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Inflammation among Children: Evidence of an Immigrant Advantage?

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Abstract

Past research on the immigrant health paradox suggests that children with immigrant parents may have a health advantage over those with U.S.-born parents, especially if the parent is a recent immigrant. Other research emphasizes the social and economic challenges children with immigrant parents face, in part due to disadvantaged social class and racial/ethnic positions. Underlying physiological changes due to chronic stress exposures among children in immigrant families is one potential health disadvantage that may not yet be apparent in traditional health measures. To explore these biological disparities during childhood, I use national biomarker and survey data from NHANES (N=11,866) to evaluate parent nativity and educational status associations with low-grade inflammation, indicated by C-reactive Protein (CRP), in children ages 2-15 years. I find that children with an immigrant parent, and particularly a low-education immigrant parent, have higher CRP, net of birth, BMI and other factors, than children with a U.S. born parent with either a low or higher education. Comparing children with low-educated parents, those with a foreign-born parent have higher predicted CRP. The findings from this study provide new evidence that children living in immigrant families in the U.S. may be facing higher levels of chronic stress exposure, as indicated by the increased risk of low-grade inflammation, than those with U.S.-born parents. The physiological changes related to increased risk of inflammation could set children in immigrant families on pathways towards mental and physical health problems throughout childhood and later in the life course.

Keywords

child health; immigrant paradox; C-Reactive Protein; stress

INTRODUCTION

Children living in immigrant families represent a quarter of all children under age 18 in the U.S. There has been continued growth in this population, increasing by 45% between 1994 and 2014, and most are U.S.-born children with an immigrant parent (Child Trends Data Bank, 2014). Although born in the U.S., children with foreign-born parents may face more stressful family conditions than those with native-born parents due to discrimination (Molina, Little, & Rosal, 2016; Ornelas & Perreira, 2011), lack of access to information and services (Berkman et al., 2004; Burgos, Schetzina, Dixon, & Mendoza, 2005; Huang, Yu, &

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Ledsky, 2006), and social or linguistic isolation (Mora et al., 2014; Shi, Zhang, van Meijgaard, MacLeod, & Fielding, 2015). These psychosocial stressors, in turn, may lead to physiological changes that occur with chronic stress exposure and pose risks to children's health and development (Shonkoff, et. al., 2012; Thoits, 2010).

Yet, there is little empirical evidence indicating whether stress exposure, and related physiological changes, is higher among children in immigrant families. In fact, some research suggests that children in immigrant families do better than would be expected based on their social class position, with fewer family- and neighborhood-level adversities (Slopen et al., 2016) and better health outcomes (Padilla, Hamilton, & Hummer, 2009) than similar children with U.S.-born parents. This may be due, in part, to immigrant supportive social networks or close neighborhood ties that protect immigrant children from the impact of social and economic stressors (Logan, Alba, & Zhang, 2002; Vega & Amaro, 1994). Given the increasing importance of this population, it is critical that we understand how the challenges of living in an immigrant family affect U.S. children's well-being.

To advance our understanding of immigrant children's stress exposure I empirically test whether children (ages 2-15 years) with immigrant and non-immigrant parents have distinct risks of low-grade inflammation. Low-grade inflammation – measured here by C-reactive protein (CRP) levels in the blood - is a biomarker of immune system dysregulation that can indicate chronic stress exposure (Black, 2002; Cohen et al., 2012). Psychosocial stressors have been linked with inflammation in adults (McDade, Hawkey, & Cacioppo, 2006; Ranjit et al., 2007) and children (Fuligni et al., 2009; Goosby, Malone, Richardson, Cheadle, & Williams, 2015).

In addition, low-grade inflammation in childhood has been associated with multiple health outcomes, including depression (Kim, Szigethy, Melhem, Saghaifi, & Brent, 2014; Gregory E. Miller & Cole, 2012) and cardiovascular risk factors (Cook et al., 2000; Slopen, Koenen, & Kubzansky, 2012) in children; and with increased risk of adult inflammation and chronic disease (Fagundes, Glaser, & Kiecolt-Glaser, 2013; G. E. Miller, Chen, & Parker, 2011). Thus, understanding potential differences in childhood inflammation disparities by parent nativity can inform both our understanding of stress exposure, as well as the risk for later health problems that may be associated with growing up in an immigrant family.

BACKGROUND

Immigration, Stress and Health

Much of our understanding of stress and health disparities among immigrant children has been informed by research related to the immigrant "health paradox." This is based on early findings that more socioeconomically disadvantaged immigrant groups have health outcomes similar to those for more advantaged white U.S.-born groups (Markides & Coreil, 1986). With respect to children, infants born to Hispanic immigrant mothers tend to have early life health advantages over those born to native mothers (Hummer, Powers, Pullum, Gossman, & Frisbie, 2007; Mendoza, 2009). Children with Mexican immigrant mothers, in particular, have also been found to have lower asthma rates in early childhood compared with children of U.S.-born Mexican mothers (Padilla et al., 2009). These immigrant health

advantages are often posited to be due to better health behaviors and health-promoting cultural practices that allow immigrants and their children to have better health than would be predicted by their disadvantaged social status (Markides & Coreil, 1986). However, empirical evidence does not always support these pathways (Padilla et al., 2009).

