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Family poverty is associated with cytomegalovirus antibody titers in U.S children

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Abstract

Background—Early life environmental and psychological influences are thought to play an important role in the development of the immune system. Antibody response to latent herpesviruses has been used as an indirect measure of cell-mediated immune function but has seldom been applied to younger age groups.

Methods—We used data from the 1999–2004 National Health and Nutrition Examination Survey (NHANES) to test for an association between family poverty and continuous antibody response to cytomegalovirus (CMV) in U.S. children aged 6–16 (N= 2,226) using OLS regression.

Results—Poverty was significantly associated with increased antibody levels among seropositive individuals. The association between income and antibody levels exhibited a threshold effect, with additional income beyond the poverty line not associated with increased antibody titers.

Conclusions—Early life social factors such as family poverty could have detrimental impacts on the developing immune system, with potentially important consequences for later life health outcomes.

Keywords

CMV; poverty; socioeconomic status; health disparities; NHANES; immunity

Background

While socioeconomic differences in chronic disease are well established, little is known about the role of the immune system in creating and sustaining health disparities. Early life environmental and psychological influences are thought to play an important role in the development of the immune system (Coe & Laudenslager, 2007; McDade, 2005). Antibody response to latent herpesviruses is an indirect measure of cell-mediated immune function in

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response to a wide range of psychosocial stressors (Glaser, 2005). However, the majority of studies utilizing this measure have focused on older age populations, with little existing data at younger ages. A recent study of 155 adolescents in Wisconsin identified an association between early childhood stress in the form of physical abuse and institutionalization and elevated antibody levels to herpes simplex virus type-1 (HSV-1) (Shirtcliff, Coe, & Pollak, 2009), and previous work identified an association between traumatic life events and elevated Epstein-Barr virus antibody levels for girls but not boys among 205 adolescents in the Great Smoky Mountains Study (McDade et al., 2000). The current study expands on this work by using data from a large, nationally representative data set to test associations between family poverty and continuous antibody levels of cytomegalovirus (CMV) in U.S. children.

Cytomegalovirus (CMV) is a highly prevalent herpes virus that is often acquired early in life, with seroprevalence in the U.S. reaching almost 50% by age 30 and over 80% for those 60 and older (Staras et al., 2006). Seroprevalence estimates of CMV among children have been reported as 36% in the US population (Bate, Dollard, & Cannon, 2010). Infection is often asymptomatic but the virus is never completely eliminated from the host, and thereafter containment of the virus becomes an immune system priority (Koch, Solana, Rosa, & Pawelec, 2006; Stowe et al., 2007). CMV infection has been linked to several chronic diseases of aging including inflammation, cardiovascular disease, frailty, depression, cognitive outcomes, and mortality (Aiello et al., 2006; Aiello, Haan, Piece, Simanek, & Liang, 2008; Appels, Bar, Bar, Bruggeman, & de Baets, 2000; Itzhaki, Wozniak, Appelt, & Balin, 2004; Liu et al., 2006; Roberts, Haan, Dowd, & Aiello, 2010; Schmaltz et al., 2005; Sorlie et al., 2000). Recent work also suggests that CMV may be a driving force behind alterations in immune function known as immunosenescence (Koch et al., 2007; Pawelec, Derhovanessian, Larbi, Strindhall, & Wikby, 2009). As a persistent infection, CMV is continually captured, processed and presented to T cells leading to continual clonal expansion and contraction of the adaptive immune system. Over time this process leads to clonal exhaustion whereby CMV specific T cells are present but anergic, leaving fewer naÔve T cells to combat novel pathogens and making the elderly particularly susceptible to infectious diseases and cancer (Almanzar et al., 2005; Pawelec, et al., 2009).

Previous work has found socioeconomic differences in the seroprevalence of CMV in the U.S. for the period 1988–1994 for ages six and over (Bate, et al., 2010; Dowd, Aiello, & Alley, 2009; Staras, et al., 2006), suggesting different levels of exposure or susceptibility based on social factors. In addition to seropositivity, CMV antibody levels among seropositive individuals differed by levels of education and income in U.S. adults ages 25 and over (Dowd & Aiello, 2009). To our knowledge, no existing study has tested the association between socioeconomic factors and continuous CMV antibodies in U.S. children.

