the Positive Parent-Child Relationship composite scale (PPCR) by calculating the average standardized score of four separate study measures that tapped qualities of the relationships between study participants and their parents during childhood. These included assessments of parental care, love and support, lack of conflict, and family cohesiveness. The measures were administered before the viral challenge. The scores from each of the individual measures were intercorrelated (r = 0.50–0.81, p values < .001; mean r = 0.63), each was highly correlated with the total PPCR score (r = 0.76–0.90, p values < .001) and loaded on a single factor (principle component loadings = 0.44–0.53). The primary analysis is based on this aggregated variable—PPCR—but we also present individual analyses

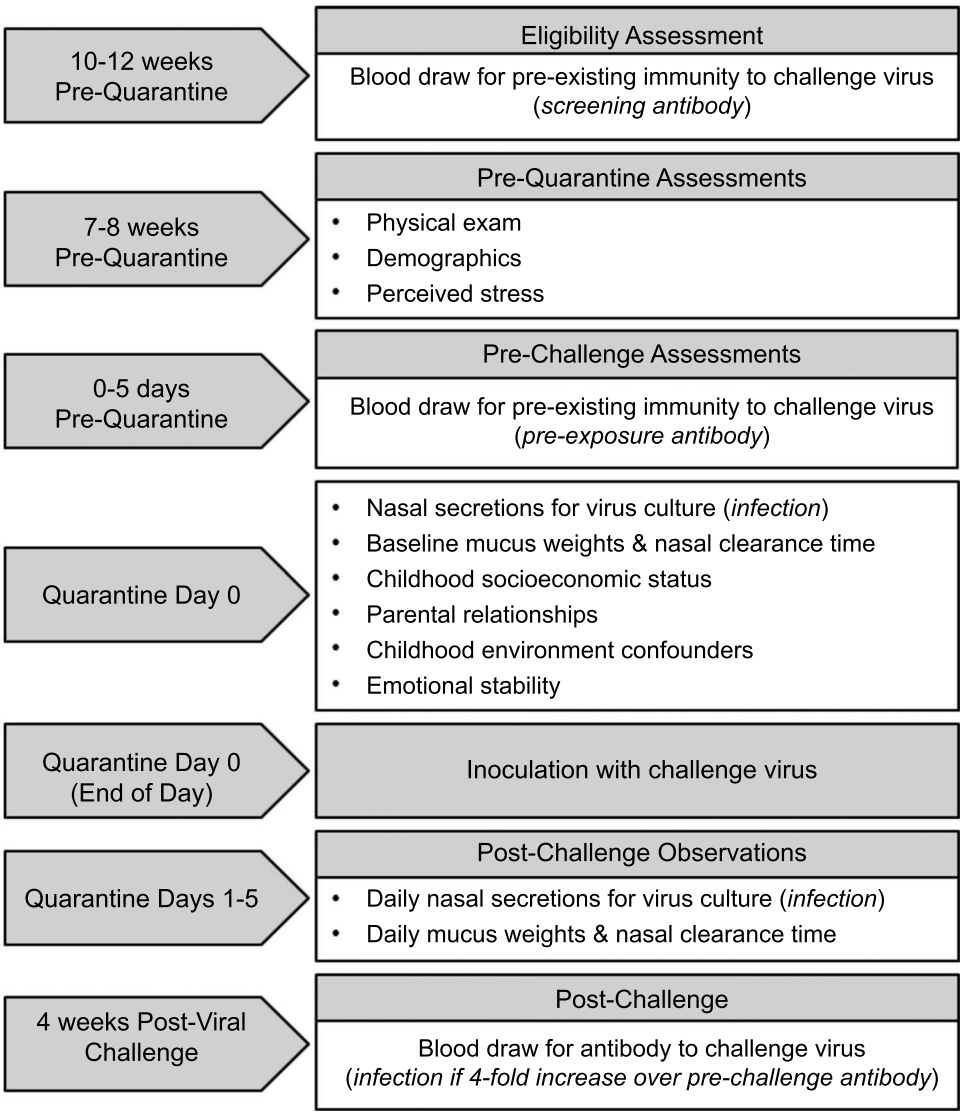

FIGURE 1. Temporal sequence of study activities.

focusing on each of the four individual scales. Each of the constituent measures is described hereinafter.