A second aspect of the immigrant health advantage is the theorized deterioration of health status with increasing time in the U.S. Although there is debate about whether adapting to the U.S. culture is the underlying reason for worsening health status among immigrants (Hunt, Schneider, & Comer, 2004), empirical evidence supports the idea that immigrant health in certain areas worsens over time and across generations, particularly among Latino immigrants (the group most widely studied with regard to the healthy immigrant effect) (Lara, Gamboa, Kahramanian, Morales, & Bautista, 2005). Of particular note is the tendency of self-reported health status to improve over time, while more specific health behaviors and outcomes often worsen. Thus, the use of biomarkers of health may be particularly useful in assessing immigrant health status to avoid confounding by self-reports.

Recent research provides support for immigrant children's health vulnerability rather than protected status. A recent meta-analysis found that despite evidence of an immigrant mortality advantage among Latin American/Caribbean-origin adults, the opposite was true for children: immigrant children had higher risks of mortality than their native-born counterparts (Shor, Roelfs, & Vang, 2017). Other studies have found higher risk of obesity (Baker, Rendall, & Weden, 2015; Lawrence, Mollborn, & Riosmena, 2016) and lower overall health status (Gelatt, 2016) among children with immigrant compared to U.S.-born parents. This indicates that immigrant children may lose early life protective factors as they experience their childhood years. One potential explanation for the increasing health risks among children in immigrant families may be the accumulation of stress from difficult social and economic conditions. Research indicates higher rates of food insufficiency, safety issues and medical insecurity among children with foreign-born than U.S.-born parents (Johnson-Motoyama, 2014). In addition, recent studies find that stressful conditions at home and in school contributes to mental distress and risky health behaviors among immigrant children/adolescents (Brietzke & Perreira, 2017; Choo, 2012; Lorenzo-Blanco et al., 2017).

In addition to inducing health behavior changes, chronic stress exposure can result in physiological changes that result in dysregulated biological systems. One measure of physiological dysregulation that may be induced by increased stress exposure is low-grade inflammation (Black, 2002; Cohen et al., 2012). In the case of inflammation, exposure to stress increases the production of cytokines, which, in turn, increase acute phase proteins (such as CRP) circulating in the blood (Black, 2002). The inflammatory response may be further enhanced when chronic stress has resulted in repeated activation of the autonomic nervous system and hypothalamic-pituitary-adrenal axis, which, in turn, become less effective in stopping the inflammatory process (Cohen et al., 2012). Detection of low levels of CRP (<10 mg/L) circulating in the blood indicates chronic, low-grade inflammation and a dysregulated immune system that may be due, in part, to psychosocial stress exposures (Black, 2003; McDade, 2007; McDade, Hawkey, & Cacioppo, 2006).

Empirical work on immigration and inflammation is limited. A recent study found that job instability and financial strain predicted higher inflammation levels in adult Mexican immigrants (Steffen, Walker, Meredith, & Anderson, 2016). Other research indicated higher levels of CRP in Chinese immigrant women reporting higher levels of acculturation stress (Fang, Ross, Pathak, Godwin, & Tseng, 2014). Regarding parent nativity, a small study found no association between mother's place of birth and CRP in 143 toddlers born in New York to Puerto Rican mothers (Kannan et al., 2013); however, broader conclusions cannot be drawn from this specific ethnic group finding. Finally, Schmeer & Yoon (2016) found that Los Angeles children with foreign-born primary caregivers had higher CRP than those with U.S.-born caregivers. Based on this limited body of research, it is not yet clear whether immigrant children experience a health advantage with respect to inflammation. This study addresses this question directly and by adding a social structural perspective, as discussed below.

Immigrant Social Status

Integrating a social structural view with immigration is essential in understanding immigrant children's social stressors and related physiological changes. Two of the most defining structural characteristics of individuals and families in the U.S. are race/ethnicity and social class. Immigrant parents become part of these structures upon arriving in the U.S. and their children are born into these social positions.

In terms of race/ethnicity, modern-day immigrant groups tend to be classified in minority racial/ethnic categories more than the historical white, European immigrants, and face challenges related to their minority racial position (Alba & Nee, 1997; Viruell-Fuentes, 2007). Reflecting these structural disadvantages, racial/ethnic disparities exist across health measures among U.S. children, with African American and Latino children often falling behind white children in numerous health outcomes (Mehta, Lee, & Ylitalo, 2013; Ogden, Carroll, Kit, & Flegal, 2012). Asian children, in general, do not show the same health disadvantage relative to white children (Mehta et al., 2013), possibly due to their higher social class position (although this may differ somewhat by specific country of origin). Social class varies substantially across racial/ethnic groups, with Latin American immigrant parents having the lowest and Asian immigrant parents having higher education upon arrival (Child Trends Data Bank, 2015).

