Disparities in comorbidity of lead and asthma among preschool children in the U.S.

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National impact of lead poisoning and asthma

Two of the leading environmentally-based diseases among children include lead poisoning and asthma. In the U.S., about 10% of all children and 6% of children under the age of 5 have doctor-diagnosed asthma (Moorman et al., 2012). Additionally, almost 3% of children aged 1 to 5 years have blood lead levels at or above 5 µg/dL (Wheeler & Brown, 2013). Landrigan and Schechter (2002) estimated the annual costs of all environmentally attributable childhood diseases at $54.9 billion in which $43.4 billion was due to lead poisoning and $2 billion was due to asthma. Racial and socioeconomic disparities have been documented for both blood lead levels (Lanphear, Weitzman, & Eberly, 1996; Pamuk, Makuc, Heck, & Reuben, 1998) and pediatric asthma (Gold & Wright, 2005) for preschool-aged children in which minority and low-socioeconomic status children are at greater risk for both conditions. The question remains as to whether these diseases are comorbid with each other for young children.

Comorbidity of lead poisoning and asthma

Available evidence of lead and asthma comorbidity for children has had mixed findings. Some studies have found no association between lead and asthma for children, but they contain some limitations regarding sample selection and measurement (Joseph et al., 2005; Myers, Rowell, & Binns, 2002). Myers and colleages did not find evidence of comorbidity among inner-city children living in Chicago. However, they compared a small sample of children matched on demographic characteristics from a tracking lead database in which all children were recorded as having some measurable level of blood lead. In this study, their hypothesis was that children with lead poisoning would have higher rates of asthma than children without lead poisoning. However, their definition of children without lead poisoning was having a BLL lower than 4

μg/dL. What the researchers may not have accounted for is that children with very low levels of lead exposure may still be at risk for asthma or wheezing. Therefore, the use of children with low but measurable BLLs as the "control group" may not have been appropriate for their hypothesis. It is possible that perhaps there is a threshold of lead related to risk of asthma that is below their cutoff. In another study, comorbidity was examined in a sample of children from a managed care organization (MCO) in southern Michigan (Joseph et al., 2005). The researchers found no association between blood lead levels and asthma for African American children and a weak but non-significant association for white children. However, blood lead levels and asthma share common risk factors based on socioeconomic status, and this particular study had difficulty separating out this effect. Although they used annual income as an adjustment to their models, they did not explore potential interactions between socioeconomic status and comorbidity. Considering that blood lead levels are purely from environmental causes while asthma is comprised of both genetic and environmental causes, it is possible that comorbidity may be more complex. It is possible that comorbidity of lead and asthma exists only among children at risk of environmental exposures related to both lead and asthma which may, in turn, be related to socioeconomic status.

On the other hand, there have been several studies to report an association between asthma and elevated blood lead levels among children (Lutz et al., 1999; Motosue et al., 2009; Smith & Nriagu, 2011). Smith and Nriagu (2011) reported that for children residing in three zip codes of Saginaw, Michigan, an asthma diagnosis in the last 12 months significantly predicted blood lead levels at or above 10 μg/dL. By selecting children that mainly lived in low-income and minority households, the researchers were able to account for socioeconomic status when looking at lead and asthma comorbidity. Among children from disadvantaged households, lead

and asthma were significantly associated. Similarly, another study found that among low-income children in Houston, Texas, lead exposure was significantly correlated with asthma diagnosis (Motosue et al., 2009). Blood lead has been associated with IgE levels in infants and young children, the latter measure having been used as a biomarker for allergies and asthma (Lutz et al., 1999; Rabinowitz, Allred, Bellinger, Leviton, & Needleman, 1990; Sun, Hu, Zhao, Lon, & Cheng, 2003).

One limitation of all of these studies is that none of them examined comorbidity within a large, nationally representative sample; rather, they focused on small samples from specific geographic areas (e.g., zip codes Saginaw, Michigan or neighborhoods in Houston, Texas). The location of studies that found no association between lead and asthma may have differed compared to the location of studies that did find an association between lead and asthma in characteristics potentially related to comorbidity, such as housing quality, racial segregation, or poverty concentration.