The parental care subscale of the Parental Bonding Instrument (PBI) (19) assesses retrospective self-report of components of the parent-child relationship. Using a 4-point rating scale (1, *very like*; 4, *very unlike*), respondents indicate the degree to which a series of phrases describing parental behaviors are like those enacted by their own parents while they were children. The time frame used is "during the first 15 years of my life." The modified version of the PBI care scale (using 6 of the original 12 items) included items chosen based on results of a factor analysis that was published in the source article (19). Examples of items included "understood my problems and worries" and "seemed emotionally cold to me" (reverse scored). Scores were coded so that higher values represent getting along with the parents. The internal reliability of the Parental Care Scale in this sample was $\alpha = .89$.

The cohesion subscale of the Family Environment Scale (FES) (20) assesses the degree of commitment and support provided by participants' family during childhood and adolescence. The modified scale used here uses five of the original nine items of the FES cohesion subscale. Using a 5-point Likert scale (1, *strongly disagree*; 5, *strongly agree*), participants indicated the extent to which they agree with statements describing their family dynamic during childhood. Participants are asked to respond in reference to "When I was growing up." Examples of items included "there was a feeling of togetherness in our family" and "there was little unity in our family" (reversed). The items were summed to create the modified cohesion subscale score. The internal reliability of the FES cohesion subscale in this sample was $\alpha = .89$.

Love, warmth, and support questions derived from the Risky Families Questionnaire (RFQ). The RFQ assesses the degree of risk of physical, mental, and emotional distress that subjects faced in their homes during childhood and adolescence (21). Subjects rate the frequency with which certain events or situations occurred in their homes during the ages of 5 to 15 years using a 5-point Likert scale (1, *not at all*; 5, *very often*). Here, based on a rotated (oblimin transformation) factor analysis of the scale, we selected two items worded in the positive direction that loaded on the same factor and reflected love, warmth, and support. The two items were as follows: "How often did a parent or other adult in the household make you feel that you were loved, supported, and cared for?" and "How often did a parent or other adult in the household express physical affection for you, such as hugging, or other physical gestures of warmth and affection?"

The two items were correlated ($r = 0.79$). The items were averaged to create a subscale representing family warmth and support.

We created the Parental Relationship Scale (PRS) to assess the extent to which participants got along with their parents during childhood. Three items each were used to assess participants' childhood relationships with their mothers and fathers, respectively. For each parent, participants were asked "How often did you get along with your mother (father)?" These two items (father and mother version) were presented three times with reference periods of participant ages 5, 10, and 15 years. Response options ranged from 1 (*all the time*) to 4 (*never*). Participants were also provided with two nonresponse options: *not sure* and *I lived apart from my mother (father) and we never spoke*. To minimize missing data, responses indicating that the participant lived apart from his or her parent and never spoke were recoded to a value of 4 (never got along with parent). A total of 36 participants reported living apart from either parent during one or more of the three reference years. (Analysis of data excluding the recoded responses yielded identical conclusions.) Aggregate parental relationship variables were then created by taking the average of all nonmissing values across ages 5, 10, and 15 years separately for each parent (98.3% of participants had maternal relationship data for 2 years or more, whereas 88.1% had paternal relationship data for 2 years or more). A total PRS score was calculated by summing the mother and father scores. All scores were reverse coded so that higher values represent getting along with the parent more frequently.

Psychological Confounders

Emotional stability and psychological stress are two of the most likely contributors to retrospective reporting bias (22,23). Emotional stability (inverse of neuroticism) was measured using a 10-item version of the emotional stability subscale from the International Personality Item Pool Big-Five Factor Markers (24). Perceived stress was assessed using the 10-item version of the Perceived Stress Scale (PSS) (25), which asks respondents to report how frequently during the past month they found their lives to be unpredictable, uncontrollable, and overloaded.

Childhood Environment Confounders

Parental divorce status was assessed by asking participants whether their parents ever separated or divorced and their age at the time the separation/divorce took place. A dichotomous variable was created indicating those participants who were 18 years or younger when their parents' marriage dissolved. Number of siblings was assessed by asking participants to report for each year spanning ages 1 through 18 years how many siblings lived with them in the home. An aggregate variable was created by taking the average number of siblings across the 18 years.

