Parenthood and Host Resistance to the Common Cold

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Objective: To determine whether parenthood predicts host resistance to the common cold among healthy volunteers experimentally exposed to a common cold virus. **Methods:** Participants were 795 healthy volunteers (age range = 18–55 years) enrolled in one of three viral-challenge studies conducted from 1993 to 2004. After reporting parenthood status, participants were quarantined, administered nasal drops containing one of four common cold viruses, and monitored for the development of a clinical cold (infection in the presence of objective signs of illness) on the day before and for 5 to 6 days after exposure. All analyses included controls for immunity to the experimental virus (prechallenge specific antibody titers), viral strain, season, age, sex, race/ethnicity, marital status, body mass, study, employment status, and education. **Results:** Parents were less likely to develop colds than nonparents were (odds ratio [OR] = 0.48, 95% confidence interval [CI] = 0.31–0.73). This was true for both parents with one to two children (OR = 0.52, 95% CI = 0.33–0.83) and three or more children (OR = 0.39, 95% CI = 0.22–0.70). Parenthood was associated with a decreased risk of colds for both those with at least one child living at home (OR = 0.46, 95% CI = 0.24–0.87) and those whose children all lived away from home (OR = 0.27, 95% CI = 0.12–0.60). The relationship between parenthood and colds was not observed in parents aged 18 to 24 years but was pronounced among older parents. **Conclusions:** Parenthood was associated with greater host resistance to common cold viruses. **Key words:** parenthood, influenza, rhinovirus, disease susceptibility, common cold.

OR = odds ratio; **CI** = confidence interval; **RV** = rhinovirus; **URI** = upper respiratory infection.

INTRODUCTION

Evidence on the potential role of parenthood in health has been provocative but inconsistent. Those with children at home report less happiness and life satisfaction (1,2), as well as more depression (3), anger (4), and anxiety (5) than those without children. In contrast, parents have lower mortality risk than nonparents, even after controlling for marital and socioeconomic status (6,7). Parenthood is also related to reduced suicide risk (8–11) and better cardiovascular health (12–14).

There is no evidence regarding the role of parenthood in the most prevalent physical diseases—upper respiratory infections (URIs). Parents with children in school or daycare are no doubt exposed to more respiratory viruses. Moreover, their host resistance could be suppressed by the enduring stress associated with increased economic and interpersonal strains of parenthood (15). On the other hand, parenthood could facilitate host resistance through increased exposure to pathogens resulting in acquired immunity or through the benefits of diverse social networks and support systems associated with school and extracurricular activities (16).

Here we use a prospective viral challenge design to assess the role of parenthood in host resistance among persons experimentally exposed to a virus. Healthy volunteers reported their parenthood status and were then intentionally exposed to either an influenza virus or one of three rhinoviruses (RVs). After viral exposure, they were observed in quarantine for either 5 days (for RVs) or 6 days (for influenza) and monitored for development of infections and illness. Analyses control for prechallenge immunity to the experimental virus (viral-specific antibody titers), study, sex, age, race, season, virus, education, body mass, employment status, and marital status. We also explored differences in the relationship between parenthood and colds according to parent sex and age, living arrangements (e.g., children living in the home versus out of the home), number of children, marital status, and employment status.

METHODS

Participants

The participants were healthy volunteers (n = 803) enrolled in one of three viral-challenge studies (16–18) conducted from 1993 to 2004. Sample sizes for the individual studies were as follows: Study 1 (n = 276), Study 2 (n = 334), and Study 3 (n = 193). Participants were recruited throughout the Pittsburgh, PA, metropolitan area via newspaper advertisements, other media, and community postings. All participants provided informed consent and received financial compensation for study participation. Study procedures were approved by the appropriate institutional review boards. Eight participants were excluded from analyses because they were missing data on at least one of the standard control variables. The remaining 795 participants were 52% female and aged 18 to 55 years (mean [standard deviation] age = 30.83 [10.1] years). The sample included 550 whites, 213 blacks, and 32 participants of other racial/ethnic backgrounds.

Procedures

The temporal sequence of the trials is outlined in Figure 1. At baseline, participants completed a telephone screening followed by an in-person health evaluation to assess study eligibility. The evaluation included blood analyses (complete blood cell count, blood enzymes, and human immunodeficiency virus), urinalysis, blood pressure readings, and a urine pregnancy test (females). Exclusion criteria included history of major nasal/otologic surgery, human immunodeficiency virus seropositivity, history of psychiatric or chronic physical illness, current use of regular medication for a chronic illness, abnormal blood or urinalysis results, pregnancy, and lactation.