METHODS

Data

Data come from the 1999–2004 U.S. National Health and Nutrition Examination Survey (NHANES), a nationally representative, cross-sectional survey of the non-institutionalized U.S. population with oversamples of the elderly, non-Hispanic blacks, and Mexican Americans. NHANES is conducted annually and data are publicly released in two-year waves (1999–2000, 2001–2002, 2003–2004), providing interview, examination, and laboratory measures. For children under 15 years of age, a proxy interview with a parent was conducted. Additional details of the NHANES survey design have been published elsewhere (*Centers for Disease Control and Prevention (CDC). National Center for Health Statistics: "NHANES 1999–2000 Public Release File Documentation"*, 2002). The Institutional

Review Boards of the University of Michigan and Hunter College approved the current study. The final analysis sample consists of 2,226 children aged 6 (earliest available age for CMV data in NHANES) and over who were CMV seropositive and non-missing on other covariates of interest.

Measures

Outcomes—Because the model linking stress, suppressed cell-mediated immunity and a subsequent rise in CMV antibody titers only applies to seropositive individuals, our primary outcome was CMV antibody titer among seropositive children (38.1% of the weighted sample). Descriptive statistics for CMV positive compared to CMV negative children are shown in Table 1. CMV specific IgG was measured with an ELISA by Quest International, Inc., Miami FL. Sera with values near the ELISA cutoff (approximately 5.2% of total) were confirmed with a second ELISA assay by bioMerieux, Inc., Durham, NC. Optical density for the IgG test was measured in AU/ml which stands for Arbitrary units/ml (National Center for Health Statistics 2005).

Predictors—Family poverty status was coded as =1 if the family's reported income divided by the federal poverty threshold adjusted for family size was <=1, and 0 otherwise. Continuous family income was also considered, with income coded as the midpoint of the reported income bracket, using $103,727 for top-coded incomes over $65,000 based on median income in this category according to the Current Population Survey. Income was adjusted for inflation to the year 2000 using the Consumer Price Index and was log-transformed due to the skewness of the distribution. Race/ethnicity was classified as non-Hispanic white, non-Hispanic black, Mexican American, and other race (non-Hispanic white as the reference category).

Covariates—Additional factors that might impact immune function and confound the relationship between family income and CMV antibody levels were considered. Since children who are immigrants may have encountered a different pathogen environment in utero and early life, we include an indicator=1 if the child was born outside of the U.S., 0 otherwise. Exposure to environmental tobacco smoke may be deleterious to a child's immune system, therefore we included an indicator for household smoking (=1 if the parent reported a smoker in the child's household; 0 otherwise). An increase in adipose tissue has been shown to alter certain immune parameters, particularly expression of inflammatory cytokines such as Il-6 (Visser, Bouter, McQuillan, Wener, & Harris, 2001; Wellen & Hotamisligil, 2005). BMI was calculated as (kg/m^2) from measured height and weight during the exam, and the continuous BMI measure was included as a covariate. Pre-natal undernutrition reflected in low birth weight may be associated with immune function later in life (McDade, 2005). Low birth weight was coded as 1 if the child was born weighing less than 2500 grams, =0 otherwise. An indicator for recent illness was coded =1 if the parent reported that the child had any of the following illnesses in the 30 days prior to being interviewed: head or chest cold, stomach or intestinal illness, flu, pneumonia, or ear infection. White blood cell count was included as a continuous measure reflecting current immune activity and as a marker of unreported acute infection or illness. An indicator for respiratory problems was coded =1 if the child currently had asthma, had coughed every day for 3 months straight during the past year, currently had bronchitis, or had wheezing or whistling in his or her chest during the past year. Controls for gender and age were also included.

