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## Home sweet home? Home physical environment and inflammation in children

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### Abstract

The home environment includes important social and physical contexts within which children develop. Poor physical home environments may be a potential source of stress for children through difficult daily experiences. Using a sub-sample from the Los Angeles Family and Neighborhood Survey (N = 425), we consider how the home physical environment affects stress-related immune system dysregulation in children ages 3–18 years. Results indicated that children in poorer quality homes had higher inflammation (measured by C-reactive protein). The associations were particularly strong for younger children. We also found that part of the home physical environment association with CRP worked through increased risk of obesity for children living in low-quality homes. Future research should assess how home physical environments could be improved to reduce stress and improve health outcomes in children.

### Keywords

Stress; CRP; L.A.FANS; Housing; Poverty; Obesity

### 1. Introduction

A healthy and safe home environment is essential for promoting and protecting health and development during childhood. Children need stable, supportive social environments and access to resources within the home to enhance cognitive, emotional, and physical development (Bronfenbrenner, 2001). Alternatively, unstable, noisy, chaotic home environments have negative effects on children's health (Dush et al., 2013) and development (Evans, 2003; Evans and English, 2002). Low-income children are more likely to face poor quality home environments, across multiple domains, than their wealthier counterparts (Bradley et al., 2001; Evans and English, 2002; Holupka and Newman, 2011); and, low-income parents are often forced to choose between healthy and affordable homes (Breyse et al., 2004). Thus, the home physical environment not only affects individual child well-being, but also is an important pathway through which socio-economic inequalities create child health and developmental disparities (Conley, 2001).

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One reason proposed for the negative effects of poor home environments on child well-being is through increased exposure to stress (Conger and Donnellan, 2007). Biological changes may occur if the body is exposed to chronic stress (repeated stress response over time), resulting in dysregulation of the neuroendocrine and immune systems; these systems may be particularly sensitive during the formative years of childhood and adolescence (Bauer and Boyce, 2004; Pervanidou and Chrousos 2012). Dysregulation of children's neuroendocrine and immune systems, in turn, is associated with poor health (Chen et al., 2006), cognitive (Keller et al., 2012), and socio-emotional (Evans and English, 2002) outcomes.

In this study, we consider how the home physical environment affects stress-related immune system dysregulation in children as indicated by increased low-grade inflammation levels (measured by C- reactive protein). We focus on the physical disorder in the home (e.g., hazards, crowding, dim lighting, clutter, uncleanliness) as a potentially important source of stress for children. Past research suggests important negative effects of the home physical environment on children's socio-emotional outcomes (Evans et al., 2001b), but it is not yet clear whether these experiences result in physiological changes and biological embedding of childhood disadvantage. We assess potential differences in the association between physical home conditions and inflammation by child age, building on past evidence of age differences in SES associations with child health (Chen et al., 2006; Chen et al., 2002). We also consider the mediating role of obesity, given that low-quality home environments may contribute to obesity through decreased opportunities for activity and poor eating or sleeping habits; and higher body mass index (BMI), in turn, is associated with higher inflammation levels in children (Dowd et al., 2010; Visser et al., 2001).

We test these research hypotheses using data from the Los Angeles Family and Neighborhood Survey (L.A.FANS). The data are representative of households in L.A. County, a geographical area that includes the least affordable housing market of all metropolitan areas in the nation (California Housing Partnership Corporation, 2014). With increasing housing costs, low-income families in Los Angeles are forced to overspend and overcrowd with little hope for homeownership. It is no coincidence that the city with the least affordable housing market also has the second highest rate of overcrowded households among major U.S. cities, with 25.6% of all families living in crowded quarters (O'Hara, 2002). The situation is worse for children; in 2013, nearly half of L.A. children (46%) lived in cramped homes compared to the state average of 28% and the national average of 14% (Kids Count Data Center, 2014). Other aspects of the home environment, such as safety and access to outdoor areas, are likely to be sacrificed within this expensive housing market, with implications for children's exposure to and ability to cope with chronic stress.

Our study provides new research on home physical conditions and child well-being, focusing on inflammation as an under-studied measure of exposure to chronic stress in children. The use of interviewer-rated home assessments and biomarker outcomes reduces biases related to parental reports. Of particular importance, we assess how associations differ by child age and when controlling for obesity. The findings from this study inform future research and policy related to housing and child well-being, and increase our understanding of how health inequalities begin during childhood.

## 2. Background

The household is a critical ecological context that affects children's health and psychosocial development (Bronfenbrenner, 1995). Compared to more distal environments, home conditions may be particularly relevant for child well-being due to the daily, repeated interactions between children and their home environments (Bronfenbrenner, 1995; Bronfenbrenner and Evans, 2000). Physical characteristics of the home are an important part of this context because they influence children's social interactions, health behaviors (sleeping, eating, physical activity), and exposure to environmental toxins (Evans, 2006). Social interactions may be impacted by the lack of adequate, organized space for members of the household to carry out daily activities.

A review of research indicates that crowded and inadequate housing increases tension, punitive punishment, aggression and conflict within the household (Evans, 2006). Physical disorder, such as lack of cleanliness and higher levels of clutter in the home, also may reflect social disorganization, with implications for children's well-being (Dunifon et al., 2004). Social disorder related to physical conditions in the home can, in turn, lead to increased psychosocial stress in children. Recent research found that emotional chaos in the home (various indicators of commotion within the home) predicted stress (abnormal cortisol responses) in young, low-income children (Lumeng et al., 2014). Other research indicates poor housing conditions are associated with increased psychological distress among mothers (Evans et al., 2000), another social risk factor that may be associated with child stress in homes with poor physical conditions.