Serum antibodies to the challenge virus were also assessed from the baseline blood draw using a microtiter neutralization assay (19) for RVs and a hemagglutination inhibition assay (20) for the influenza virus. To maximize infection

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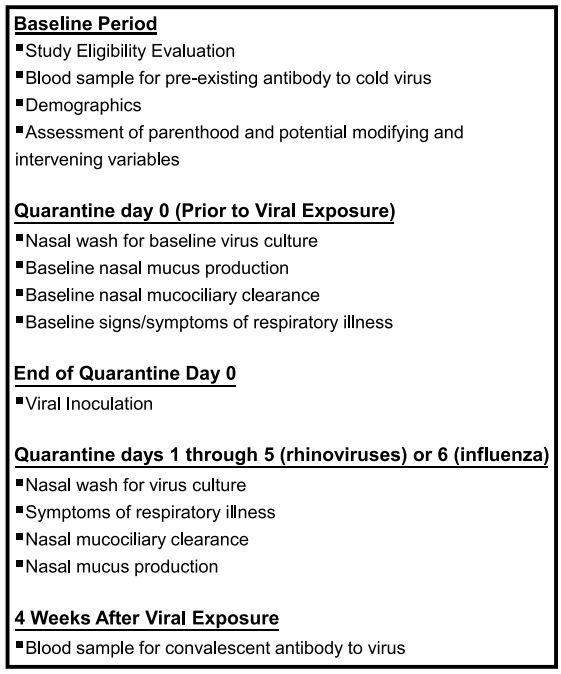

Figure 1. Temporal sequence of a trial.

rates in Study 3, only participants with baseline virus-specific antibody titers of four or fewer were included. Antibody titer was not used as a screening variable in Studies 1 and 2. Demographic data were also collected at baseline. At 2 to 8 weeks after screening, participants who passed the baseline health evaluation were quarantined in preparation for viral inoculation. During the first 24 hours of quarantine (before viral exposure), a nasal examination and nasal wash were performed. Participants were excluded at this point if viral pathogens were isolated from nasal wash samples or if they had nasal congestion, nasal discharge, mucosal edema, or URI symptoms. During this period, baseline respiratory symptoms and two objective signs of illness (nasal mucociliary clearance and nasal mucus production) were also assessed. Participants rated the 24-hour severity of the following eight respiratory symptoms on a scale from 0 (none) to 4 (very severe): congestion, runny nose, sneezing, cough, sore throat, malaise, headache, and chills (21). Nasal mucociliary clearance was used as a marker of nasal congestion and was measured as the time required for a dye administered to the nostrils to reach the nasopharynx (22). Mucus production was assessed by collecting used tissues in sealed plastic bags (22). The bags were weighed, and the weight of the tissues and bags was subtracted to calculate mucus weight.

At the end of the 24-hour quarantine (Quarantine Day 0), participants received nasal drops containing one of four viruses: RV21 (n = 129), RV23 (n = 106), RV39 (n = 522), or Influenza A/Texas/36/91 (n = 38). Disease expression in all four viruses is a common cold-like upper respiratory illness. Participants exposed to one of the three RVs were given 100 to 300 tissue culture infectious dose 50; those exposed to influenza were given a 10^5 tissue culture infectious dose 50.

After viral exposure, quarantine continued for an additional 5 days (RVs) or 6 days (influenza). During this time, participants were evaluated daily for URI symptoms, nasal mucociliary clearance, and nasal mucus production using the procedures used at baseline. Daily nasal wash samples were frozen and later cultured for the respective challenge virus using standard techniques (23). Four weeks after the viral challenge, an additional blood sample was collected and assayed for specific antibodies to the challenge virus. Serum antibody titers are reported as reciprocals of the final dilution of serum.

Parenting Status

At baseline, all participants were asked, "How many children do you have?" Responses were used to create a dichotomous parenthood variable (1 = parent, 0 = nonparent). Data on children's residential status (i.e., living in the home versus living outside the home) were collected only from the participants in Studies 2 and 3 (192 parents).

Personality as an Alternative Explanation

To control for personality variables that might explain both selection into parenthood and cold susceptibility, we measured extraversion and agreeableness using modified versions of Goldberg's adjective scales (24).