Although there are no available data on traditional markers of stress in the NHANES publically available dataset, we were able to identify a measure of food insecurity, which could provide some information on household exposure to socioeconomic stressors among

those living in poverty. We examined whether household food insecurity was associated with CMV seropositivity and with CMV antibody levels. To examine household food insecurity, we included an indicator of 1 if the child's household reported being food insecure with hunger or food insecure without hunger and 0 otherwise.

Statistical Analyses

All analyses were performed using STATA (version 10.1, College Station,TX). Sample means for all variables were calculated by CMV serostatus, using Pearson's chi-squared to test for significant differences by serostatus status for categorical outcomes and Wald tests for continuous outcomes.

Next, we conducted ordinary least squares (OLS) regressions to describe the association of each covariate with CMV antibody titers. The first model tested the association of family poverty status and food insecurity with CMV antibody levels adjusting for age, gender, race/ethnicity, foreign-born status, and survey year. The second model proceeded to adjust for the additional physical risk factors, including BMI, low birth weight, recent illness, white blood cell count, whether the child has asthma or chronic respiratory problems, household smoking, and food insecurity. Model specifications included tests for both linear effects of income as well as non-linear/threshold effects, and final models include only threshold effects (poor v. non-poor). In the final set of analyses, we stratified by age (6–10 years v. 11–16 years) to test whether nonlinear effects may be accounted for by accumulation of exposure to income related stressors.

RESULTS AND DISCUSSION

Summary statistics are presented in Table 1. Mean age of the children in the study sample was 10.9 years (range 6 to 16 years). Table 2 presents results from OLS models of continuous CMV antibody response. Poverty status was significantly associated with higher CMV antibody levels among seropositive children. Blacks had marginally significant higher levels of CMV antibody levels compared to whites in Model 1 (p=.074). Males had significantly lower antibody titers compared to females. BMI was the only physical risk factor significantly associated with antibody levels; higher BMI was associated with higher antibody levels. Additional models tested for continuous as well as non-linear effects of income by quartile (available upon request). In models with continuous log income, increasing income was not associated with antibody levels. Examining food insecurity as a measure of household stress exposure, showed a significant association with seropositivity to CMV but not with CMV antibody level (see Tables 1 and 2). In Table 3 we stratified by age (6–10 years v. 11–16 years) to test whether the children's nonlinear income antibody function found in our analysis was due age-related accumulation of exposure to income related stressors. Table 3 shows that the relationship between poverty status and higher CMV antibody levels was statistically significant only among older children upon stratification.

Our results are consistent with previous work that found associations between income, level of education, and race/ethnicity and CMV antibody levels in U.S. adults (Dowd & Aiello, 2009). In contrast to the adult study, we found a non-linear association whereby income below the poverty line was associated with higher antibody levels, but additional income beyond the poverty line was not. It is possible that for adults, SES represents a more cumulative measure of disadvantage in which stressors have a longer window of opportunity to translate into physiological dysfunction, whereas more extreme material and psychological hardship is needed to detect detrimental physiological effects in children. Our tests of age stratification supports the hypothesis that exposure to poverty may become more

detrimental to the immune system, as measured by CMV antibody levels, when children are exposed to socioeconomic stressors for longer periods of time over the life-course.

While the observed differences in CMV antibody levels by poverty status in children may be relatively small, over a lifetime these differences could have an important impact on health and aging. CMV has been called the "driving force" behind age-associated alterations to the T-cell immune system, and the cost of constant CMV immunological vigilance throughout life may be high (Koch, et al., 2007). In ageing populations, late-stage differentiated CD8+ cells tend to be present in large amounts in the elderly – some have termed this “memory inflation” predominantly associated with an accumulation of CMV-specific cells.(Pawelec et al., 2005) This large clonal expansion of CMV-specific CD8 T cells found in aging populations limits the capacity of the immune system to mount an efficient immune response (Trzonkowski et al., 2004). The reduction in the number of naÔve cells able to combat new antigens (Pawelec, et al., 2005) resulting from CMV-specific clonal expansion can directly hinder immune response in medically important ways, as observed in poorer immune responses to influenza vaccination among those with higher CMV IgG antibody levels (Trzonkowski et al., 2003). These effects may be especially relevant for older adults, for whom poor vaccine response is related to higher rates of clinical illness such as influenza infection (Gravenstein et al., 1994). Whether such differences are clinically important in children is not known, and future work should test whether increases in CMV antibody levels are associated with greater susceptibility to other infections or reduced immune response to vaccination in children.