Disease Outcomes

The primary outcome was clinical colds, which subsumes both infection and objective signs of clinical illness (See Diagnosis of Clinical Illness).

Infection

Infection is the replication of the virus. When an upper respiratory virus replicates, it can be found in nasal secretion samples. Samples collected daily in a saline wash of the nose were frozen and later cultured for the challenge virus using standard techniques (26). Because the immune system responds to infection by producing antibody to the virus, increases in viral-specific antibody level provide an indirect marker of infection. Hence, we also compared virus-specific antibody levels measured in serum collected before and 28 days after exposure (26). Infection was operationally defined as recovery of the challenge virus on any of the five postchallenge days or a ≥4-fold rise in virus-specific serum neutralizing antibody titer (preexposure to 28 days postexposure) (26).

Signs of Illness

We assessed two *objective markers of upper respiratory illness*: nasal mucus production and nasal mucociliary clearance function. Daily mucus production was assessed by collecting used tissues in sealed plastic bags (27). The bags were weighed, and the weight of the tissues and bags was subtracted resulting in the weight of mucus produced. Clearance function refers to the effectiveness of nasal cilia in clearing mucus from the nasal passage toward the throat and is subjectively experienced as congestion. Clearance function was assessed as the time required for a saccharin-dyed solution administered into the anterior nose to be tasted by the participant (27). To create baseline-adjusted daily scores for each measure, we subtracted the appropriate baseline (day before challenge) score from each of the five postchallenge daily scores (14). Negative adjusted scores were reassigned a value of 0. Average nasal clearance scores were calculated by taking the mean of the adjusted daily clearance scores over the 5 postchallenge days. Total mucus weight scores were created by summing the average daily scores across the 5 days.

Diagnosis of Clinical Illness

Participants were diagnosed as having a clinical illness if they were both infected with the challenge virus and met either of the following criteria: total baseline-adjusted mucus weight of 10 g or more or average (across all postchallenge days) baseline-adjusted nasal mucociliary clearance time of 7 minutes or longer (14).

Data Analysis

All continuous variables were first centered at their respective means. Hierarchical logistic regression analyses were then conducted to test the buffering effect of a positive family environment on the link between childhood SES and development of a clinical cold. In the unadjusted model, clinical colds were first predicted from the main effects of childhood SES and PPCR in the first step, followed by the interaction effect of childhood SES and PPCR in the second step. In the adjusted model, control variables (e.g., age, sex, race/ethnicity, years of education, body mass index, season of the year, and baseline immunity to the challenge virus in model with standard controls) and their interactions with childhood SES were included in the first step along with childhood SES and PPCR. The interaction of childhood SES and PPCR was included in the second step. The observed significant interaction between childhood SES and PPCR was then probed with tests of simple slopes between childhood SES and clinical colds at one standard deviation (SD) above and below the mean PPCR score. A series of parallel logistic regression models were then tested to examine the buffering effects of each of the constituent measures of the overall positive family environment composite variable.

To rule out alternative explanations of primary results, additional potential adult psychological and child environment confounds (i.e., neuroticism, perceived stress, parental divorce, number of siblings) and their interactions with childhood SES were included in models as additional covariates in two separate follow-up models.

All statistical analyses were performed using Stata 14, and results are reported as b(SE) along with p values as well as odds ratios (ORs) along with their corresponding 95% confidence intervals (95% CIs).

RESULTS

The sample had a mean (SD) age of 29.66 (10.89) years and was 42.0% female (58.0% male). Most (69.9%) participants self-identified as white (24.4% black/African American, 1.7% Asian or Pacific Islander, 1.1% Hispanic or Latino, 2.8% "other"); and the average (SD) educational attainment was 14.07 (1.88) years, or "some college."

Table 1 presents means and SDs for the childhood socioeconomic and family environment measures, and potential confounders. Table 2 presents the correlations among these variables. Across measures, ratings of family relationships and environment were largely favorable.