In addition to social relationships, low-quality physical housing environments may affect children's health and stress-coping behaviors. Having an unsafe interior or exterior, no access to a yard or patio, crowding, and poorly-lit interiors reduce children's creative play, ability to move about, and outdoor playtime (Maitland et al., 2013; Marino et al., 2012). These conditions can increase sedentary behaviors and reduce children's ability to cope with the stressful environments (Wells and Evans, 2003). Research also suggests that more chaotic homes increase unhealthy, stress-related eating behaviors in young children (Lumeng et al., 2014). Crowded, inadequate and disorganized home environments also impact children's sleep habits, directly and indirectly through increased psychosocial stress; and, poor sleep quality may contribute to elevated inflammation (Quist et al., 2015). Thus, lack of adequate home physical conditions may impact behaviors that increase inflammation and risk of obesity in children (McCurdy et al., 2010).

Finally, the home physical environment embodies environmental toxins that can be found in the air (dust, noise), walls (mold, lead paint), floor (unsafe objects), and other areas within the home. Children with environmental toxins (including noise) in the home have been observed to have worse developmental, physical health, and behavioral outcomes (Cohen and Spacapan, 1984; Evans, 2006; Koger et al., 2005; Krieger and Higgins, 2002); and elevated noise has been associated with biomarkers of stress in children (Evans et al., 2001a; Hohmann et al., 2013).

Existing empirical evidence supports the importance of ensuring a high quality home physical environment for child health (Leventhal and Newman, 2010). In a study of four housing characteristics (housing type, cost, quality, and stability) poor housing quality most consistently predicted low-income children's emotional and behavioral problems and lower cognitive skills (Coley et al., 2013). Several studies indicate that household social and physical disorder (including hazardous, crowded, cluttered, and unclean homes) are associated with worse health in children (Breysse et al., 2004; Cutts et al., 2011; Delgado et al., 2002; Dush et al., 2013; Leventhal and Newman, 2010; Suglia et al., 2010).

Limited research also finds that home physical environments may be associated with increased levels of chronic stress in children. One study using an index of interviewer-assessed housing quality found that poorer quality home environments were associated with higher psychological distress (children's behavioral problems reported by parents) and learned helplessness in third through fifth graders, net of family income (Evans et al., 2001b). Another study of predominantly white children from low-income, rural communities found cumulative risk (including an indicator of substandard housing) to be associated with higher blood pressure and neuroendocrine markers of stress in children between the ages of 8 and 10 years (Evans and English, 2002).

## 2.1. Housing and inflammation

This study aims to assess how children's home quality is associated with a biomarker of chronic stress exposure that has not been extensively studied in children –low-grade inflammation. Inflammation is a normal part of the body's response to a threat, increasing the production of, among other things, C-reactive protein (CRP). CRP is a sensitive marker of inflammation and levels normally rise quickly in response to a physiological threat – infection or injury – and then decline soon after the threat is resolved (Dowd et al., 2010). However, low levels of CRP indicate a dysregulated immune system and increase the risk of chronic disease (McDade, 2012).

During childhood, inflammation has been linked with depression (Kim et al., 2014; Miller and Cole, 2012) and, in some studies, cardiovascular risk factors in youth (Slopen et al., 2012). Further, elevated inflammation in childhood may increase the risk for adult inflammation (Fagundes et al., 2013; Miller et al., 2011).

Exposure to psychosocial stress has been posited as a cause of low-grade inflammation and part of the biological embedding of social and economic disadvantage (Miller et al., 2011). Recent studies of U.S. children have found associations between elevated CRP and living in high-risk neighborhoods (Broyles et al., 2012), experiencing maltreatment (Cicchetti et al., 2015; Danese et al., 2011; Gonzalez, 2013), and living in low-income families (Dowd et al., 2010). Among children in Avon, England, externalizing behavior at age 8 was associated with higher CRP at age 10 (Slopen et al., 2013a); and adverse life events from early to mid-childhood were associated with higher CRP at ages 10 and 15, with some of the effects mediated by obesity (Slopen et al., 2013b). These suggest that risky social and economic conditions may result in systemic inflammation during childhood.

Importantly, research has found that everyday sources of stress, not just traumatic events, may have physiological implications for children. Adolescents with higher daily stressors (Fuligni et al., 2009) and those experiencing lower levels of positive affect (Chiang et al., 2015) had higher levels of CRP. Further, an intervention study of 11-year old rural African American children found that a program aimed at reducing psychosocial stress in children significantly decreased inflammation, in part due to improved parenting in the treated group (Miller et al., 2014). These studies indicate the potential for non-traumatic, chronic psychosocial stress occurring in the home environment to contribute to dysregulated immune systems in children and adolescents.

In this study, we hypothesize that a poor quality home physical environment (HPE) will be associated with higher stress-related inflammation in children through increased exposure to chronic psychosocial stress. Housing research suggests that the overall quality of the home environment, rather than subscales or individual indicators, are more important for studies of psychosocial distress (Evans et al., 2000). Following cumulative risk theory (Barocas et al., 1985) and past research (Bradley et al., 2001; Evans et al., 2001b), we assess how the cumulative number of risky HPE factors, rather than one indicator alone, influences children's inflammation levels.