Racial/ethnic and socioeconomic disparities in lead poisoning and asthma

Although overall lead levels have been decreasing dramatically since regulations have eliminated the use of lead in paint and gasoline, there remains socioeconomic and racial/ethnic disparities in blood lead levels among preschool-aged children (Jones et al., 2009). Children in poverty and those who are non-Hispanic black are at the highest risk for elevated blood lead levels. Asthma prevalence among preschool children has been increasing over time and it shows similar patterns of disparities in that those disproportionately affected are black and low-income children (Gold & Wright, 2005).

The disease patterns for both pediatric asthma and lead poisoning are very similar (Joseph et al., 2005; Lanphear, Weitzman, & Eberly, 1996). Both have similar patterns of racial

and socioeconomic disparities such that minority and low-SES groups tend to be disproportionately affected. Additionally, both are suggested to be caused and exacerbated by household conditions (although this is truer for lead poisoning than for asthma).

For children of this age, racial disparities in exposure to lead have primarily been attributed to differences in the home environment, specifically regarding lead-contaminated dust (Lanphear & Roghmann, 1997; Lanphear et al., 1996; Lanphear et al., 2002). In older homes (especially homes built before 1950), the use of lead-based paint was common; therefore, paint may begin to peel and collect as dust within the home where children will be exposed (Levin et al., 2008; Pamuk et al., 1998).

Socioeconomic disparities in blood lead levels have been found for young children. Pamuk and colleagues (1998) identified evidence of an income-blood lead level gradient in which each decrease of income was related to an increase in the concentration of lead in the blood. This income gradient was also found to vary by race/ethnicity, showing the strongest relationship among black children. Additionally, higher blood lead levels are associated with dilapidated environments as well as the presence of nutritional deficiencies in iron and calcium, which is more common among children than adults and among low-income than high-income individuals (Bellinger, 2004).

In the U.S., asthma prevalence is higher for black children than it is for white children, even among a middle class population (Gold & Wright, 2005). Among children and adults with asthma, severity is worse for blacks and Hispanics compared to whites. It has also been found that socioeconomic disparities in asthma may be related to environmental conditions (Gold & Wright, 2005). Disparities in asthma have been related to a number of housing conditions, including indoor environmental exposures that lead to asthma exacerbation include tobacco

smoke, cockroaches, animal dander, dust mites, and mold (Gold & Wright, 2005; Wright & Subramanian, 2007).

Environmental pathway for lead poisoning and asthma

Asthma diagnosis is determined by both environmental and genetic factors (Burke, Fesinmeyer, Reed, Hampson, & Carlsten, 2003; Wright & Subramanian, 2007). There are numerous risk factors for asthma (e.g., air pollution, road proximity), but substandard housing is one of the primary risk environments for preschool-aged children. The association between household exposures and asthma has been repeatedly acknowledged in the research (Breysse et al., 2004; Elliott et al., 2007). Indoor environmental exposures including tobacco smoke, cockroaches, animal dander, dust mites, and mold can lead to asthma exacerbation (Wright & Subramanian, 2007). In particular, early exposure to cockroach allergens (also a marker of substandard housing) has been linked to the development and severity of asthma symptoms (Arruda et al., 2001; Olmedo et al., 2011; Rosenstreich et al., 1997; Togias, Fenton, Gergen, Rotrosen, & Fauci, 2010).

Lead is still commonly found in the soil near roads, in building materials in older homes (e.g., pipes, paint), and certain items in the home (e.g., clay pots from Mexico) in addition to a number of industrial sites and occupational exposures (e.g., auto repair, battery reclamation) due to its wide use historically (e.g., leaded gasoline, paint, pesticides) (Agency for Toxic Substances and Disease Registry, 2007). However, the primary exposures for lead poisoning in preschool-aged children occur due to housing factors, parental occupation, and urban residence (Levin et al., 2008). Older and deteriorating housing (particularly homes built prior to 1978) may contain lead-based paint which can be ingested when children eat paint chips or inhaled from lead dust. An additional route of lead exposure is through particular parental occupations (e.g., battery

reclamation, automobile repair) in which parents inadvertently bring lead dust into the home via their clothing. Finally, the association between lead and urban residence may have more to do with exposure to air pollution via roadways or industrial sites (Brink et al., 2013; Kim, 2004). Preschool children are particularly vulnerable to lead toxicity because they have more "hand to mouth activity" (Lanphear & Roghmann, 1997) and absorb lead more easily into their systems (up to 50%) compared to adults (10-15%) (Bellinger, 2004; Papanikolaou, Hatzidaki, Belivanis, Tzanakakis, & Tsatsakis, 2005). Additionally, low-income and minority children may be at further risk of lead exposure because they are more likely to live in older and substandard housing and urban areas. Additionally, occupations in which there is lead exposure tend to be disproportionately represented by low-income adults, which could partially explain socioeconomic disparities in lead poisoning (Tong & Lu, 2001; Vivier et al., 2011).