TABLE 1. Sample Descriptives (n = 176)

	Mean	SD	Minimum	Maximum
Childhood SES, parental home ownership, y	11.42	6.98	0	18
Positive parent-child relationship composite (PPCR)	0.01	0.85	−2.27	1.52
Parental care (PBI)	17.94	4.39	6	24
Family cohesion (FES)	17.54	4.35	5	25
Family warmth (RFQ)	3.86	0.99	1	5
Get along with parents (PRS)	5.60	1.32	2	8
BMI, kg/m^2	27.32	6.52	18.86	50.95
Prechallenge antibody titers	2.34	2.73	1	16
No. siblings	1.61	1.31	0	8.5
Early parental divorce, %	42			
Neuroticism	34.32	7.49	16	50
Perceived stress	12.19	5.69	0	31

SD = standard deviation; SES = socioeconomic status; PPCR = Positive Parent-Child Relationship composite scale; PBI = Parental Bonding Instrument; FES = Family Environment Scale; RFQ = Risky Families Questionnaire; PRS = Parental Relationship Scale; BMI = body mass index.

Tests of Hypotheses

We first fit an unadjusted model that examined whether our aggregate assessment of growing up in a positive family environment (PPCR) moderated the association between lower childhood SES and increased risk of developing a cold. As previously reported from these data (12), the fewer number of childhood years spent living in homes owned by their parents, the greater was the risk of developing a cold. Each year not living in a parent owned home was associated with a 5% increase in risk (b(SE) = −0.06 (0.02), OR = 0.95, 95% CI = 0.90–0.99, p = .021). There was no main effect of PPCR (b(SE) = −0.07 (0.20), OR = 0.93, 95% CI = 0.63–1.37, p = .722). However, here we found that PPCR moderated the association of parental home ownership with cold risk (b (SE) = 0.08 (0.03), OR = 1.08, 95% CI = 1.02–1.15, p = .010). A moderation effect was also found in an adjusted version of this model controlling for the seven standard covariates and each of their interactions with childhood SES (Table 3). As illustrated in Figure 2 (based on adjusted model), each additional year not living in a parental owned home was associated with a 2% increase in risk of developing a cold among those who reported growing up in a less positive family environment (i.e., one SD below the sample mean; b(SE) = −0.02 (0.005), OR = 0.98, 95% CI = 0.97–0.99, p = < .001). In contrast, there was no association between childhood SES and colds among participants who reported growing up in a more positive family environment (i.e. one SD above the sample mean; b(SE) = −0.001 (0.01), OR = 1.00, 95% CI = 0.98–1.01, p = .900).

Finally, it is possible that other individual difference factors, such as emotional stability and perceived stress, and other features of the childhood family environment, including number of siblings

TABLE 2. Correlations Among Study Variables

	1.	2.	3.	4.	5.	6.	7.	8.	9.
1. Childhood SES (years of parental home ownership)									
2. Positive parent-child relationship (PPCR)	0.23**								
3. Parental care (PBI)	0.10	0.90***							
4. Family cohesion (FES)	0.27***	0.88***	0.72***						
5. Family warmth (RFQ)	0.13†	0.89***	0.81***	0.73***					
6. Get along with parents (PRS)	0.28***	0.75***	0.54***	0.54***	0.50***				
7. No. siblings	−0.07	−0.06	−0.05	−0.09	−0.10	−0.03			
8. Early parental divorce	−0.36***	−0.33***	−0.19**	−0.34***	−0.19*	−0.41***	−0.08		
9. Emotional stability	−0.02	0.28***	0.19*	0.33***	0.13†	0.29**	0.08	−0.05	
10. Perceived stress	−0.07	−0.19**	−0.16*	−0.18*	−0.09	−0.23**	−0.03	0.13†	−0.44***

PPCR = Positive Parent-Child Relationship composite scale; PBI = Parental Bonding Instrument; FES = Family Environment Scale; RFQ = Risky Families Questionnaire; PRS = Parental Relationship Scale.

Results reflect bivariate correlations.

* p < .05.

** p < .01.

*** p < .001.

† p < .06–.09.