Based on developmental theory (Bronfenbrenner, 1995), we also assess the role of child age in moderating the effect of the HPE on inflammation. Younger children are likely to spend more time at home and be less able to escape the threats that the physical environment presents than adolescents. Thus, a low-quality HPE may have a stronger effect on younger children's inflammation. On the other hand, if biological differences by HPE emerge due to the accumulation of stress over time, then HPE may be more strongly associated with elevated inflammation in older children. Past studies of the SES-health gradient among children have found that the association varies by age as well as the specific health outcome under analysis (Chen et al., 2002, 2006). Studies of household income and inflammation in children to date have found no significant differences by age (Dowd et al., 2010), but the effects of home conditions have not been explored.

Finally, we test if the associations between the HPE and children's inflammation are due to increased risk of obesity. We posit that low-quality HPE will be associated with higher obesity risk, as homes with unsafe exteriors or no outside space may reduce the chances for children to exercise. Further, disordered and unsafe interior home conditions may induce unhealthy eating and sedentary behaviors (i.e., TV watching and video games) as ways of coping with difficult home environments. We test whether obesity is a key pathway between the HPE and inflammation, since overweight children have higher levels of inflammation (Visser et al., 2001).

A recent review of research in this area found no studies of home physical characteristics and child obesity (Gundersen et al., 2011); however, one related study found cumulative risk exposure (including 3 indicators of poor physical conditions in the home as part of the broader index) was linked to weight change during childhood (Wells et al., 2010). Another study found various family stressors to be associated with increased risk of obesity in children and adolescents (Garasky et al., 2009). The results indicated no association between

housing stressors (moving to cheaper housing, moving in with others, sending a child to live elsewhere, or spending greater than 30% of income on housing) and the risk for childhood obesity, but home physical conditions were not considered (Garasky et al., 2009).

### 3. Methods

#### 3.1. Data

This study uses unique data from the Los Angeles Family and Neighborhood Survey (L.A.FANS), which includes measures of social and economic conditions at the household and parent levels, along with biomarkers in children. L.A.FANS was designed as a stratified probability sample of neighborhoods (census tracts), blocks within these neighborhoods, households within these blocks, and children and adults within these households (Sastry et al., 2006). Poor and very poor census tracts and households with children were oversampled. Wave 2 (2006–2008) includes the biomarker measure of inflammation –CRP obtained by licensed phlebotomists—in a random sub-sample of children ages 3–18. Wave 1 did not collect biomarkers and thus was not included in this study.

The analytical sample used in this study consists of children who participated in the blood spot collection (425 of the 600 randomly selected for participation) for whom sufficient blood was obtained (N = 414). Since CRP values above 10 mg/L are considered acute responses to infection or injury rather than a response to chronic stress (McDade, 2007), we limit our sample to children with CRP levels  $\leq$  10 mg/L (383 of the 414 cases). A further 30 cases were dropped due to missing independent variables resulting in a sample size of 353.

Descriptive and regression analyses were adjusted to account for unequal selection probability and the clustered survey design (Peterson et al., 2011). Weights were calculated by multiplying the wave 2 child weights by the blood spot specific weights to account for oversampling, attrition by wave 2 and non-response to the blood spot portion of the study. We applied the combined weights to the descriptive and regression results to provide analyses representative of children in L.A. County.

Table 1 below presents the weighted descriptive statistics for the analytical sample. The mean child age was 11 years and 22% of the children were obese based on BMI cutoffs by age and gender. Although the mean family income-to-poverty ratio was under 3 (less than 3 times the poverty line), it varied substantially (standard deviation = 3). Virtually all (96%) primary caregivers were mothers. Of these caregivers, 53% were immigrants, 63% were Hispanic, and 36% had less than a high school degree (Table 1).

#### 3.2. Measures

Inflammation is measured by the plasma equivalent concentration (in mg/L) of C-reactive protein (CRP) levels obtained from assaying reconstituted dried blood spots (McDade, 2007). Using a high sensitivity enzyme immunoassay, low levels of CRP can be detected in the blood. As previously stated, our outcome of interest is chronic, low-grade inflammation as a measure of physiological stress response in children. Thus, we limit our sample to children with CRP  $\leq$  10 mg/L. Although this cutoff has been established in studies of adults,

it has been consistently used in research on children due to the lack of established cut offs for children (Broyles et al., 2012; Murasko, 2008; Warnberg et al., 2007).

The independent variables of interest are measures of the child's home physical environment (HPE), including, whether the home: had no patio or yard, was unsafe inside, was unsafe outside, was dark/minimally lit, had little or monotonous décor, was crowded, was cluttered, and was unclean. The first measure (no patio/yard) was reported by the primary caregiver, while the other measures were observed by interviewers while they were in the home. Interviewers were trained and had a guide to follow during this observation process (Appendix A). Prompts were given for what the interviewers should look for in the home environment. Unsafe conditions in the home were indicated by the presence of one or more potentially dangerous health or structural hazards such as broken glass, peeling paint, or frayed electrical wires. The external home environment, including the yard, patio, porch, and/or exterior halls and stairs, was also rated for safety. Examples of unsafe exterior conditions included unlit entrance or stairway, broken steps, or large ditches. Households were considered crowded if there were many people living in a very small house or apartment, if it was difficult to find a private place to interview respondents, or if there were frequent interruptions and people bumping into each other. Clutter was indicated by the disorganization of objects in the home; such as, a vacuum cleaner, schoolwork, or shoes and socks left out. Cleanliness of homes was assessed by the level of trash, dirty dishes, or dust in visible rooms (Peterson et al., 2011).