Apart from SES disadvantages, racial/ethnic minority status is related to psychosocial stress associated with being in an "other" group and experiences of discrimination (Finch, Hummer, Kolody, & Vega, 2001; Viruell-Fuentes, 2007). These minority status stressors have been linked with physiological changes across various biological systems (Berger & Sarnyai, 2015; Brody et al., 2014; DeSantis et al., 2007), including inflammation in children (Goosby et al., 2015).

A second social structure related to immigrant status is social class position, which for children is often measured by their parents' education. Parent education has important consequences for all children, as it corresponds with neighborhood, school, housing and other conditions within which children live and develop (Child Trends Data Bank, 2015). More educated immigrant parents may not only have better work and income opportunities

—they may also be better at navigating educational and other opportunities for their children than low-educated immigrant parents. Lower social class families have long been associated with worse outcomes among children (Bradley & Corwyn, 2002; Goodman, 1999; West, 1997).

Increased exposure to psychosocial stress is one proposed pathway through which social class works to affect child health disparities, including inflammation. Initial research on the links between low social class and stress focused on parental stress leading to worse child outcomes (Conger et al., 1992; Elder & Caspi, 1988; A. P. Jackson, Brooks-Gunn, Huang, & Glassman, 2000). More recent research suggests that lower class family environments contribute to physiological changes that reflect children's direct exposure to stress, not just via parenting stress (Evans & Kim, 2007). Related to inflammation, parent education has been associated with higher levels of C-reactive protein (CRP) in children in two national-level studies (Dowd, Zajacova, & Aiello, 2010; Schmeer & Yoon, 2016b).

Research considering both parent immigrant and education status for child outcomes indicates that these aspects of immigrant families are related and may work together to affect child well-being. One study found that the higher risk of economic instability and poverty among children with immigrant parents was partially explained by the lower education level of immigrant parents (Van Hook, Brown, & Kwenda, 2004). Other research has found that pre-migration parent education level was the most important predictor of academic achievement among children with an immigrant parent (Pong & Landale, 2012). Finally, a study of the U.S. and United Kingdom found significant moderation of foreign-born parent status by parent education in assessing the health of 5-year old children. In the U.S. sample, among children with low-educated mothers, foreign-born parent status was protective of asthma, but the maternal immigrant advantage was not significant among more educated mothers. The opposite parent nativity effect was found for internalizing behaviors, which were higher among children with foreign-born mothers (in both the U.S. and UK); there was no significant difference by maternal educational status in the U.S. but education did moderate the effect in the UK (Jackson, Kiernan, & McLanahan, 2012). This suggests potential benefits and costs of parent immigrant status, and that effects may differ by child outcome and parent education level.

Study Contribution

This study builds on the above research by assessing how having immigrant parents is associated with low-grade inflammation in children. I focus on low-grade inflammation (CRP < 10mg/L) as both potential outcome of chronic stress exposure and an important precursor to later health problems. If living in an immigrant family is associated with more chronic stress in children, I would expect to find a positive association between having a foreign-born parent and children's CRP levels. However, if foreign-born parents are able to buffer their children from stress, possibly due to strong family, community, or transnational ties (Viruell-Fuentes & Schutz, 2009), there may be either a null or negative association between foreign-born parent status and children's CRP.

In evaluating the role of parent nativity in inflammation disparities I integrate parent education to determine how the risk of low-grade inflammation differs across children by

both their parent nativity and social class status. I also consider whether race/ethnicity accounts for some of the parent nativity/education status disparities in child inflammation. Given past research that has documented a potential health benefit of being born to immigrant mothers for Mexican American infants (Hummer et al., 2007), after assessing full-model effects, I stratify models by child race/ethnicity to assess whether Mexican American or other racial/ethnic groups show distinct associations between parent nativity/education status and children's CRP.

Importantly, I evaluate these associations net of prenatal and birth conditions (e.g. maternal smoking, low birth weight), which are lower among Hispanic and immigrant infants (Hummer et al., 2007), and may reduce inflammation in childhood. Other health factors considered are body mass index (BMI) and asthma, both of which have biological links with inflammation (Michelson, Williams, Benjamin, & Barnato, 2009) and differ by parent nativity status (Baker et al., 2015; Hillemeier, Landale, & Oropesa, 2015). However, unlike infant/prenatal health conditions, current BMI and asthma may be part of the biological pathway between stress and inflammation (Quinn, Kaufman, Siddiqi, & Yeatts; Shankardass et al., 2014). Thus, I test BMI and asthma as potential mediators of parent nativity status associations with CRP.