TABLE 3. Model Relating Seven Standard Covariates, Childhood SES, Positive Parent-Child Relationship, and Their Interaction to Cold Risk

Variable	*b*(SE)	OR	95% CI	*p*
Intercept	−1.14 (0.31)	0.32	0.17–0.59	<.001
Childhood SES (years of parent home ownership)	−0.12 (0.05)	0.89	0.81–0.98	.017
Positive parent-child relationship (PPCR)	0.17 (0.24)	1.19	0.74–1.91	.480
Age	0.03 (0.02)	1.03	1.00–1.07	.064
Sex	0.32 (0.40)	1.38	0.63–3.03	.428
Race	0.13 (0.50)	1.14	0.43–3.00	.796
Prechallenge antibody titers	−0.21 (0.11)	0.81	0.65–1.01	.056
BMI	0.06 (0.03)	1.06	0.99–1.13	.087
Season	0.06 (0.24)	1.06	0.67–1.68	.810
Current education	0.07 (0.11)	1.07	0.87–1.33	.512
Childhood SES by age	0.003 (0.002)	1.00	1.00–1.01	.240
Childhood SES by sex	0.02 (0.06)	1.02	0.90–1.14	.791
Childhood SES by race	0.07 (0.07)	1.08	0.95–1.22	.259
Childhood SES by prechallenge antibody titers	0.004 (0.02)	1.00	0.97–1.04	.780
Childhood SES by BMI	−0.01 (0.01)	0.99	0.98–1.00	.217
Childhood SES by season	−0.02 (0.03)	0.98	0.92–1.05	.587
Childhood SES by current education	−0.01 (0.02)	0.99	0.96–1.02	.411
Childhood SES by PPCR	0.10 (0.04)	1.11	1.03–1.19	.006

SES = socioeconomic status; OR = odds ratio; CI = confidence interval; PPCR = Positive Parent-Child Relationship composite scale; BMI = body mass index.
Results are from logistic regression analyses.

in the home, and parental divorce/separation status might explain the current findings given that these factors are thought to be associated with positive relationships with parents and could feasibly be responsible for what seems to be positive relationship buffering of childhood SES associations with health outcomes. To address this issue, we reran analyses statistically adjusting for each of these factors and their interactions with childhood SES. As presented in Table 4, in both models, adjusting for these potential confounding variables had no discernible effect on the childhood SES-by-PPCR interaction.

Constituent Measures of Overall Positive Family Environment

We also examined each constituent measure of our overall family environment composite variable (i.e., PBI parental care subscale, FES family cohesion subscale, RFQ family love and support, and the PRS: getting along with parents). As presented in Table 5 (models adjusted for seven standard controls), analogous findings to primary results were observed; that is, each of the constituent measures interacted with the number of years that participants' parents owned a home to predict cold risk (p values < .05).

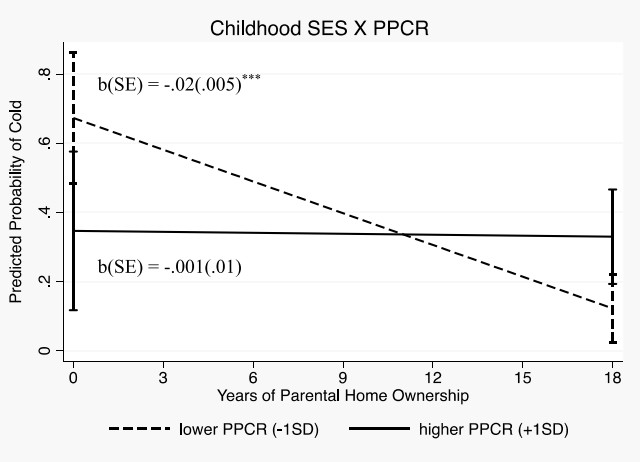

FIGURE 2. Moderating effect of positive parent-child relationship (PPCR) on the association between years of parental home ownership and cold risk. Fewer years of parental home ownership was associated with greater risk of developing a cold only among those with lower childhood PPCR scores. Parental home ownership was not associated with cold risk among those with higher childhood PPCR scores. Error bars represent 95% confidence intervals.

TABLE 4. Covariate-Adjusted Models Relating Childhood SES, Positive Parent-Child Relationship, and Their Interaction to Cold Risk

Variable	*b*(SE)	OR	95% CI	*p*
Model 1: adult psychological covariates				
Childhood SES	−0.06 (0.03)	0.94	0.90–0.99	.027
Positive parent-child relationship (PPCR)	0.02 (0.23)	1.02	0.66–1.60	.913
Childhood SES by PPCR	0.07 (0.03)	1.07	1.00–1.14	.036
Model 2: childhood environment covariates				
Childhood SES	−0.08 (0.04)	0.93	0.86–1.00	.050
Positive parent-child relationship (PPCR)	0.11 (0.23)	1.11	0.71–1.75	.639
Childhood SES by PPCR	0.09 (0.03)	1.09	1.02–1.17	.007

SES = socioeconomic status; OR = odds ratio; CI = confidence interval; PPCR = Positive Parent-Child Relationship composite scale.