Outcome Measurement

The primary outcome measure was the development of a clinical cold after viral exposure. An individual was considered to have a cold on meeting *both* the criteria for infection and for illness expression. Participants were determined to be infected if the challenge virus was isolated in nasal secretions during any of the quarantine days after viral exposure or if participants experienced a fourfold or greater increase in specific antibody to the challenge virus from before exposure to 28 days after exposure.

We used two *objective* measures of illness: adjusted average daily mucus weights (in grams) and adjusted average mucociliary nasal clearance times (in seconds) (16). To maintain comparability across trials where there were differences in the number of days in which participants were quarantined (5 days for those exposed to RVs versus 6 days for those exposed to influenza viruses), we calculated *average* daily values for all continuous measures of objective illness. All daily measures were adjusted (Daily measure − Baseline measure) for baseline values. Adjusted values below 0 were scored as 0 (18). Participants met objective illness criteria if they had an adjusted average mucus weight of at least 2 g or an adjusted average mucociliary nasal clearance time of at least 7 minutes (16).

Standard Control Variables

The 11 standard control variables used in the study included prechallenge viral antibody titer to the challenge virus (titers 1, 2, 4, 8, 16, 32, and 64), age, self-reported race (white, black, or other), body mass index (kg/m^2), education (<2 years of college, 2 years of college/associate's degree, bachelor's degree or higher), sex, marital status (never married/never lived with a partner, currently married/living with a partner in a marriage-like relationship, separated/divorced/formerly lived with a partner in a marriage-like relationship/widowed), employment status (employed or not employed), season of study participation (spring, summer, fall, or winter), virus (RV21, RV23, RV39, or influenza A), and study (1, 2, or 3). Categorical variables were dummy coded.

Possible Intervening Variables

At baseline, we assessed psychosocial variables that could possibly link parenthood to cold susceptibility. The Perceived Stress Scale was used to assess the degree to which situations in life are perceived as stressful (25). The 10-item scale taps how unpredictable, uncontrollable, and overloading respondents find their lives. Tobacco and alcohol use were assessed via questionnaire. Current smokers were defined as those who smoked cigarettes, cigars, or a pipe on a daily basis. Participants were asked to quantify their average number of alcoholic drinks consumed per day separately on weekdays and weekends. Participants were considered to be drinkers if they indicated that they drank alcohol at least once per week. Sleep habits were measured using selected questions from the Pittsburgh Sleep Quality Index (26), a scale that asks respondents to evaluate their sleep habits over the past month. We focused on sleep duration (hours of actual sleep per night) and sleep efficiency (hours spent engaged in actual sleep each night divided by total number of hours spent in bed each night) because deficiencies in both had been found to be associated with increased risk for colds in earlier trials (16,27).

We used the Social Network Index (16) to evaluate social network diversity and size. Social network diversity refers to the number of types of social relationships (roles) in which the respondent has regular contact. The Social Network Index measures an individual's participation in 12 broad types of social relationships. Possible social roles include being a spouse, parent, parent-in law, child, close family member, neighbor, friend, coworker, employee, classmate, fellow volunteer, religious group member, and nonreligious group member. For our study, we excluded the spouse and parent (having a child) roles with a resulting 10 possible roles. Social network size is the total number of people

with whom the respondent has regular contact (i.e., at least once every 2 weeks) within these roles.

Statistical Analyses

Logistic regression was used to evaluate the relationship between parenthood and colds, adjusting for the standard controls. Odds ratios (ORs) and 95% confidence intervals (CIs) were used to estimate the ratio of risk to parents relative to nonparents.

To test for interactions with age, sex, marital status, race, and employment status, we used first-order cross-product terms for parenthood and these proposed modifier variables. Interaction terms were entered into individual regression equations with the corresponding main effects and control variables.

To test for intervening factors, we added the potential variables to the regression equation. Mediation was supported if the addition of these covariates substantially reduced the association of parenthood and colds.

RESULTS

Of the 795 study participants included in our analyses, 616 (77.5%) were infected. Of these, 70% shed virus and 47% seroconverted. Of the study participants, 255 (32.1%) developed clinical colds. When entered into the logistic regression model together, the following standard control variables were related to *decreased* cold incidence: younger age (β = 0.03, $p < .002$), higher preexposure antibody titers ($p < .001$), exposure to RV21 as opposed to RV39 (β = 0.60, $p < .03$), enrollment in Studies 2 and 3 compared with Study 1 (β = 0.81, $p = .003$ and β = 0.85, $p = .004$, respectively), high or low levels of education (compared with those with 2 years of college: β = 0.56, $p = .01$), and study participation during the spring (β = 0.88, $p = .007$), summer (β = 2.15, $p < .001$), or fall (β = 0.75, $p = .03$), compared with winter. The other standard controls were not related to colds.