This study aims to inform research on child stress and health by improving our understanding of potential physiological effects of stress occurring among children in immigrant families in the U.S. The use of biological data (CRP) captures unseen and early development of physiological differences and potential pre-cursors to chronic health problems between children in immigrant and non-immigrant families. Further, the use of biomarker data reduces the biases created by parent reports and medical diagnosis, which are likely to differ across ethnic and immigrant groups. The integration of parent education with nativity status, and specific consideration of children's race/ethnicity, allow for further delineation of children who may be at higher risk of stress created by disadvantaged family contexts.

METHODS

Data & Sample

The data used here come from the physical health exam and interview data collected through the continuous National Health and Nutrition Surveys (NHANES), a cross-sectional nationally-representative survey fielded every two years. I appended the data across the six waves where CRP was collected in children (1999 to 2010). Mobile labs were used to obtain blood samples from children that were then analyzed for the presence of high-sensitivity CRP, with a minimal detectable level of 0.1 mg/L. Values below the detection level were included in the analysis but considered censored, since the true values were unknown. Cases with CRP above 10 mg/L (3% of the sample) were dropped as potential cases of current or recent illness or injury to focus on low-grade inflammation (Black, 2002).

Household and parent-level data came from surveys of a household respondent for each child. For children under age 16, this was a parent. Thus, the sample was limited to children ages 2-15 to ensure the household respondent was the child's parent. Of these children, 33%

were missing CRP values and were dropped from the sample. Children missing CRP values were more likely to be young (mean age 6 vs 9 years), white, and have a U.S.-born and higher educated parent respondent. I dropped an additional 2,801 due to missing data on the independent variable. I was unable to impute these missing values because the models (using Tobit regression) could not be run with imputed data in Stata. Those missing independent variables did not differ in mean CRP or immigrant parent status from those with all independent variable data. The final sample size was 11,866. The subsample sizes and descriptive statistics by race/ethnicity are noted in Table 1.

Measures

Inflammation was measured as a continuous indicator of level of CRP in the blood, converted to mg/L, and ranged from 0.1-10 mg/L after dropping the cases above 10 mg/L. CRP was logged in the analysis to adjust for its skewed distribution.

Data from the household respondent interview were used to determine parent nativity and education status. One parent was selected as the respondent and no information was available about the other parent. For immigrant status, parent respondents were asked about their place of birth, categorized as born in the U.S., Mexico, or in another unspecified country. I dichotomized this variable to indicate parent nativity as foreign-born (1) or U.S.-born (0). No questions were asked about time in the U.S. or acculturation in the parent module. Health information (including CRP) was also unavailable for the parent respondent.

The parent respondent also reported his/her education level, which was categorized by NHANES as: less than 9th grade, 9-12th grade, high school diploma or GED, some college, and college degree. Following past research (M. I. Jackson et al., 2012), I created a dummy variable of low parent education, an indicator of low social class, classified as high school degree or less (1) or more than a high school degree (0). I then integrated the parent nativity and low education variables to create the following four parent status groups: immigrant, low education; immigrant, higher education; U.S.-born, low education; and U.S.-born, higher education.

In secondary models I stratified the sample by child race/ethnicity. Child race/ethnicity in NHANES was parent-reported and coded in the following categories: Mexican-American, other Hispanic, non-Hispanic black, non-Hispanic white, and other race (including multi-racial children).

The parent was also asked if the child was born in the U.S., Mexico, or another country, which was used to construct a dummy variable indicating whether the child was U.S.-born. Given that the vast majority of children were born in the U.S. (95%), I was unable to analyze this variable within parent nativity categories. It was included as a control variable.

At the child level, I also controlled for birth/prenatal conditions and recent illness. Birth conditions assessed included: maternal age at birth, whether the mother smoked during pregnancy, low birth weight (< 5.5 pounds) and high birth weight (> 9 pounds). Recent illness was assessed by three dummy variables based on parent reports that the child was ill in past several weeks with a cold, infection or stomach illness. In controlling for these early

life and recent health disadvantages, the analysis reduced the influence of infant health or recent illness to better isolate immigrant parent effects on low-grade inflammation that may reflect increased stress exposure during childhood.

Other control variables included in all models were: child age in years, child gender (male=1), parent respondent gender (male = 1), parent respondent age, household size, and whether the parent respondent had a marital, cohabiting or no partner (converted to dummy variable of no partner in the household), and NHANES wave dummy variables.

Two additional health measures were included as potential mediators between parent nativity/education status and CRP: body mass index (BMI) and asthma. BMI z-scores were calculated using CDC growth charts which standardize BMI by age and gender compared with a healthy reference population (Kuczmarski et al., 2002) and was included as a linear variable. Household respondents also reported whether they were ever told that the child had asthma, coded as a dummy variable.

Statistical Analysis

Due to the censored nature of the dependent variable, I used Tobit regression to test associations between parent nativity/education and log CRP. I weighted both the descriptive statistics and regression analyses with individual medical exam weights computed for the 12-year period to account for non-response and oversampling. I also adjusted all regression models for heteroskedasticity in the error term.