Research has shown that children with low levels of exposure may suffer from compromised developmental and intellectual performance later in life (Bellinger, 2004; Canfield et al., 2003; Chiodo, Jacobson, & Jacobson, 2004). In one study, each increase of 10 µg/dL was associated with a 4.6-point decrease in IQ among a sample of children age 5 and under. Of particular importance is that children with lead levels below 10 µg/dL had a greater loss of IQ than for children at or above this level. There is no safe threshold for lead in children, as studies have shown negative impairments even as low as 3 µg/dL of blood lead levels (Chiodo et al., 2004). Chelation is the only medical treatment for lead, but it is only used for individuals with blood lead levels at much higher levels (~ 30 µg/dL or higher) (Needleman, 2009). The next step is lead poisoning prevention.

The home environment is the primary setting in which preschool-aged children can be at risk of chronic conditions, notably asthma and lead poisoning. Older and/or substandard homes

are more likely to have multiple environmental hazards which can contribute to both asthma symptoms and lead poisoning as well as infectious disease transmission, injuries, and developmental disabilities (Saegert, Klitzman, Freudenberg, Cooperman-Mroczek, & Nassar, 2003). House dust and fine airborne particles have been identified as risk factors both blood lead levels and asthma (Lanphear et al., 1998; Levin et al., 2008; Wilson et al., 2010; Wright & Subramanian, 2007). The suggested mechanism behind any comorbidity between lead and asthma is primarily due to substandard housing that presents common exposures conducive to both diseases. Additionally, although there have been improvements in housing conditions over time, there seems to be little change in disparities related to housing (Jacobs, Wilson, Dixon, Smith, & Evens, 2009). Low-income families are more likely to live in older housing where lead paint may have been applied and where roof leaks and an aging structure may augment exposure to asthma triggers such as molds, dust mites, cockroaches, and other allergens. Deterioration of housing has been shown to be associated with indoor allergen levels, specifically for cockroaches, even when controlling for socioeconomic status (Rauh, Chew, & Garfinkel, 2002). Particularly, public and low-income housing has been associated with a higher likelihood of exposure to rats, cockroaches, and leaking water pipes as well as asthma diagnosis compared to private housing (Northridge, Ramirez, Stingone, & Claudio, 2010; Rosenfeld et al., 2011). This indicates a health disparity based on available housing since residents of public housing also tended to have lower median income than residents of private housing.

Purpose of current study and hypotheses

The purpose of the current study is to determine whether blood lead levels and asthma are comorbid conditions, and if comorbidity only exists for socially disadvantaged groups. Then analyses will determine whether particular individual and household characteristics predict lead

and asthma comorbidity for preschool-aged children. As such, three questions are addressed in this research. First, are blood lead levels and asthma diagnosis comorbid conditions for children under the age of 6? It is expected that elevated lead levels and asthma are comorbid conditions. However, it is also expected (due to mixed results in the previous research), that this may only be true in particular conditions (e.g., low-SES children living in older homes). Second, are there particular groups (i.e., racial/ethnic minorities or children in lower socioeconomic status families) that are at a higher disadvantage for comorbidity of blood lead and asthma? It is expected that both non-Hispanic black and low-SES children will be more likely to have elevated blood lead levels (i.e., 2 μg/dL) and more likely to have current doctor-diagnosed asthma compared to white children. Finally, are children at a higher risk of comorbidity due primarily to the characteristics of the home? It is expected that living in homes older than 1978 will be a risk factor for lead and asthma comorbidity, and it is expected that this risk factor will be stronger for minority and low-SES children compared to white and high-SES children, respectively. Additionally, models will be stratified by housing tenure to explore potential differences in lead and asthma comorbidity between children who live in homes that are owned and children who live in rented homes or some other arrangement.

Method

The National Health and Nutrition Examination Survey (NHANES) is a series of cross-sectional surveys conducted by the National Center for Health Statistics and provides information regarding the health and nutritional status of adults and children in the United States (CDC, 2003-2010). The NHANES is a series of cross-sectional surveys using a stratified multistage probability sample of the civilian non-institutionalized population of the U.S. The data is comprised of questionnaires including demographic, socioeconomic, diet, and other

health-related variables, a medical examination component, and laboratory testing. Data from the NHANES are used primarily to analyze the comorbidity of elevated blood lead levels (EBLLs) and asthma diagnosis. Additionally, other demographic, socioeconomic, and housing variables are included to help explain the relationship between EBLLs and asthma diagnosis.