Results are from logistic regression analyses. All models reported in this table use the positive parent-child relationship composite variable. In model 1, adult psychological covariates include emotional stability, perceived stress, and product terms reflecting the interaction of these variables with Childhood SES. In model 2, the childhood environment covariates include parental divorce, number of siblings, and product terms reflecting the interaction of these variables with Childhood SES.

As depicted in Figure 3, the association between fewer years and parental home ownership and greater cold risk was evident among those who reported less parental care as assessed by the PBI (b(SE) = −0.02 (0.01), $p < .001$, OR = 0.98, 95% CI = 0.97–0.99), less family cohesion as assessed by the FES (b (SE) = −0.02 (0.01), $p < .001$, OR = 0.98, 95% CI = 0.97–0.99), less family warmth, love and support as assessed by the RFQ (b (SE) = −0.02 (0.01), $p < .001$, OR = 0.98, 95% CI = 0.97–0.99), and getting along with their parents less frequently as assessed by the PRS (b(SE) = −0.02 (0.004), $p < .001$, OR = 0.98, 95% CI = 0.97–0.98). By contrast, the increased cold risk associated with fewer years of parental home ownership was attenuated (not reaching traditional levels of significance) among participants who reported parent relationships characterized by greater levels of care (b(SE) = −0.01 (0.01), $p = .388$, OR = 0.99, 95% CI = 0.98–1.01); cohesion (b(SE) = −0.004 (0.01), $p = .584$, OR = 1.00, 95% CI = 0.98–1.01); warmth, love, and support (b(SE) = −0.005 (0.01), $p = .514$, OR = 1.00, 95% CI = 0.98–1.01); and getting along with one another (b(SE) = 0.004 (0.01), $p = .609$, OR = 1.00, 95% CI = 0.99–1.02).

Exploratory Analyses

Do maternal and paternal relationships similarly buffer the effect of low childhood SES? We used the PRS, the only component relationship scale that asked participants about their relationships with *each* of their parents, as a data source to provide a preliminary answer to this question. The main effects of paternal and maternal relationship and each of their interactions with parental home

TABLE 5. Separate Models With Standard Controls Relating Childhood SES, Each of the Four Positive Family Environment Measures, and their Interactions to Cold Risk

Variable	*b*(SE)	OR	95% CI	*p*
Model 1: parental care (PBI)				
Childhood SES	−0.11 (0.05)	0.90	0.82–0.98	.019
Parental care	0.07 (0.05)	1.07	0.98–1.17	.135
Childhood SES by parental care	0.01 (0.01)	1.01	1.00–1.03	.049
Model 2: family cohesion (FES)				
Childhood SES	−0.10 (0.05)	0.90	0.82–0.99	.031
Family cohesion	−0.01 (0.04)	0.99	0.91–1.08	.817
Childhood SES by family cohesion	0.01 (0.01)	1.01	1.00–1.03	.043
Model 3: family warmth (RFQ)				
Childhood SES	−0.11 (0.05)	0.89	0.81–0.98	.017
Family warmth	0.17 (0.20)	1.19	0.80–1.77	.399
Childhood SES by family warmth	0.07 (0.03)	1.07	1.01–1.13	.014
Model 4: get along with parents (PRS)				
Childhood SES	−0.09 (0.05)	0.91	0.83–1.001	.074
Get along with parents	0.05 (0.17)	1.05	0.76–1.47	.757
Childhood SES by get along with parents	0.08 (0.03)	1.08	1.03–1.14	.003

SES = socioeconomic status; OR = odds ratio; CI = confidence interval; PBI = Parental Bonding Instrument; FES = Family Environment Scale; RFQ = Risky Families Questionnaire; PRS = Parental Relationship Scale.

Results are from logistic regression analyses.