Because the dependent variable was logged the effects are best expressed as exponentiated coefficients for ease of interpretation. These exponentiated coefficients can then be expressed as percent change in CRP for a given unit change in the independent variable, where:

$$\% \text{ change in CRP} = (e^{\beta_1} - 1) * 100.$$

In reporting the results, I show the full sample results first, with models that sequentially included race/ethnicity and then BMI and asthma variables. This allowed for an assessment of how the associations changed when controlling for race/ethnicity, and whether BMI and asthma served as potential mediators of the associations. I then provide results from models stratified by race/ethnicity, that included all controls (BMI and asthma as well). I report significance up to $p < 0.05$ for the full sample and $p < 0.1$ for the sub-sample models due to small race/ethnicity sizes of some parent immigrant/education groups within the sub-samples.

Although not shown here, I tested models controlling for poverty and income status, which may be a pathway through which immigrant and low educated families might affect child inflammation. These models indicated that the results were robust to inclusion of household income or poverty category, none of which were significantly associated with inflammation when education and the other variables were included in the models. Additional cases are lost due to missing income data, thus the models reported here do not include household income.

I conducted sensitivity analyses by categorizing CRP into no inflammation versus low inflammation (>1 and >3 cut offs tested), as research indicates that these cutoffs correlate with moderate and high risk of metabolic and cardiovascular health problems in adults (Pearson et al., 2003; Ridker, 2003). These models, which allowed for imputed data, were run using multiply imputed data. Results were consistent with the Tobit model results, which are preferred due to the lack of a consensus about inflammation risk cut off levels among children.

RESULTS

Table 2 shows the results for the full sample of children, indicating a significant difference across parent nativity and education status groups, with the largest difference between children in low-educated, immigrant families compared with those in higher-educated, U.S.-born families (124% difference in predicted CRP). Children in immigrant families with a low-educated parent had significantly higher CRP than children in immigrant families where the parent had higher education by 54%. Comparing within low-parent education categories, children with a foreign-born had 73% higher CRP than those with a U.S.-born, low-education parent. The foreign-born parent disadvantage was not only found for lower-SES children: those with a higher-educated immigrant parent had 43% higher CRP than those with a higher-educated, U.S.-born parent. Comparing children with a U.S.-born parent, there was a predicted 29% higher CRP for those with a low education rather than high education parent. Children with a foreign-born, higher education parent were not statistically different in predicted CRP than children with low-educated, U.S. born parents.

Model 2 included controls for children's race/ethnicity. Although the associations between parent nativity/education status and CRP remained significant, they were reduced in size by controlling for race/ethnicity effects, particularly for children with immigrant parents. All of the significant differences across immigrant/education status categories remained significantly different in Model 2.

Model 3 further incorporated child BMI and asthma diagnosis into the models to assess the role of these chronic health conditions in accounting for inflammation disparities. Again, the effect sizes decreased across the parent immigrant/education status categories, but most remained statistically significant. The previously observed significant difference in CRP between children with low-educated and higher-educated parents in immigrant families became insignificant when accounting for the children's BMI and asthma diagnosis. Of these two chronic health conditions that may be worsened by stress, only BMI was significantly associated with CRP. Post estimation tests confirmed that BMI was a significant mediator of the associations between immigrant and U.S.-born parents in low-education families and CRP, but did not mediate the difference in higher-education families (as also evidenced by the increase rather than decrease in the coefficient on immigrant, higher education).

Table 3 shows the race/ethnic group stratified models. The models include all controls from Model 3, but they are not reported for brevity. As Table 3 shows, children with foreign-born, low-educated parents had higher predicted CRP than those with U.S.-born, higher educated

parents across various race/ethnic groups. Other Hispanic was the only group where we saw no difference in inflammation by parent status (Model 6). Mexican American, African American and white children with immigrant low-educated parents also had higher estimated CRP than children with U.S.-born low-educated parents. CRP was an estimated 23%, 42%, and 75% higher in Mexican American, African American, and white children (respectively) with an immigrant, low-educated parent compared to those with a U.S.-born, low-educated parent.

Only Mexican American children showed a significant difference in CRP between low- and higher-educated immigrant parents, with CRP an estimated 36% higher for Mexican American children with a low educated compared with higher educated immigrant parent. There was no difference in CRP for children who had a U.S.-born parent with a low versus higher education, except among white children. Among white children, those with a U.S.-born, low education parent had an estimated 19% higher CRP than those with a U.S.-born, higher educated parent. White children with a low educated immigrant parent had CRP 109% higher than children of the most advantaged group (U.S.-born, higher educated parent) (Model 9).

DISCUSSION

In this study I assessed low-grade inflammation, a biomarker measure of exposure to chronic psychosocial stress, among children with immigrant and U.S.-born parents. In doing so, I considered children's social class and race/ethnicity status, in addition to parent nativity, as critical to understanding the potential physiological consequences of chronic stress exposure. Attention to the intersection between immigrant status and social class was particularly important in capturing the potential double burden of children whose families face stressors related to both immigrant and lower social class status.