The data used in the current analyses combines four 2-year files (i.e., 2003-2004, 2005-2006, 2007-2008 and 2009-2010) and includes variables from the questionnaires and laboratory testing. Cases were selected to include only respondents who were from age 1 to 5 in order to investigate potential associations between blood lead level and asthma diagnosis among preschool-aged children. Additionally, current analyses were limited to two race categories of non-Hispanic white and non-Hispanic black. This resulted in a sample size of 2,897. After excluding children who did not provide a venous blood draw (n=958) and children with missing data on other variables of interest (n=84), the final sample size was 1,855.

Elevated blood lead levels and asthma diagnosis

Lead levels were determined using venous blood draws during the laboratory testing for all respondents age 1 and older. The lower limit of lead detection in the blood ranged from .18 to .21 μg/dL across the four data files. Based on the current literature on the negative effects of lead even at relatively low levels (Bellinger, 2004; Canfield et al., 2003; Chiodo et al., 2004), a dichotomous variable was created to compare children with levels at or above 2 μg/dL to those with lead levels below 2 μg/dL (reference).

The adult respondent was asked whether a doctor, nurse, or other medical professional ever said that the child in question has asthma. If the respondent said yes, the child was coded as having a current asthma diagnosis (reference: never diagnosed with asthma).

Housing characteristics

Respondents were asked what year their home was originally built, which was provided in six categories from "before 1940" to "1990 to present". Based on evidence that older housing is at risk for lead paint as well as a number of factors that increase exposure to allergens that are known to exacerbate asthma (Jacobs et al., 2009; Wilson et al., 2010), this variable was recoded to compare children living in homes built before 1978 to children living in homes that were built in 1978 or later (reference). Additionally, a large proportion of respondents reported that they did not know the age of the home. These responses were recoded into a separate category for age of housing. It is possible that when respondents do not know the age of their home, the home may be older. Additionally, when comparing multiple categories of housing age across different ranges of child blood lead levels, the unknown category for age of housing has a pattern very similar to that of older housing (i.e., pre-1978) in that it has higher proportions of children in categories of lower lead levels (i.e., < 5 μg/dL) (Jones et al., 2009).

Additionally, respondents were asked about their current housing tenure, specifically whether their home was owned or being bought, rented, or some other arrangement. This variable was recoded to compare children living in homes that were owned by the respondent to children living in any other arrangement in which the home was not owned (reference).

Sociodemographic characteristics

The questionnaire data provides a ratio of family income to poverty threshold that is computed by dividing family income by the poverty threshold for the respective year. Respondents reported their total annual income for themselves and other members of their family (defined as two or more related people). If respondents did not answer, they were asked to select 1 of 11 income categories. In latter cases, the midpoint of each income category was used to calculate the income-poverty ratio. The ratio ranged from 0 to 5 (note, any ratio above 5 was

capped). This variable was recoded to compare children from families with a ratio of less than 2 (i.e., below 200% of the poverty threshold) to children from families with a ratio of 2 or higher (i.e., at or above 200% of the poverty threshold).

Race/ethnicity is a recoded variable provided in the NHANES, and it consists of five categories: Mexican American, other Hispanic, non-Hispanic white, non-Hispanic black, and other race (including multi-racial). Only children who reported non-Hispanic black and non-Hispanic white were included in the current analyses. Race was dichotomized to compare black and white (reference) children.

Control variables for association with elevated blood lead levels (EBLLs)

Some demographic and health access characteristics were included as controls for the regression model because there are changing trends of EBLLs over time as a result of removing lead from common sources such as paint and gasoline as well as research that has found that children are more likely to have elevated blood lead levels when they are younger. Since data is combined from a span of 8 years of data collection (i.e., four 2-year files), the survey cohort was also included as a control variable in the regression model. Child's age was coded to compare each year of age with age 5 as the reference group. Additionally, gender and health insurance status were also included since they may influence the predictor of asthma diagnosis (a predictor of interest in the model) since children with asthma are more likely to be boys and are more likely to have health insurance. The child's sex was coded to compare boys to girls (reference). Health status was recoded to compare children with any type of health insurance to children without any health insurance (reference).