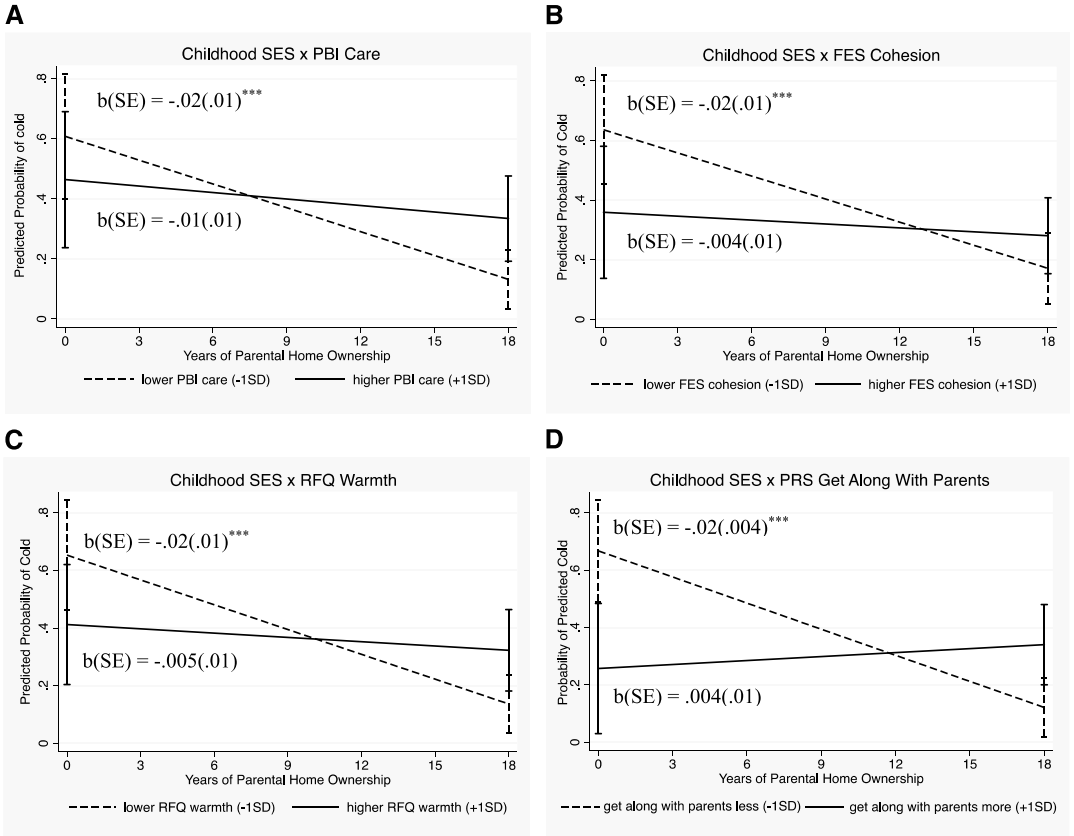

FIGURE 3. Moderating effect of PBI parental care (A), FES family cohesion (B), RFQ family warmth (C), and getting along with parents (D) on the association between parental home ownership and cold risk. Fewer years of parental home ownership was associated with greater risk of developing a cold only among those reporting lower parental care, family cohesion, family warmth, and getting along with parents while growing up. Parental home ownership was not associated with cold risk among those who reported greater parental care, family cohesion, family warmth, and getting along with parents while growing up. Error bars represent 95% confidence intervals. PBI = Parental Bonding Instrument; FES = Family Environment Scale; RFQ = Risky Families Questionnaire.

ownership were entered into two separate base models with standard covariates and their interactions with parental home ownership. Results revealed that an amicable relationship with both parents buffered the relation between lower childhood SES and greater susceptibility to developing a cold (childhood SES by paternal relationship: (b(SE) = 0.09 (0.03), p = .008, OR = 1.09, 95% CI = 1.02–1.16); childhood SES by maternal relationship: b (SE) = 0.09 (0.04), p = .040, OR = 1.09, 95% CI = 1.00–1.19).

DISCUSSION

The present data provide strong evidence for the potential role of parent-child relationship quality during childhood in protecting individuals from risks to adult health associated with low childhood SES. Using a unique viral challenge paradigm with objective assessment of disease susceptibility, we found that the association of low childhood SES with increased risk of adult disease susceptibility is attenuated by a history of positive relationships with parents during childhood.