These interviewer observations are similar to those used to measure the physical environment in the HOME scale (Bradley and Caldwell, 1979; Wen-jui, Leventhal and Linver, 2004) and in other studies of housing quality (Evans et al., 2000). Following past research on housing quality (Coley et al., 2013; Evans et al., 2000), these variables were dichotomized to reflect a negative home characteristic and then added together to produce a measure of the number of low-quality HPE indicators, ranging from 0 to 8. Research suggests that an overall housing quality index is more reliable than any specific indicator or subscale (Evans et al., 2000). We conducted a factor analysis and found one factor emerged for these 8 indicators. In addition to number of risk factors, we further characterized low-quality HPE as two or more and three or more low-quality indicators to test for threshold effects that may not be captured with a linear measure. As Table 1 shows, in this sample, the mean number of low-quality HPE indicators was 1.3, with 34% of children experiencing two or more and 15% three or more low-quality HPE conditions.

Child obesity, a potentially important predictor of inflammation and mediator of the HPE effects, was defined based on BMI (weight (kg)/height (m)<sup>2</sup>). Height and weight were measured by trained interviewers and BMI z-scores were calculated using age- and gender-specific growth curves of a reference population. We categorized a child as obese if their z-score was ≥ 95th percentile (Cole et al., 2000; Kuczmarski et al., 2002). In regression models we included a dummy variable for child obesity, with overweight or normal BMI as the omitted category. Including child overweight as a separate category from normal BMI did not change the results; and, the effect of overweight status was not significantly different from normal BMI.

Family income was included in regression models to assess the HPE effects net of economic status. In L.A.FANS, total family income sums all earned and transfer income (including income from government cash benefits and food stamps) and missing income data was imputed (Peterson et al., 2011). Using total family income, we calculated the income-to-poverty ratio by dividing income by the federal poverty line (FPL) for a given family size in the year for which the income was reported (the year prior to the survey year). In regression models we controlled for whether the measure included an imputed income component.

Other independent variables were child and primary caregiver control variables. Child-level controls included: child age, gender, recent illnesses (dummy), and recent infection (dummy). Primary caregiver controls included: immigrant status (born in the U.S.), race/ethnicity (White, Latino, African American, other), education (less than high school degree), self-reported depression (reported being depressed or on anti-depressants in the past year), marital status (married, separated/divorced, cohabiting, never married, widowed), number of children in the household, and number of years in the current home. Neighborhood poverty category was also included. L.A.FANS defined neighborhood poverty by the percent of the population living in poverty: very poor (the top 10% of the neighborhood poverty distribution), poor (those in the 60–89 percentiles of neighborhoods), and non-poor (those in the bottom 60% of the neighborhood poverty distribution) (Sastry et al., 2006).

### 3.3. Statistical methods

In developing our multivariate regression models, we logged the CRP measure and conducted Tobit regression analysis to account for censoring of the CRP values below the detectable level, consistent with past research in this area (Dowd et al., 2010). Standard errors were adjusted for heteroskedasticity and we used the cluster command in Stata to account for clustering of the data at the survey strata level. Research suggests that adjusting for clustering at higher geographic levels also accounts for clustering at lower levels, including the household level (Angeles et al., 2005; Cameron and Miller, 2015).

All regression analyses include the weights and control variables previously described. We report regression coefficients, which can be interpreted as percent change in CRP for each unit change in the independent variable. We conducted two-tailed tests of  $p < 0.05$  for statistical significance.

Obesity mediation was assessed by entering the obesity variable sequentially to illustrate changes in associations between CRP and the independent variables when obesity was included. We also conducted Sobel-Goodman mediation tests to assess the statistical significance of obesity as a mediator of our HPE measures and family income.

To test for age moderation effects, we created child age\*HPE interaction terms and included them in the regression models along with the main effect and control variables. We assessed the significance of the interaction terms, and the joint significance of the interaction terms with the HPE main effect variables when considering evidence of moderation.



## 4. Results

We first provide descriptive statistics for CRP and key child characteristics in L.A.FANS and in a national sample, the National Health and Nutrition Examination Survey (NHANES) during the same time period (2006–2008). We provide the NHANES data as a reference group, given the limited population-level data on CRP levels in children. Mean CRP among all children in the L.A.FANS sample is 2.5, compared to 1.7 in the U.S. (Table 2). When limited to those  $\leq 10$  mg/L, the focus of this study, mean CRP values are 1.1 in L.A. FANS and 0.93 in NHANES. Given that obesity and Mexican-American origin have been linked with higher CRP levels in children (Dowd et al., 2010), the higher CRP values in L.A.FANS may be due to the higher percentage of Hispanic and obese children in L.A. compared with the nation as a whole (see Table 2).

Turning to the regression results, Table 3 shows the associations between the HPE measures and CRP in children with CRP  $\leq 10$  mg/L. As Model 1 shows, the total number of low-quality HPE indicators was associated with CRP, independent of income and other socio-demographic controls, increasing CRP by 11% with each additional indicator. Models 2 and 3 indicate the significance of HPE threshold effects; children in homes with two or more low-quality HPE indicators had, on average, 30% higher CRP than those with 0 or 1 indicator. Those in the riskiest home environments (3+ low-quality HPE indicators) had 62% higher CRP than those in homes with 0–2 negative HPE indicators.