Of the study participants, 337 (42%) were parents, with a mean (standard deviation) of 2.38 (1.43) children. Participants without children tended to be younger and more educated than those with children (Table 1). They were also more likely to be white and unmarried (Table 1).

In a regression analysis including the 11 standard control variables, parenthood was associated with decreased cold incidence (OR = 0.48, 95% CI = 0.31–0.73). Additional analyses of the association between parenthood and the two objective illness criteria found that parents had both lower adjusted average daily mucus weights (1.63 versus 2.90 g, $F[1,773] = 7.01$, $p = .008$) and lower adjusted average mucociliary nasal clearance times (3.31 versus 4.45 seconds, $F[1,773] = 8.86$], $p = .003$).

Parents also had more diverse social networks than nonparents (mean = 5.17 versus 4.61 social roles, $F[1,781] = 17.20$, $p < .001$). However, neither this potential intervening variable nor those unrelated to parenthood (perceived stress, tobacco/alcohol use, sleep efficiency, or sleep duration) altered the association between parenthood and colds when added to the equation.

A categorical measure of prechallenge antibody was included as a standard control variable in the regressions reported above. However, given the importance of exposure-related immunity as an explanation for the parent effect, we fit separate regressions (adjusted for the standard controls) *stratified by*

TABLE 1. Sample Characteristics Based on Parenthood Status (n = 795)

Variable	Parents	Nonparents	p
Age, M (SD), y	36.74 (9.29)	26.48 (8.38)	<.001
Sex, *n* (%)			
Women	189 (56.1)	231 (50.4)	.12
Men	148 (43.9)	227 (49.6)	
Race/ethnicity, *n* (%)			
White	183 (54.3)	367 (80.1)	<.001
Black	142 (42.1)	71 (15.5)	
Other	12 (3.6)	20 (4.4)	
Education, *n* (%)			
<2 y of college	239 (70.9)	226 (49.3)	<.001
2 y of college/associates degree	51 (15.1)	115 (25.1)	
Bachelor's degree or higher	47 (13.9)	117 (25.5)	
Marital status, *n* (%)			
Currently married/living with someone in a marriage-like relationship	116 (34.4)	170 (37.1)	<.001
Never married and never lived with someone in a marriage-like relationship	110 (32.6)	233 (50.9)	
Separated	33 (9.8)	12 (2.6)	
Divorced/formerly lived with someone in a marriage-like relationship	73 (21.7)	42 (9.2)	
Widowed	5 (1.5)	1 (0.2)	

M = mean; SD = standard deviation.

antibody titers. Parenthood was associated with decreased cold incidence among those with antibody titers of four or fewer (n = 519, OR = 0.46, 95% CI = 0.29–0.75) and those with antibody titers more than four (n = 276, OR = 0.37, 95% CI = 0.15–0.91).

We also considered the possibility that the relationship between parenthood and colds might be related to personality characteristics because several studies have found that increased extraversion and agreeableness were both associated with decreased cold risk (16,18,28). Adding extraversion and agreeableness as covariates, however, did not reduce the effect size observed between parenthood and colds (without covariate OR = 0.48, 95% CI = 0.31–0.73; with covariate OR = 0.46, 95% CI = 0.30–0.71).

Next, we explored how the number of children predicted colds using three dummy-coded categories (childless [contrast group], one to two children, three or more children). The standard controls were included as covariates in the model. Parenthood was associated with fewer colds among those with one to two children (OR = 0.52, 95% CI = 0.33–0.83), and those with three or more children (OR = 0.39, 95% CI = 0.22–0.70) compared to those without children.

We also explored how parent/child living arrangements were related to colds. We had data on parent/child living arrangements in Studies 2 and 3 (192 parents). Of these, 71 (37%) of 192 parents did not live with any of their children. We compared childless individuals (reference group) to those with only nonresidential children and those with at least one child living in the home, adjusting for the standard controls. Those with children at home were less likely to develop colds than those childless (OR = 0.46, 95% CI = 0.24–0.87). Having only nonresidential children was even more protective (OR = 0.27, 95% CI = 0.12–0.60).