Given the evidence for the role of CMV in diseases of aging, the differences in CMV antibody levels by poverty status in U.S. children points to one potential link between early life SES and later life health. Differences in CMV antibody levels among children were not accounted for by conditions such as asthma, exposure to environmental tobacco smoke, or recent acute infection, factors that might influence immune function. Nonetheless, the causes of differences in CMV antibody levels cannot be inferred from the existing cross-sectional data. Besides the aforementioned study of early childhood trauma and HSV-1 in adolescents, many studies have used herpesviruses, including CMV, as a marker of subclinical immune response to psychosocial stressors (Esterling, Antoni, Kumar, & Schneiderman, 1993; Glaser, 2005; Herbert & Cohen, 1993). Children with poverty-level income are more likely to experience psychological stressors such as violence, interpersonal conflict and marital disruption, as well as a more stressful physical environments characterized by crowding, noise, and substandard housing environments (Evans, 2004; Evans & English, 2002). Thus, it is possible that our results reflect the impact of psychological stress associated with poverty on down-regulation of the cellular immune response.

The majority of literature on childhood SES, stress and health outcomes has focused on the hypothalamic pituitary-adrenal (HPA) axis (Adam, 2007), with maternal glucocorticoid regulation during pregnancy and early childhood hypothesized to have important consequences for childhood immune function (Coe & Laudenslager, 2007). The current results are consistent with the limited existing work that has looked directly at childhood socioeconomic status and other dimensions of immune function. For example, lower childhood SES was found to be associated with decreased resistance to upper respiratory infections in adulthood (Cohen, Doyle, Turner, Alper, & Skoner, 2004), and a pro-inflammatory phenotype in adolescence (Miller & Chen, 2007).

There are several limitations to this study. It is possible that increased levels of CMV IgG specific antibodies may indicate recent primary infection. There was no association between household size and CMV antibody levels in our sample, which is considered an important

surrogate marker of exposure to infection and increased likelihood of transmission (Fowler & Pass, 2006; Staras, et al., 2006). CMV IgM antibody, which would indicate recent infection or reinfection, was not measured in this sample. Our sample of seropositive children are very different than the children that are seronegative in terms of their socioeconomics, race/ethnicity and likely other health behaviors and exposures that were not available in the NHANES dataset. This may have limited our ability to detect differences in antibody levels by SES due to less variation in both SES and race/ethnicity within the seropositive category. NHANES has very few psychosocial stress measures that are publically available and measured in children. We attempted to look at a surrogate marker of socioeconomic stress in the household by using a measure of food insecurity. This measure was not statistically significantly associated with CMV antibody levels but did predict seropositivity. It is possible that food insecurity may increase susceptibility to CMV through poor nutrition and lack of vitamins but it did not lead to an increase in antibody levels among those who are seropositive. Other childhood stressors should be examined in relation to CMV antibody levels in younger age study populations in the US.

In sum, this study provided novel evidence of differences in an indirect marker of cell-mediated immunity in U.S. children by poverty status. Longitudinal studies of CMV antibody response in children would help to better elucidate the specific social and environmental mechanisms associated with these differences. The short and long-term health implications of such differences should also be investigated.

CONCLUSION

This study identified an association between family poverty and an indirect measure of cell-mediated immunity in a nationally representative sample of U.S. children. Among children seropositive for CMV, those with a family income below the poverty line had significantly higher antibody titers to CMV. We found no associations between race/ethnicity and antibody titer after controlling for SES. This work is consistent with recent work that identified an association between early childhood trauma and higher antibody titers to HSV-1 in adolescents (Shirtcliff, et al., 2009). Our findings suggest that more general early life social factors such as family poverty could also have detrimental impacts on the developing immune system.