In Models 2 and 3, the family income-to-poverty ratio (IPR) was significantly associated with lower CRP, although the effect was quite small. Moving from being at the poverty line (IPR = 1) to income two times the poverty line (IPR = 2) was associated with a 3% decline in CRP. Although the small association between income and CRP could be due to mediation by HPE, we found no evidence of mediation in supplemental analyses (reported at the bottom of Table 3). Most other control variables were not associated with inflammation, with the exception of child gender (boys had lower inflammation, on average, than girls) and primary caregiver's (PCG) immigrant status (in some models). The effect of PCG "other race" was also significant, but few of our sample children had PCGs in this category (6%).

Models 4 and 5 present the results from regression models controlling for child obesity. Not surprisingly, obese children had significantly higher CRP than overweight and normal BMI children. We also found evidence that obesity mediated part of the HPE effects, with significant attenuation of the HPE indicators when controlling for obesity and significant Sobel-Goodman tests (reported at the bottom of Table 3). Obesity mediated the small effect of income as well.

Table 4 shows our results when assessing the role of child age as a moderator of the HPE effects. We found negative age interactions and joint significance of the HPE threshold variables with the age interactions (F-tests indicated at the bottom of Table 4). The negative interaction effects indicate that the associations between the HPE and children's CRP become smaller with increasing child age. The joint effect of the HPE and age interaction variables remained significant even when controlling for obesity (Table 4, Models 9 & 10).

To provide an interpretation of the age effects, Fig. 1 shows the predicted percent change in CRP by age for children who were exposed to two or more and three or more low-quality HPE indicators. These effects were calculated based on results from Models 7 and 8. As the figure illustrates, young children had the highest predicted increases in CRP in these risky home environments, with substantially higher CRP among children in households with three or more low-quality HPE indicators. Although the effect size decreased with age, it remained significant through age 16 for children living in homes with three or more low-quality HPE indicators. The effects were smaller, but still strong, for those exposed to two or more low-quality HPE indicators. The association between CRP and 2+ HPE indicators became insignificant after age 12.

## 5. Discussion

Despite general improvements in living conditions of children in the U.S. over the past 40 years, research suggests that large inequalities in home quality remain (Bradley et al., 2001; Conley, 2001; Evans et al., 2005). Recent findings suggest that housing conditions are critical to understanding the lives of low-income families with children and that housing quality, compared to other housing measures, is most strongly associated with child emotional, behavioral and cognitive development (Coley et al., 2013).

In this study, we considered how housing conditions measured as the home physical environment (HPE) contributed to inequalities in biological markers of stress in children. Our outcome, low-grade inflammation assessed by CRP levels, is an important indicator of a dysregulated immune system linked to exposure to chronic stress (Kemeny, 2009). Elevated inflammation is also currently being explored as a potential correlate of depression in children (Heim and Binder, 2012), as well as other health conditions that emerge during childhood and later in the life course (Crosswell et al., 2014; Slopen et al., 2012).

Using data from Los Angeles County, we found that a substantial portion of children (34%) experienced two or more physical risk factors in their HPE, and 15% lived in homes with 3 or more physical risk factors. The total number of low-quality HPE indicators and categorical risk level (2+ and 3+ low-quality indicators) were associated with higher CRP levels net of control variables. The effect of living in a home with three or more risk factors was particularly high, increasing CRP by an estimated 62%.

We also explored the role of obesity in mediating the associations between the HPE and CRP. Our results partially supported this hypothesis, with some of the HPE effects working through increased risk of child obesity. Further analyses indicated that the HPE had an important effect on obesity net of the control variables. This may reflect, in part, cumulated biological responses to chronic stressors and stress-coping behaviors (e.g., unhealthy eating, TV-watching) among children with low-quality HPE. It may also be that low-quality HPE restricted the physical activity of children. Both psychosocial stress and reduced activity have the potential to increase obesity risk in children (Wilson and Sato, 2014).

It is important to note that living in homes with multiple physical risk factors was associated with 20–30% higher CRP when controlling for obesity. Thus, the physical conditions of the

home were associated with immune system dysregulation in children that reflected not only increases in adiposity, but also immune system changes potentially related to exposure to chronic psychosocial stressors (Charmandari et al., 2012; Kemeny, 2009).

The negative HPE effects were particularly pronounced in younger children, with smaller, but still significant associations in adolescents. This finding is consistent with prior research on housing and children's emotional and behavioral outcomes (Coley et al., 2013). These findings may indicate that young children are particularly vulnerable to household disadvantage because the majority of their time is spent in the home and, unlike adolescents, they are unable to escape poor home conditions (West, 1997; West and Sweeting, 2004). It may also be that young children are more susceptible to the psychosocial threats related to poor physical qualities in the home than are older children.

There are many challenges to conducting research on bio-social processes. One such challenge is the lack of adequate data, including longitudinal data and large, population-based samples, particularly among children. This study focused on children from L.A. County and provided a snapshot of their home environment and physiological health at one point in time. This limits the generalizability to only children in L.A., and also carries the risk of null findings due to small numbers, less variability, and potential measurement error in estimating associations. The relatively small sample size ( $N = 353$ ) also makes sub-population analyses difficult. Although child age interactions with HPE were statistically significant, the age-specific effects should be viewed with caution due to the relatively small number of children at each age.