We then examined differences in the relationship between parenthood and colds according to prechallenge viral-specific antibody level, sex, age, marital status, race, and employment status using separate regression models for each potential modifying variable. Of these variables, only age (continuous variable) demonstrated a statistically significant interaction with parenthood in predicting objective colds ($\beta = -0.053$, p = .009; Fig. 2). Using tertiles of age and adjusting for the standard controls, we found that parents aged 37 to 55 years (n = 177) were less likely to develop colds than nonparents in the same age group (n = 70, OR = 0.28, 95% CI = 0.14–0.57). This was also true for parents aged 24 to 36 years (n = 120) when they were compared with nonparents in the same age group (n = 150, OR = 0.37, 95% CI = 0.18–0.74). Rates of colds among younger parents (n = 40, aged 18–23 years) were not different from those of the younger nonparents (n = 238, OR = 1.79, 95% CI = 0.66–4.82).

Finally, separate analyses indicated that the association between parenthood and colds in the entire sample was not attributable to variation in infection rates (OR = 1.07, 95% CI = 0.64–1.79). Instead, it was due to differences between parents and nonparents in the expression of illness among infected participants (n = 616, OR = 0.44, 95% CI = 0.28–0.68).

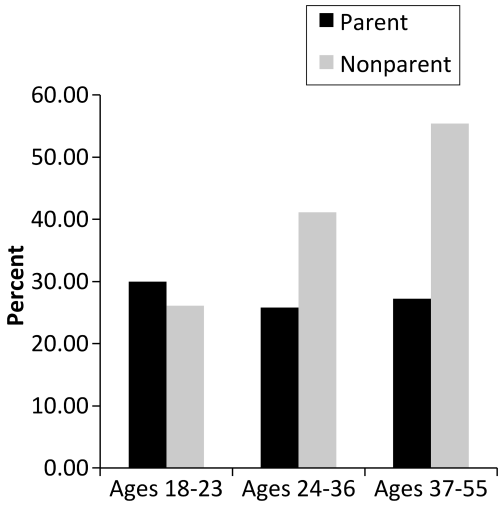

Figure 2. Unadjusted percent of participants who developed clinical colds by age and parenting status.

DISCUSSION

We found that parenthood predicted a decreased probability of colds among healthy individuals exposed to a cold virus. This association was independent of prechallenge viral-specific immunity (viral antibody titer to the challenge virus), with similar relations between parenthood and colds occurring among people above and below median antibody levels. The association of parenthood and colds was also independent of age, race, body mass index, education, employment status, sex, season of study participation, virus type, and which of three studies participants were enrolled in. Importantly, it was also independent of marital status and stable social traits. These are important because married people are more likely to have children, and marriage has consistently been associated with less morbidity and mortality (29,30). Similarly, extraversion and agreeableness are stable social traits that could plausibly select people into becoming parents and themselves have been associated with increased resistance to upper respiratory infectious illness (18).

Instead of self-reported symptoms, we used objective markers of illness in defining colds (16). In this way, we were able to avoid associations that could be interpreted as biases in how parents report physical symptoms. However, analyses using the standard (modified Jackson) criteria (21,23) for colds based on symptom scores (data not reported) yield the same conclusions.

Our results also indicate that risk decreases as the number of children increases. This assertion is limited, however, because we had few participants with greater than three children to provide a clear idea of what happens in very large families. There is, however, a clear increase in protection from one to two children to three or more children, suggesting that whatever parenthood provides is not derived from just being a parent but from resources provided by individual children. Further, the protective effect of parenthood was observed among both

parents whose children lived at home and those whose children lived away from home. The lack of difference here suggests that daily and intensive contact with one's children is not critical to the protective effect of parenthood. Alternative possibilities include the influence of parenthood on the feelings of purpose in life, emotional experiences, or whatever resources children might provide in less numerous interactions.

We found no statistical interactions between parenthood and prechallenge antibody level, parent sex, marital status, race, or employment status in predicting colds. However, we did find that the relationship between parenthood and colds varied according to parent age, with parenthood protective for those in their mid-20s and older. It is possible that the youngest parents may be unready psychologically and economically to fulfill the parental role and hence do not accrue the benefits that older parents do (31–33). Younger parents are also likely to have younger children (child age not assessed here) who require more attention. Alternatively, as parents age, they may put more emphasis on the positive aspects of parenthood and less on the negative ones.

It is important to note that older parents have more children and have fewer children living at home. Consequently, while each may contribute individually to host resistance, the substantial contributions of parent age, number of children, and residency to disease susceptibility may be due partly or entirely to their conjunction.