Statistical methods

First, descriptive statistics of sociodemographic characteristics, housing factors, and health outcomes were provided for the full sample. Next, chi-square tests were computed to look at associations between (1) poverty status and housing characteristics, (2) health outcomes (i.e., EBLLs and asthma diagnosis) and housing and sociodemographic characteristics, and (3) comorbidity and housing and sociodemographic characteristics. The svychisq procedure in R version 3 was used due to the complex sample design of the NHANES. Chi-square significance tests for differences in each characteristic by blood lead level and asthma, respectively, and *P*-values from those comparisons are displayed in Tables 1 and 2. Chi-square significance tests were also run to compare characteristics between children living in homes that are owned compared to children living in homes that are rented or some other arrangement (Table 3).

In order to test for associations between lead and asthma and the potential underlying mechanisms, nested binary logistic regressions were performed. Tables 4 and 5 display the odds ratios (ORs) and 95% confidence intervals for the variables in each of the nested models. Additionally, goodness-of-fit was tested for each model using likelihood ratio tests. The first model estimates the association between elevated lead level and asthma diagnosis, after adjusting for survey cohort, age and gender of the child, and health insurance status. The second model adds the age of the home and housing tenure to determine whether housing characteristics explain the relationship between lead and asthma comorbidity. The third model adds poverty status to assess the effect of socioeconomic status on the relationship between lead and asthma and because it is associated with housing characteristics. The final model adds race to the regression equation to determine how this variable may interact with poverty status and housing characteristics to predict health outcomes in children. Models stratified by poverty status are also considered.

Models stratified by housing tenure are also considered to determine whether different factors predict children's blood lead level depending on whether they live in homes that are rented compared to homes that are owned. In each stratified sample, four nested models are computed as they were for the full sample (with the removal of housing tenure as a predictor).

Results

Associations with elevated blood lead levels and asthma diagnosis

In the chi-square analyses of associations between EBLLs and the sociodemographic and housing factors (see Table 1), lead was significantly associated with poverty status and child's age and race as well as the age of home and housing tenure. Children with lead levels at or above 2 µg/dL were disproportionately below the poverty threshold, black, and younger. Additionally, children with elevated lead levels were living disproportionately in homes older than 1978 or in which the age was unknown and in homes where the tenure was not ownership.

Table 1 Weighted percentages of sociodemographic and housing factors by lead level with design effects (n = 1855).

	(%)	
	No lead	Lead level
Comorbid condition		
Asthma diagnosis*		
No asthma	72.7	27.8
Asthma	63.6	36.4
Demographics		
Gender		
Male	69.6	30.4
Female	73.0	27.0
Age***		
1	61.5	38.5
2	63.5	36.5
3	72.3	27.7
4	77.1	22.9
5	80.8	19.2
Health care access		
Health insurance		
No health insurance	66.1	33.9
Any form of insurance	71.7	28.3
Sociodemographic characteristics		

Poverty status***		
Below 200% threshold	60.4	39.6
At or above 200% threshold	80.7	19.3
Race***		
White	76.8	23.2
Black	50.4	49.6
Housing characteristics		
Age of housing***		
Pre-1978	67.7	32.3
1978 and newer	82.2	17.8
Don't know age of housing	54.1	45.9
Housing tenure***		
Own	76.2	23.8
Do not own	63.0	37.0

* P ≤ 0.05; ** P ≤ 0.01; *** P ≤ 0.001

In the same chi-square analyses for asthma diagnosis and sociodemographic and housing factors (see Table 2), the patterns were generally the same. Asthma diagnosis was associated with poverty status, child's age and race, and child's health insurance status as well as the age of the home and housing tenure. Children with an asthma diagnosis were disproportionately below the poverty threshold and black but older. There was also a higher asthma diagnosis rate among children with health insurance. Additionally, children with an asthma diagnosis were more likely to be in homes age 1978 and newer or in homes where the age was unknown in addition to homes where the tenure was not ownership. The pattern of results for asthma diagnosis differs from that of lead levels for age of the child as well as the age of the housing.

Table 2 Weighted percentages of sociodemographic and housing factors by asthma diagnosis with design effects (*n* = 1855).