In our models, socio-demographic variables that should reflect disadvantage and stress exposure, such as low primary caregiver education and single-parent households were not associated with inflammation, unlike studies of other health outcomes in children (Goodman et al., 2005; Schmeer, 2012). This may be due to sample size or because the sample was 60% Hispanic and had a large percent of immigrant households. It may also be that inflammation is more sensitive to proximate stressors, rather than larger socioeconomic conditions. For example, parental education was not associated with CRP in a national sample of children (Dowd et al., 2010), neighborhood poverty was not associated with elevated CRP in a sample of Louisiana children (Broyles et al., 2012), and little is known about associations between family structure and inflammation in children. Thus, more research is required to establish which social and demographic characteristics of children's environments are associated with inflammation.

This study is also limited by relying on interviewer assessed home quality. Although interviewers were trained and given guidelines to follow during their rating process, we do not have a measure of inter-rater reliability to determine how successful the training was in producing consistent results across interviewers. We tested models including interviewer effects, and the results were unchanged. The use of biomarker data collected by technicians for our dependent variable eliminated concerns about error in the dependent variable being correlated with interviewer observations. Further, past research suggests that interviewer-rated home observations are preferred over respondents' reports of home qualities (Evans et al., 2001b).

Despite the limitations, this research contributes to our understanding of health inequalities in children and the potential role of chronic stress underlying these inequalities. More specifically, our research suggests that housing conditions, and the related home environment, have potentially important implications for child well-being, net of other social and economic characteristics of the family. The finding that younger children had higher risks of elevated inflammation in homes with poor physical conditions suggests that biological embedding of disadvantage may begin early in the life course.

This research also points to the need to address housing issues as part of policies and programs aimed at improving child health. Although social structures, such as socio-economic position and race/ethnicity clearly shape individuals' health status, living conditions are more amenable to targeted policy efforts. Further research is needed to assess whether policies and programs that provide housing assistance to improve the home physical environment could reduce stress and improve health outcomes in children within existing structural constraints.

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## Appendix A

Pebbley, Anne R., and Narayan Sastry. 2004. “The Los Angeles Family and Neighborhood Survey: Field Interviewer Manual.” Santa Monica, CA.

Excerpt from the Field Interviewer Manual (Pp. 8–163—8–166) [All text is quoted directly from the manual. Italics added to indicate instructions guiding the interviewer’s observations.]:

*Now you will take the computer back from the respondent and answer some observation questions about the household. Tell respondents you have a few administrative things to take care of on the computer that will take you a few minutes—that they can take a break until you finish.*

*The objective of these questions is to collect information about the physical environment and factors that may have an impact on children. You will read these questions to yourself and base your answers on what you know or have seen—do not explore the home in an attempt to better or more completely answer the questions. Do not discuss your observations with anyone in the household. This may be difficult if you see a hazard you feel could put a child at risk of injury. In cases such as these, talk to your FS.*

## Unsafe interior

*The first of these household observation questions appears below:*

ENVIRONMENT INSIDE HOME IS UNSAFE FOR YOUNG CHILDREN, ONE OR MORE POTENTIALLY DANGEROUS HEALTH OR STRUCTURAL HAZARDS. (EX: FRAYED ELECTRICAL WIRES, MICE OR RATS, GLASS, POISONS, FALLING PLASTER, BROKEN STAIRS, PEELING PAINT, CLEANING MATERIALS LEFT OUT, FLAMES AND HEAT WITHIN REACH OF YOUNG CHILD.)



*Note the things in the interior of the house that might be dangerous to a young child and that you can actually see. However, remember that you are not doing a safety inspection—you're looking for things that create an obvious health or safety risk to a child. Look for signs that would indicate health or structural hazards. For example, you may not see a mouse but you may notice mouse droppings. Or if none of the kitchen cabinets have doors on them, you may assume that the child could have access to any hazardous materials that are in the cabinets. Or if the face plates are missing from sockets and electrical switches, consider that hazardous as well. Remember that for this question you are only recording your observations about the inside of the house. If you see any of the items mentioned in the examples on the outside of the house or in the hallways or other common areas of an apartment building but do not see anything hazardous inside the home, indicate "No" for this question.*

## Unsafe exterior

*The next question asks about the outside of the home:*

ENVIRONMENT OUTSIDE HOME (YARD, PATIO, ENTRYWAY OR PORCH, HALLS AND STAIRS) IS UNSAFE FOR YOUNG CHILDREN. (EX: UNLIT ENTRANCE OR STAIRWAY, BROKEN STEPS, BROKEN GLASS, BROKEN TOYS, LARGE DITCHES, ALCOHOL OR DRUG PARAPHENALIA).

*For this question, note those things on the outside of the house may be dangerous to a young child. In addition to the examples given in the question, rusty metal objects, discarded refrigerators with doors attached, and discarded condoms may be considered hazardous and pose an obvious health or safety risk to a child. If you see any of the items mentioned in the examples on the inside of the house but do not see anything hazardous outside the home or in the hallways or other common areas of an apartment building, answer "No" for this question.*

## Lighting

*The next question is about the amount of light in the home:*

INSIDE OF HOME IS DARK. (EX: DARK ROOMS OR DRAPES)

*We ask this question because dark rooms with little light are less stimulating to a child.  
Answer:*

*"Yes" if the room or rooms you have seen have relatively little light or are fairly dark.*

## Décor

*For the next question, you will need to determine if the house visually stimulating:*

HOUSEHOLD HAS NO DECORATION OR DECOR IS MONOTONOUS. (EX: NO PICTURES OR NICK-NACKS, NO PLANTS, NO OR VERY LITTLE FURNITURE IN LIVING ROOM OR DINING ROOM)

*You are not rating the respondent's taste in decor. A respondent may live in a house that uses a lot of colors that you find unappealing, but it may still be visually stimulating. If the house*

*has no pictures on the walls or no knickknacks, plants, sculptures, statues, or other items or color schemes that draw your eye, answer “Yes.”*

## **Crowding**

*In the next question, you will need to determine if the house is crowded:*

INSIDE OF HOME IS CROWDED. (EX: MANY PEOPLE LIVING IN A VERY SMALL HOUSE OR APARTMENT, DIFFICULT TO FIND A PRIVATE PLACE TO INTERVIEW RESPONDENTS, FREQUENT INTERRUPTIONS AND PEOPLE BUMPING INTO EACH OTHER).