The regression results indicated that children with foreign-born parents with low education had the highest predicted levels of inflammation. When compared with the most advantaged group (children with a U.S.-born, higher education parent), these children had a predicted 124% higher CRP. Within social class, children with an immigrant parent were predicted to have significantly higher CRP, although the gap was larger between immigrant and U.S. parents with low education (73%) than between those with higher education (43%). The smallest significant difference was between children with a U.S.-born parent: those with a lower educated parent had 29% higher predicted CRP than those with a higher education. Children with a higher educated immigrant parent had no discernable advantage in terms of inflammation over children living in U.S.-born low (or high) education families. Although there is little literature within which to situate the magnitude of these effects, a recent study of adults indicates that a 25% difference in CPR levels is important and has clinical relevance (Liu et al., 2017). The CRP disparities found here, which are estimated to be substantially larger than 25%, may indicate clinically important differences in inflammation and related health-risks among children.

These full sample disparities were attenuated somewhat by controlling for child race/ethnicity and BMI z-score. The attenuation by race/ethnicity reflected the higher likelihood

that children with immigrant parents were also racial/ethnic minorities and may have experienced stress related to their race (in addition to family immigration and social class statuses) that contributed to their higher levels of inflammation. BMI, but not asthma, was a partial mediator of the effect of having a low-educated, foreign-born parent. Given that high BMI can be, in part, induced by exposure to psychosocial stress (Wilson & Sato, 2014), the mediating effect may capture an additional biological disadvantage of children's exposure to stress in lower-SES immigrant families.

When I stratified the sample by child race/ethnicity, I found that the results largely held across several racial/ethnic groups, including Mexican American, African American and white children. The "other Hispanic" and "other race" groups showed no significant differences, most likely due to small sample size. Notably, in none of the racial/ethnic groups did children in immigrant families have an advantage over children with U.S.-born parents with respect to inflammation, even when comparing within social class.

There are few other studies of inflammation in children with which to compare these results. One other study using data from the Los Angeles Family and Neighborhood Survey (L.A. FANS) found higher inflammation in children with foreign-born primary caregivers (almost all Latinos) living in Los Angeles (Schmeer & Yoon, 2016). In relation to the broader literature on immigrant child health, this study contrasts evidence of an immigrant health advantage found for birth and maternal-reported health measures among children born to immigrant mothers (Hendi, Mehta, & Elo, 2015; Hummer, Powers, Pullum, Gossman, & Frisbie, 2007; Mendoza, 2009; Padilla, Hamilton, & Hummer, 2009). However, the immigrant health disadvantage in inflammation is consistent with recent research that suggests children in immigrant families have higher risks of mortality (Shor et al., 2017), BMI (Baker et al., 2015; Lawrence et al., 2016) and worse global health status (Gelatt, 2016) than those with U.S.-born parents. The inflammation disadvantage may also be particularly relevant for second generation immigrant children (most of the immigrant children in this sample), consistent with the idea that immigrant health status tends worsen across generation and over time (Lara et al., 2005).

The stratified models further showed that the Mexican American immigrant disadvantage (the group where much of the immigrant health advantage has been found) is concentrated among low-SES immigrant children. This finding is consistent with a recent study that found parent acculturation stress combined with economic hardship increased stress (as measured by cortisol levels) and behavioral problems in preschool children from Latino immigrant families (Mendoza, Dmitrieva, Perreira, Hurwich-Reiss, & Watamura, 2017). This double burden was less relevant for inflammation in the other race/ethnic groups, where the predicted CRP levels did not differ between lower and higher SES immigrant children. This may be due, in part, to the relatively smaller sample sizes and smaller percent of immigrant children in the other race/ethnic sub-samples. However, the more similar-sized coefficients for children in the low-SES and higher-SES immigrant families suggests that immigrant status rather than SES may be more important for inflammation among the non-Mexican minority children.

This is also one of the few studies able to compare black children in immigrant and native-born families. The finding that African American children in low-SES immigrant families have higher CRP than those in low-SES, native-born families is in contrast to a recent study of child health among Black children in the U.S. Using National Health Interview Survey data, the researchers found an immigrant health advantage across multiple maternal-reported measures of child health status (Hendi, Mehta, & Elo, 2015). However, the Hendi et. al. sample included a substantial portion of foreign-born children, while, as previously mentioned, the sample used here does not. Further, since birth conditions, BMI and asthma were controlled for in these models, the results may indicate higher exposure to chronic stress among black children in immigrant families despite their health advantages in other areas. The relatively small difference in coefficient size between black children in high and low SES families indicates that their minority and immigrant status may matter more for inflammation (possibly due to discrimination and other social stressors) than their social class status.

In sum, the findings suggest a relatively large immigrant disadvantage in inflammation among children in the U.S., and that the disadvantage extends across racial groups (with the exception of “other Hispanics”). This finding contributes to recent evidence that the immigrant health advantage found in adults and infants may not extend to children (Shor et al., 2017). Further, the immigrant advantage in child health often differs across health outcomes. Immigrant disadvantages may be more pronounced in child health measures that are influenced by stress more directly, such internalizing behaviors (Jackson, Kiernan, & McLanahan, 2012), obesity (Wilson & Sato, 2014) and inflammation.