Our assessment of parent-child relationship quality was broader than the parental nurturance measure used in MIDUS (10) and the scales used in studies of childhood adversity and concurrent physiology (e.g., Refs. (4–9)). Our aggregate measure (PPCR) included assessments of parental care, love and support, lack of conflict with parents, and family cohesiveness. These constituents of relationship quality were moderately to highly intercorrelated and loaded on a single principle component. Participants who scored higher on PPCR were protected from the increased cold risk associated with low childhood SES, whereas those with lower PPCR scores were not. This pattern of results also was found in separate analyses of the four constituent dimensions. Together, these findings suggest that there are multiple, independent, features of positive parent-child relationships that can offset health risks in adulthood associated with low childhood SES.

Using the single scale that separately assessed childhood relationships with each parent (PRS), we found that the buffering effect of positive relationship quality was evident for both maternal and paternal relationships. This finding contrasts with the earlier report from MIDUS (10) wherein the buffering effect on metabolic syndrome was evident only for relationships with mothers. This discrepancy may be due to differences in the physiological systems involved in metabolic syndrome and infectious disease. Alternatively, it may be attributable to a cohort effect (MIDUS participants were born between 1921 and 1971, and PCS3 participants were born between 1952 and 1991), with fathers of the later-born adults (PCS3) being subject to different norms and expectations promoting closer relationships with their

children. It also is possible that parental nurturance (used in MIDUS) is more relevant to relationships with mothers than with fathers. In contrast, the PRS, which distinguishes between maternal and paternal relationships, makes a more general assessment of getting along with parents. Thus, the present study may capture other aspects of relationships with fathers (and possibly mothers) that act as effective buffers.

We have framed our results as suggesting that parental-child relationship quality is a moderating factor. Alternatively, one could hypothesize that low SES in childhood diminishes parental quality, which in turn enhances the risk of poor health in later life. However, parental quality was not directly associated with risk of disease susceptibility in our data; hence, there is no support for this alternative hypothesis.

A potential limitation of this work is the retrospective reports of childhood SES and of relationships with parents. However, by adjusting for emotional stability (neuroticism) and psychological stress, we eliminated two of the most likely contributors to reporting bias that might account for our findings (22,23). The study also might be limited by the use of home ownership as a marker of childhood SES. Clearly, home ownership would not be a sensitive measure of childhood SES in metropolitan areas with high costs of living where renting is not uncommon among persons with high incomes (11,12). However, because the data were collected in the Pittsburgh area where housing costs are low, years of parental home ownership is a reasonable marker of childhood SES. Home ownership is known to approximate the amount of wealth a family possesses and may be even more important than income and education in shaping health outcomes (13,17,28). Finally, we adjusted for psychological and childhood environment characteristics that potentially could have accounted for the buffering effect of PPCR, but none of these reduced the PPCR effect.

Unclear is how positive relationships with one's parents early in life buffer against the long-term health effects of childhood disadvantage. Nurturing parents may model coping strategies that facilitate their children's capacities for managing adversity and in turn attenuating physiological consequences (29). Positive relationships also may imbue children with the sense that the world is a safe place and other people can be trusted (30). Consequently, children may read less threat into their social worlds, resulting in a reduction in physiological wear and tear (3). Positive parent-child relationships also may enhance feelings of optimism, control, and emotional awareness, as well as promote the ability to develop stronger and more supportive adult relationships, all characteristics thought to buffer the negative impact of adverse experiences on physical health (31–33). These psychosocial effects may in turn affect the immune system's development in a manner that promotes subsequent resistance to viral infection, for example, by promoting stronger antiviral activity and better inflammatory regulation (7,12,34). In addition, nurturing parents may put more effort into reducing children's exposure to household toxins (cigarette smoke) and environmental pollutants that typically are associated with low SES. They may also help to promote better health practices such as a healthy diet, exercise, adequate sleep, and dental care, and prevent children from engaging in harmful practices such as smoking, excessive alcohol consumption, and use of illicit drugs, practices that may be maintained throughout the life course. However, there was no support for smoking, alcohol consumption, or physical activity in adulthood as mediators of the association of low childhood SES and cold susceptibility in this sample (12).

These data suggest the possibility that health disparities associated with low childhood SES may be mitigated by interventions that are designed to strengthen relationships between children and their primary caregivers (35). They also suggest the importance of following up on the validity of the proposed immune and behavioral mediators that themselves may turn out to be effective downstream targets of intervention.

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