*If there are many people living in a small household (as indicated on the household roster) but you do not feel it is crowded, answer “No.” However, if the interview was interrupted or others were observing or distracting you or the respondent because there is no private space, answer “Yes.”*

## **Clutter**

*Now you will need to assess the amount of clutter:*

ALL VISIBLE ROOMS OF HOUSE/APARTMENT ARE MINIMALLY CLUTTERED OR NOT CLUTTERED AT ALL. (EX: VISIBLE ROOMS ARE NEAT OR ARE MINIMALLY CLUTTERED WITH CLOTHES, VACUUM CLEANER, CHILDREN’S SCHOOL WORK, SHOES AND SOCKS, OTHER OBJECTS).

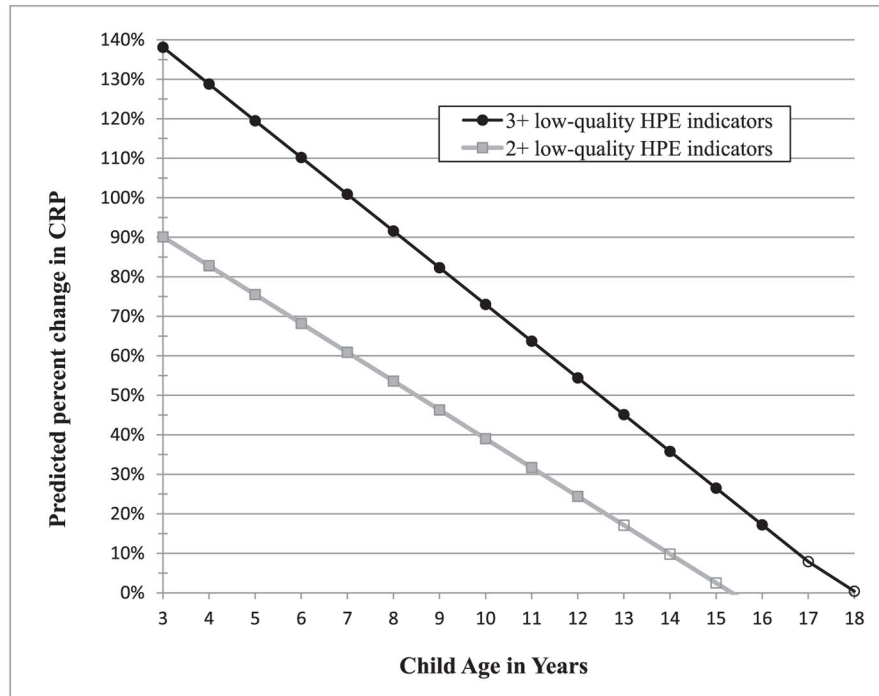
*Clutter may be a hazard to children, so observe the amount of disorder in the home’s physical environment. Look to see if toys, clothes, books, dishes, or other items are out of place—for instance, they are on the floor, tables, shelves, or scattered throughout the house. If there are small children, you will probably see some clutter—the amount of clutter may grow, the longer you remain in the household. Observe and record what you see.*

## **Cleanliness**

*The next question asks about the cleanliness of the home:*

ALL VISIBLE ROOMS OF THE HOUSE/APARTMENT ARE CLEAN OR REASONABLY CLEAN. (EX: NO TRASH STREWN AROUND, NO OR FEW DIRTY DISHES IN KITCHEN, FLOOR AND FURNITURE HAVE BEEN CLEANED OR DUSTED FAIRLY RECENTLY).

*A clean home provides a healthier environment for children. Notice if there are empty food containers, newspapers, junk mail, catalogues, magazines, etc., on the tables, countertops, floors, or strewn about may indicate a house that is not very clean. Notice the trash—is it overflowing? Does it have an odor? If there is a baby in the house, there may be an odor if a diaper needs to be changed or the lid to the diaper pail or trash is not airtight. If you see dust in the corners, dirty dishes stacked on the counters or table, or other signs of a dirty environment, answer “No” for this question. If the household is reasonably clean, answer “Yes.”*



**Fig. 1.** Predicted percent change in CRP by child age for children 3–18 years with CRP  $\geq$  10 mg/L. Based on results from Models (7) & (8).