Abbreviations

CMV	cytomegalovirus
BMI	body mass index
NHANES	National Health and Nutrition Examination Survey
OLS	ordinary least squares
SES	socioeconomic status

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Table 1

Descriptive Statistics, weighted: NHANES 1999–2004, Ages 6–16 (N=4667)

	Seropositive for CMV (n=2226)		Seronegative for CMV (n=2441)		
	Mean or proportion	**Linearized standard error**	**Mean or proportion**	**Linearized standard error**	***p***
CMV optical density	1.727	0.028	0.269	0.006	<.001
In poverty	0.333	0.017	0.189	0.014	<.001
Food insecurity	0.186	0.014	0.102	0.010	<.001
Family income	$40,680	$1,626	$51,547	$1,794	0.000
Age	11.048	0.096	10.852	0.071	0.086
Male	0.505	0.017	0.545	0.014	0.109
Race/ethnicity					
Black	0.193	0.019	0.124	0.013	<.001
Mexican American	0.189	0.019	0.086	0.014	<.001
White	0.481	0.027	0.700	0.022	<.001
Other race	0.137	0.019	0.090	0.015	<.001
Foreign born	0.101	0.012	0.034	0.005	<.001
Physical risk factors					
BMI	20.274	0.172	20.220	0.160	0.801
Low birth weight (<2500g)	0.071	0.008	0.061	0.005	0.267
White blood count	7.026	0.073	7.081	0.080	0.511
Recently sick	0.300	0.011	0.287	0.014	0.417
Asthma/chronic respiratory problems	0.140	0.011	0.148	0.010	0.515
Smoker in present household	0.221	0.018	0.264	0.015	0.061

Table 2

Ordinary Least Squares (OLS) regression models of CMV optical density from ELISA of seropositive children, NHANES 1999–2004, ages 6–16 (n=2226)*

VARIABLES	Model 1		Model 2	
	Coeff.	*p*	Coeff.	*p*
Age	0.011	0.131	−0.001	0.904
Male	−0.096	0.013	−0.087	0.026
Race/ethnicity (ref=white)				
Black	0.082	0.101	0.074	0.130
Mexican American	0.010	0.835	−0.019	0.720
Other race	0.084	0.278	0.063	0.433
Foreign born	−0.037	0.413	−0.031	0.513
In poverty	0.120	0.013	0.141	0.005
Food insecurity			−0.050	0.314
Physical risk factors				
BMI			0.014	0.000
Low birth weight (<2500g)			−0.010	0.855
Recently sick			−0.019	0.579
White blood cell count			0.003	0.748
Asthma/chronic respiratory problems			0.077	0.204
Smoker in present household			−0.091	0.074
Constant	1.713	0.000	1.560	0.000
R-squared	0.031		0.046	

*Models also adjusted for survey year.

Table 3

Ordinary Least Squares (OLS) regression models of CMV optical density from ELISA of seropositive children, NHANES 1999–2004, stratified by age groups (n=2226) *

	Ages 6–10 (n=551)		Ages 11–16 (n=1675)	
	Coeff.	***p***	**Coeff.**	***p***
Age	0.007	0.802	0.009	0.543
Male	0.053	0.498	−0.149	0.003
Race/ethnicity (ref=white)				
Black	0.041	0.628	0.094	0.117
Mexican American	−0.018	0.819	−0.012	0.843
Other race	0.064	0.707	0.049	0.512
Foreign born	−0.062	0.531	−0.009	0.884
In poverty	0.093	0.293	0.168	0.002
Food insecurity	−0.061	0.538	−0.069	0.272
Physical risk factors				
BMI	0.030	0.005	0.010	0.019
Low birth weight (<2500g)	−0.110	0.304	0.052	0.470
Recently sick	−0.039	0.650	−0.012	0.823
White blood cell count	−0.002	0.898	0.004	0.736
Asthma/chronic respiratory problems	0.107	0.258	0.058	0.427
Smoker in present household	0.004	0.972	−0.126	0.016
Constant	1.202	0.001	1.529	0.000
R-squared	0.050		0.061	

* Models also adjusted for survey year.