This study is not without its limitations. First, the race/ethnicity-specific findings should be viewed with some caution, particularly for the groups with smaller sample sizes. In addition, though the white and African American samples were large, their small percent of immigrant parents, especially when separated by educational status, meant the cell sizes were relatively small for comparing across these groups. Further, the 1% of white children with a low-educated immigrant parent may be a select group that has higher inflammation due to unobserved characteristics.

Also of concern is the potential for unobserved characteristics to account for the associations between children with immigrant parents and inflammation in the full sample. The data were limited in information about parents and in providing only cross-sectional information. Thus, I am unable to attribute causality to these findings and cannot speak to the selectivity of having immigrant versus nonimmigrant parents. However, because the results were opposite of the immigrant paradox, the pro-health selectivity of immigrant parents is less of a concern.

The use of a biomarker as a measure of past exposure to stress has disadvantages and advantages. The disadvantages are that psychosocial stress was not measured directly, and that stress is not the only reason for inflammation. Environmental pollutants, injury and illness may also contribute to higher levels of inflammation. Although I control for several health measures, environmental exposures in the home and elsewhere, which may be higher among some immigrant populations (Perla, Rue, Cheadle, Krieger, & Karr, 2015), may be

part of the observed association between living in an immigrant family and higher risk of inflammation in children. On the other hand, the inflammation biomarker has been substantiated in the literature as reflecting chronic stress exposure (Black, 2003; McDade et al., 2006) and is not biased by parental reports that could differ by immigrant status and education. Using a biomarker outcome also has the advantage of capturing physiological conditions that may not be apparent to parents or doctors but could lead to health problems later in childhood and adulthood.

Finally, I did not have data on more than one parent or other household members. Thus, some of the immigrant families may have included a U.S.-born parent and the U.S.-born families may have included an immigrant parent. This may have resulted in an underestimation of the disparities between children in fully-immigrant versus fully-native families. Further, although I controlled for child nativity, there was not a large enough sample of foreign-born children to compare foreign and U.S.-born children within immigrant families.

Notwithstanding these limitations, the findings from this study provide new evidence that children living in immigrant families in the U.S. may develop physiological disadvantages related to chronic stress and other exposures. Chronic inflammation has been associated with multiple health outcomes in children, including depression (Kim, Szegedy, Melhem, Saghaei, & Brent, 2014; Gregory E. Miller & Cole, 2012) and cardiovascular risk factors (Cook et al., 2000; Slopen, Koenen, & Kubzansky, 2012). Most recently, elevated CRP has been linked lower cognitive abilities in children (Cullen et al., 2017), which may contribute to later developmental disadvantages. This suggests that a large, and growing, percent of children in the U.S. may be exposed to “hidden” health and development risks related to living in an immigrant family. Given the current and future direction of U.S. immigration rhetoric and policies, it is likely that stress levels will only increase over time in immigrant families, with implications for future child health disparities.

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Weighted descriptive statistics of full sample and sub-samples by child race/ethnicity. NHANES 1999–2010. Children ages 2–15 years with C-Reactive Protein (CRP) 10mg/L.

Table 1

Variables	Mean or % of sample					
	All	Mexican American	Other Hispanic	African American	Other race	White, non-Hispanic
CRP (mg/L)	0.84	1.14	1.10	0.92	0.80	0.73
<i>Parent nativity, education status</i>						
Immigrant, low education	12%	54%	35%	5%	13%	1%
Immigrant, higher education	8%	9%	25%	5%	27%	4%
U.S.-born, low education	34%	21%	22%	52%	28%	34%
U.S.-born, higher education	47%	16%	18%	37%	32%	60%
<i>Child characteristics</i>						
White, non-Hispanic	58%					
Mexican American	14%					
Other Hispanic	6%					
African American	14%					
Other race	7%					
Child BMI z-score	0.46	0.64	0.67	0.60	0.21	0.39
Ever told child has asthma	15%	11%	21%	21%	17%	14%
Child U.S.-born	95%	87%	85%	97%	89%	98%
Age in years	9.9	9.5	9.7	9.7	9.7	10.0
Male	52%	51%	50%	50%	52%	53%
Recent cold	23%	26%	26%	23%	20%	22%
Recent infection	5%	7%	7%	5%	8%	5%
Recent stomach illness	9%	8%	10%	8%	12%	10%
<i>Prenatal/birth conditions</i>						
Mother's age at birth	27.0	25.4	26.3	24.7	27.3	27.9
Mother smoked when pregnant	17%	6%	10%	15%	11%	22%
Child born low birth weight	8%	8%	6%	13%	7%	7%
Child born high birth weight	13%	13%	11%	8%	10%	16%
<i>Household characteristics</i>						

Variables	Mean or % of sample					
	All	Mexican American	Other Hispanic	African American	Other race	White, non-Hispanic
Parent male	51%	55%	44%	27%	51%	56%
Parent age	39.5	37.9	39.3	38.1	39.8	40.2
Household size	4.52	5.10	4.71	4.51	4.53	4.36
Parent no partner	26%	22%	31%	59%	20%	19%
N	11866	3792	829	3191	610	3444

Table 2

Associations between parent status and logged CRP from Tobit regression models. NHANES 1999–2010. Children ages 2-15 years with C-Reactive Protein (CRP) ≥ 10 mg/L. N=11866. Coefficients and standard errors shown.