**Table 1**Weighted descriptive statistics of children with CRP  $\geq 10$  mg/L, L.A.FANS Wave 2, N = 353.

Variables	Mean	Std. Dev.	Min	Max
<i>Home Physical Environment (HPE) Measures</i>				
Total # HPE indicators	1.3	1.5	0	8
Lower home quality (2 + HPE indicators)	34%			
Lowest home quality (3 + HPE indicators)	15%			
<i>Specific HPE indicators</i>				
No patio or yard	19%			
Crowded	20%			
Unsafe inside house	4%			
Unsafe outside house	3%			
Dark inside house	17%			
No decorations on walls	19%			
Cluttered	41%			
Not clean	18%			
<i>Child Characteristics</i>				
Child age in years	10.9	3.4	3	18
Child obese	22%			
Child was sick in past few days	12%			
Child had infection in past 3 weeks	3%			
Child male	53%			
<i>PCG<sup>a</sup> and Household Characteristics</i>				
PCG is mother	96%			
PCG < high school degree	36%			
PCG U.S.-born	47%			
PCG non-Hispanic white	25%			
PCG Hispanic	63%			
PCG African American	6%			
PCG married	59%			
Family income to poverty ratio	2.7	3.3	0	19.3
Number of children in household	2.5	1.1	1	7

<sup>a</sup>Primary caregiver.

Weighted descriptive statistics for children ages 3–18 years National Health and Nutrition Examination Survey (NHANES) and L.A.FANS Wave 2.

**Table 2**

	Mean <sup>a</sup>	Std. dev. <sup>a</sup>	Min <sup>b</sup>	Max <sup>a</sup>	% CRP 10 mg/L	Mean age	% Male	% Hispanic	% Obese
NHANES (2005–2008)									
All (N = 4304)	1.7	5.7	0.1	136	96%	12	51%	19%	12%
CRP 10 mg/L	0.93	1.7	0.1	10		12	51%	18%	12%
L.A.FANS (2006–2008)									
All (N = 404)	2.5	5.4	0.078	63	95%	11	51%	60%	25%
CRP 10 mg/L	1.1	1.9	0.078	10		11	53%	61%	22%

<sup>a</sup>All CRP values in mg/L.

<sup>b</sup>Determined by the lab's lowest detection level.

Table 3

Weighted Tobit regression models of CRP in Children 3–18 years with CRP 10 mg/L. L.A.FANS Wave 2, N = 353.

Models	(1)	(2)	(3)	(4)	(5)
<i>Home Physical Environment (HPE)</i>					
Total low-quality HPE indicators	0.11** (0.028)				
2+ low-quality HPE indicators		0.29** (0.040)		0.20* (0.100)	
3+ low-quality HPE indicators			0.62** (0.075)		0.34* (0.114)
<i>Child characteristics</i>					
Obese				0.87* (0.34)	0.83* (0.35)
Age	-0.014 (0.031)	-0.014 (0.032)	-0.011 (0.033)	-0.016 (0.037)	-0.015 (0.037)
Recently ill	-0.092 (0.80)	-0.092 (0.77)	-0.13 (0.79)	0.059 (0.73)	0.041 (0.74)
Infection in past 3 weeks	0.90 (0.50)	0.93 (0.48)	0.82 (0.48)	0.55 (0.65)	0.51 (0.64)
Male	-0.57** (0.22)	-0.57* (0.23)	-0.55** (0.21)	-0.55* (0.21)	-0.54** (0.20)
<i>PCC<sup>a</sup> and Household Controls</i>					
PCC U.S. born	-0.093** (0.018)	-0.081** (0.021)	-0.081 (0.050)	0.068 (0.11)	0.047 (0.12)
PCC Hispanic <sup>b</sup>	-0.12 (0.26)	-0.12 (0.27)	-0.17 (0.27)	-0.15 (0.17)	-0.19 (0.19)
PCC African American <sup>b</sup>	-0.26 (0.34)	-0.30 (0.32)	-0.13 (0.33)	-0.26 (0.31)	-0.17 (0.31)
PCC other race <sup>b</sup>	-0.66** (0.22)	-0.71** (0.23)	-0.73** (0.21)	-0.52** (0.21)	-0.55** (0.20)

Models	(1)	(2)	(3)	(4)	(5)
Variables					
Family income-to-poverty ratio	(0.089) -0.027	(0.086) -0.031**	(0.11) -0.029*	(0.056) -0.019	(0.088) -0.019
Log likelihood	(0.015) -424	(0.011) -424	(0.014) -422	(0.0099) -410	(0.011) -410
Sobel-Goodman mediation tests: Income by HPE	n.s.	n.s.	n.s.		
HPE by obesity				*	**
Income by obesity				**	**

Robust standard errors in parentheses.

\*\* p < 0.01,

\* p < 0.05.

<sup>a</sup>PCG = Primary caregiver. All control variables included in models; insignificant household and PCG controls not shown for brevity.

<sup>b</sup>Ref: non-Hispanic white.

**Table 4**

Weighted Tobit regression models of CRP in Children 3–18 years with CRP  $\geq 10$  mg/L including *home physical environment* \* *child age* interaction effects. L.A.FANS Wave 2, N = 353.

Variables	Models			
	Age interactions		Controlling for child obesity	
	(7)	(8)	(9)	(10)
2+ low-quality HPE indicators	1.12 ** (0.38)		0.94 ** (0.27)	
3+ low-quality HPE indicators		1.66 ** (0.34)		1.09 * (0.54)
Age * HPE interaction	-0.073 (0.037)	-0.093 ** (0.029)	-0.065 * (0.030)	-0.066 (0.039)
Child age	0.0056 (0.027)	0.0025 (0.037)	0.0016 (0.036)	-0.0050 (0.042)
Child obese			0.85 * (0.34)	0.81 * (0.38)
F-test joint significance HPE main effects and HPE * age interaction terms	**	**	**	*

Robust standard errors in parentheses.

\*\*  
p < 0.01,

\*  
p < 0.05.

All control variables included in all models but not shown for brevity.