

Socioeconomic Status and Health: A Role for Immune System Mechanisms?

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September 2012

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Background

Low socioeconomic status (SES) has consistently been linked to poor health outcomes and higher mortality. This association has been found in different time periods, in locations around the world, in both genders, in all ages, across the entire range of SES, and for most health conditions (Adams et al. 2004; Phelan et al. 2004; Cutler et al. 2006). The physiological mechanisms behind the SES-health linkage, however, are not well understood (Dowd et al. 2007).

Immune system mechanisms such as chronic inflammation and age-related immune function decline (“immunosenescence”) are promising but under-studied explanations for how socioeconomic disadvantage might act physiologically to accelerate the onset of chronic disease and death (Dowd & Aiello 2009). Cytomegalovirus (CMV), a highly prevalent and generally asymptomatic herpesvirus that persists in the host throughout life, is hypothesized to play an important role in chronic inflammation and immunosenescence, which in turn leads to chronic disease and death (Roberts et al. 2010; Crumpacker 2010; Pawelec et al. 2005, Pawelec et al. 2010). Cross-sectional studies have found suggestive evidence for the role of inflammation and immunosenescence as mediators of the SES-health relationship. For example, lower income and educational attainment have been linked to increased CMV prevalence and higher antibody levels, indicating that the immune system is less able to keep the virus under control (Dowd, Aiello, et al. 2009; Dowd et al. 2007). A handful of longitudinal studies support the CMV-

immunosenescence connection, but they have largely ignored SES (Pawelec et al. 2009). Other studies support the CMV-inflammation link, as well as the connection between chronic inflammation and chronic disease, but don't focus on SES (Sansoni et al. 2008).

Psychosocial stress is believed to be a mediator of the low SES–poor health relationship, and may work (at least partially) through the immune function pathway. Low SES individuals experience a greater number of stressful experiences—such as negative events, traumas, and discrimination—and typically have reduced self-mastery and social support, two factors which can mitigate the negative impact of stressful events (Cutler et al. 2006; Thoits 2010). Chronic stress can lead to physiological dysregulation (broadly called allostatic load), including of the immune system (McEwen 2008). For example, stress has been linked to increased levels of antibodies of latent herpesviruses like CMV, indicating worse immune functioning (Dowd et al. 2007); the number of experienced stressors has been found to be cross-sectionally associated with general physiological dysregulation (Glei et al. 2007).

Despite this suggestive evidence, little longitudinal, population-based research has been conducted to study the pathways linking SES, stress, and immune function over time. The present study begins to address this question by evaluating whether SES is associated with immune function in a sample of adults in Detroit, Michigan. It will also determine whether this relationship is mediated by experienced stressors.

Data and Measures

Data are from the first two waves of the Detroit Neighborhood Health Study (DNHS). DNHS is an ongoing longitudinal study that is currently collecting the third and final wave of annual data, which includes SES, demographic information, biomarkers of immune function,

chronic disease diagnoses, and mortality outcomes. DNHS is representative of the adult population of greater Detroit, Michigan. It has 1,500 total participants of mean age 46.

DNHS contains several measures of SES (educational attainment, employment status, and income) and immune function (CMV, and the inflammation markers C-reactive protein (CRP) and Interleukin-6 (IL-6)). Experienced stressors are measured in response to survey questions determining whether respondents have experienced a number of specific stressors (including mental illness, problems with drugs or alcohol, divorce, inadequate healthcare, and assault).

Approximately 350 respondents have measures of CMV, CRP, and IL-6 in the first wave of DNHS. In the second wave, 500 respondents have the three immune function measures. Not all respondents were in both waves; 250 respondents have measures of CMV in both waves while 180 have measures of CRP and IL-6 in both waves.

In this preliminary analysis, educational attainment is categorized as less than high school, high school graduation or GED, some college, and college graduation or more. CMV is measured both with continuous antibody levels and based on seropositivity. CRP is measured 1) dichotomously as high (>3 mg/L) vs. normal, 2) dichotomously as very high (>10 mg/L) vs. normal, and 3) continuously in mg/L. IL-6 is measured continuously as pg/mL.

Preliminary Results

Initial results show some evidence of a relationship between educational attainment and immune function. In logistic regression models, the odds of CMV seropositivity in each wave declines as educational attainment increases (p-values on joint tests of all education categories are 0.09 in wave 1 and 0.03 in wave 2). Similarly, when CMV is measured continuously, the level of CMV antibodies is negatively associated with education in each wave (p-values on joint tests

are 0.02 and 0.03 in waves 1 and 2, respectively). The probability of having high (>3 mg/L) and very high (>10 mg/L) levels of the inflammatory marker CRP is lower with greater educational attainment in both waves, but the relationship is not statistically significant in either wave. There is not a significant relationship between education and continuous CRP levels. Levels of the inflammatory cytokine IL-6 are negatively associated with educational attainment (p-value on joint test = 0.03), though only in wave 2.

In subsequent work, I will consider income and employment as alternative measures of SES, and add demographic controls to the models. I will additionally test whether experienced stressors act as mediators of the relationship between SES and immune function.

Conclusion

In this study, I have begun to investigate whether there is a relationship between SES and immune function, and whether this relationship is mediated by stress. Preliminary results suggest that there is a link between higher educational attainment and better immune function, though it may depend on which biomarker is used to measure immune function.

A better understanding of the role that SES and stress play in immune function is crucial. Immune function may be an important pathway leading from social disadvantage to health disadvantage; additional research in this area could help the medical and health policy communities set priorities for addressing these health disparities.

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