We found no behavioral explanations for why parenthood was associated with fewer colds. Although parents had more diverse social networks than nonparents do (although marriage and children were not included in calculating diversity), this factor could not account for the association of parenthood with colds. Perceived stress, tobacco use, alcohol use, and sleep habits also could not account for the relation. It is possible that parenthood is associated with relevant behavioral factors not mentioned here such as loneliness or depressive symptoms, or positive emotions, purpose in life or life satisfaction. A positive emotional style predicts greater host resistance among individuals experimentally exposed to common cold viruses (34). Loneliness (35–37) and depression (38) have been associated with the dysregulation of immune response, and purpose in life and life satisfaction have been linked with enhanced immune function (39). Hence, it is possible that one or more of these untested pathways could account for the protective effect of parenthood.

Whatever the behavioral pathway, greater risk for colds among nonparents was not attributable to an increased risk of infection but instead to an expression of illness among infected participants. A possible pathway here is the release of cytokines in the nasal passage that affects triggering of symptoms (34). Local (nasal) cytokines have been found to mediate the association between psychosocial variables (e.g., stress, positive affect) and cold risk (34,40). Parenthood may similarly improve regulation of the cytokine response, increasing cold resistance.

As indicated earlier, the interpretation of our data as attributable to psychological or behavioral differences between parents and nonparents is dependent on the assumption that prechallenge immunity was adequately assessed. The antibody assays used for both RV (neutralization assay) and influenza (hemagglutination inhibition assay) assess the functional roles of serum immunoglobulins A, M, and G. We found a substantial effect of antibody levels (e.g., those with undetectable antibody were more than 13 times more likely to develop a cold than those with titers of 32–64). Although nasal secretory immunoglobulin A (not assessed here) could play a role, earlier works in RV trials indicate that it is highly correlated with serum antibody and does not predict above and beyond the serum markers (41–45). It is also possible that cell-mediated immunity could be affected differentially by parenthood and in turn account for the role of parenthood in susceptibility. To the best of our knowledge, there are no studies of T-cell responsiveness in RV infections. However, works on influenza viruses suggest that cellular immunity plays a major role in recovery but not in resistance to infection (46,47). Overall, it is plausible that cellular immunity may influence the outcomes in our study, but there is no hard evidence that it would predict susceptibility to colds above and beyond serum antibody levels or that it would be more (or differentially) sensitive to the exposures associated with being a parent than serum antibody levels.

Finally, because previous exposure to a virus results in quicker and greater antibody response on a subsequent exposure (anamnestic response), it is possible, for those with previous exposure, that the antibody levels we assessed before the viral challenge underestimate the available antibody to fight infection. Kinetic studies of antibody response to RVs find that this secondary response does not occur until at least 7 days after infection (45,48), too late to play a role in our study. However, the inclusion of antibody titer assessments at 5 to 6 days after challenge would have further helped to address parent-related exposure as an explanation for the beneficial role of parenthood.

Our study is consistent with a small literature on parenthood and physical health indicating a protective effect of parenthood. Although it is well established that social relationships and certain social roles (e.g., marriage (29,30), church memberships (49,50)) can be linked to a range of positive physical health outcomes, the role of parenthood in physical health has not been well explored. Interestingly, here we have controlled for marriage in evaluating the role of parenthood in health, but few marriage studies do the converse. That is, there is a possibility that the beneficial effects of marriage may be partly or wholly attributable to parenthood. Moreover, our results, although provocative, have left room for future studies to pursue how various aspects of parenthood (e.g., frequency of contact with children, quality of parent/child relationships) might be related to physical health and how parenthood could "get under the skin" to influence physical health.

This study has several strengths. Its prospective design enables us to rule out the possibility that the cold itself influenced participant reports of parental status. The 11 standard control variables were chosen to eliminate the possibility that the associations we found were attributable to their impact on both parent status and cold susceptibility. Particularly, we measured and controlled for preexisting serum antibody to the

challenge virus before viral inoculation, allowing us to substantially reduce the possibility that parents may demonstrate greater viral resistance simply because having children resulted in exposure to more viruses and hence a greater probability of previous virus-specific immunity. Because the sample was made up of volunteers who could take 6 to 7 days away from their families, the parents in this sample may represent a somewhat unusual group. However, this issue is attenuated by the fact that the association between parenthood and colds held among those without children living at home.

Parenthood has been hypothesized to have both positive and negative implications for health. Here we find only positive implications for susceptibility to the cold. The associations we report are substantial, all exceeding two-fold effects.

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