Variables	Model 1	Model 2	Model 3
<i>Parent nativity and education status</i>			
Immigrant, low education	2.24 ^{**ab} (0.14)	1.57 ^{**ab} (0.12)	1.43 ^{**a} (0.099)
Immigrant, higher education	1.43 ^{**} (0.13)	1.24 [*] (0.12)	1.30 ^{**} (0.12)
U.S.-born, low education	1.29 ^{**} (0.072)	1.23 ^{**} (0.070)	1.17 ^{**} (0.061)
U.S.-born, higher education	REF	REF	REF
<i>Child race/ethnicity</i>			
Mexican American	—	1.74 ^{**} (0.11)	1.43 ^{**} (0.082)
Other Hispanic	—	1.64 ^{**} (0.15)	1.31 ^{**} (0.11)
African American	—	1.28 ^{**} (0.072)	1.07 (0.056)
Other race	—	1.01 (0.11)	1.07 (0.11)
White, non-Hispanic		REF	REF
<i>Child health</i>			
Child BMI z-score	—	—	1.87 ^{**} (0.039)
Ever told child has asthma	—	—	1.08 (0.063)
<i>Control variables</i>			
Child U.S.-born	1.13 (0.11)	1.13 (0.11)	1.06 (0.094)
Age in years	1.04 ^{**} (0.0066)	1.04 ^{**} (0.0066)	1.03 ^{**} (0.0063)
Child male	0.87 ^{**} (0.040)	0.87 ^{**} (0.040)	0.87 ^{**} (0.037)
Recent cold	1.83 ^{**} (0.096)	1.80 ^{**} (0.094)	1.84 ^{**} (0.093)
Recent infection	1.37 ^{**} (0.13)	1.34 ^{**} (0.13)	1.20 [*] (0.10)
Recent stomach illness	1.19 [*] (0.093)	1.19 [*] (0.094)	1.13 (0.083)
Parent male	1.05 (0.057)	1.07 (0.058)	1.10 (0.056)
Parent age	1.00 (0.0028)	1.00 (0.0029)	1.00 (0.0028)
Household size	0.99 (0.018)	0.98 (0.018)	1.01 (0.017)

Variables	Model 1	Model 2	Model 3
Parent no partner	1.27 ** (0.074)	1.20 ** (0.073)	1.21 ** (0.067)
Mother's age at birth	0.99 ** (0.0043)	0.99 * (0.0043)	0.99 (0.0041)
Mother smoked during pregnancy	1.33 ** (0.086)	1.41 ** (0.093)	1.19 ** (0.072)
Child born low birth weight	0.92 (0.074)	0.91 (0.074)	1.06 (0.080)
Child born high birth weight	1.01 (0.072)	1.03 (0.074)	0.90 (0.060)

**
p<0.01,

*
p<0.05. All models include wave dummy variables; not reported for brevity.

^aSignificantly different from U.S.-born, low education at p<0.05

^bSignificantly different from Immigrant, higher education at p<0.05

Table 3

Associations between parent status and logged CRP from Tobit regression models by child race/ethnicity, NHANES 1999–2010. Children ages 2–15 years with C-Reactive Protein (CRP) 10mg/L. N=11866. Coefficients and standard errors shown.

Variables	Model 4 Mexican American	Model 6 Other Hispanic	Model 7 African American	Model 8 Other race	Model 9 White, non-Hispanic
<i>Parent nativity and education status</i>					
Immigrant, low education	1.22 ^{ab} (0.099)	1.07 (0.19)	1.47 ^a (0.24)	1.80 ⁺ (0.60)	2.09 ^{***a} (0.59)
Immigrant, higher education	0.90 (0.10)	1.16 (0.23)	1.26 (0.19)	1.39 (0.36)	1.34 (0.27)
U.S.-born, low education	0.99 (0.094)	1.15 (0.23)	1.03 (0.074)	1.49 (0.38)	1.19 [*] (0.090)
U.S.-born, higher education	REF	REF	REF	REF	REF
N	3792	829	3191	610	3444

^{***} p<0.01,

^{*} p<0.05,

⁺

p<0.1. All models include control variables listed in Table 2, Model 3.

^a Significantly different from U.S.-born, low education at p<0.05

^b Significantly different from immigrant, higher education at p<0.05