	(%)	
	No asthma	Asthma
Comorbid condition		
Lead level		
< 2 µg/dL	90.2	9.8
>= 2 µg/dL	86.1	13.9
Demographics		
Gender**		
Male	86.7	13.3
Female	91.6	8.4
Age***		
1	92.4	7.6

2	88.7	11.3
3	90.6	9.4
4	91.3	8.7
5	82.9	17.1
Health care access		
Health insurance*		
No health insurance	94.4	5.6
Any form of insurance	88.6	11.4
Sociodemographic characteristics		
Poverty status***		
Below 200% threshold	85.6	14.4
At or above 200% threshold	92.0	8.0
Race***		
White	91.0	9.0
Black	81.7	18.3
Housing characteristics		
Age of housing***		
Pre-1978	96.6	3.4
1978 and newer	89.4	10.6
Don't know age of housing	81.2	18.8
Housing tenure***		
Own	91.2	8.8
Do not own	85.4	14.6

* P ≤ 0.05; ** P ≤ 0.01; *** P ≤ 0.001

Descriptive statistics for total sample and by housing tenure

The sample of preschool children was proportionately distributed by age and gender. Additional descriptive statistics for the full sample as well as associations of these variables by housing tenure are provided in Table 3 below. Statistically significant differences are found for housing tenure and socioeconomic status, race, housing age, and child health. For children living in homes that are not owned, there are higher proportions of children who are black, below 200% of the poverty threshold, and live in housing that was built before 1978 or where age of housing was unknown. Additionally, rates of both asthma diagnosis and elevated blood lead levels (i.e., at or above 2 μg/dL) are higher among children who live in homes that are not owned.

Table 3 Weighted percentages of health and housing factors by housing tenure with design effects (n = 1855).

(%)

	Total	Rent (n = 983)	Own (n = 872)
Sociodemographic characteristics			
Housing tenure			
Rent	46.6	-	-
Own	53.4	-	-
Race***			
White	79.0	59.9	90.4
Black	21.0	40.1	9.6
Poverty status***			
Below 200% threshold	46.6	72.2	31.2
At or above 200% threshold	53.4	27.8	68.8
Health care access			
No insurance	7.5	8.3	7.1
Insurance	92.5	91.7	92.9
Housing characteristics			
Age of housing***			
Pre-1978	59.4	34.1	47.9
1978 and newer	16.8	26.4	48.9
Don't know age of housing	23.8	39.5	3.2
Health characteristics			
Asthma diagnosis***			
No asthma	89.0	85.4	91.2
Asthma	11.0	14.6	8.8
Elevated blood lead level***			
Below 2 µg/dL	71.3	63.0	76.2
At or above 2 µg/dL	28.7	37.0	23.8

* $P \leq 0.05$; ** $P \leq 0.01$; *** $P \leq 0.001$

Regression analyses

To determine what variables may influence the association between EBLLs and asthma diagnosis, nested binary logistic regressions were computed to predict asthma diagnosis by EBLLs, housing characteristics, and sociodemographic characteristics. Table 4 presents the results of the multivariate models examining the association of EBLL with the presence of an asthma diagnosis for the full sample. In the initial model, asthma diagnosis was entered after adjusting for a number of demographic and health access factors that have been shown to be associated with EBLLs. Child's age, gender, and health insurance status as well as the survey cohort were included. After adjusting for these factors, children with an asthma diagnosis were

1.6 times more likely to have an EBLL compared to children without an asthma diagnosis. Among the variables used for adjustment, younger children (i.e., ages 1 to 3) were more likely to have an EBLL compared to children age 5. Additionally, survey cohort was significant and showed that EBLLs decreased with each wave. Gender and health insurance status were not significant.

When housing characteristics were added to the model the association between EBLLs and asthma diagnosis was reduced (Table 4, Model 2). However, the effect of EBLLs was significant, and children with an asthma diagnosis were 1.5 times more likely to have an EBLL. The effects of housing characteristics were significant predictors of EBLLs. Children living in homes older than 1950 were 2.5 times more likely to have an EBLL, and children living in homes where the age was unknown were 3.4 times more likely to have an EBLL. The variable of housing tenure was also significantly related to lead; children living in homes that were not owned were 1.5 times more likely to have an EBLL. It is also important to note that housing tenure and age of home are significantly related. A higher proportion of homes in which age is unknown are also homes that are not rented or under some other arrangement. All other predictors displayed the same pattern of results as the first model.

Adding poverty status to the model reduced the association between asthma diagnosis and EBLLs as well as reduced the effects of housing tenure and whether the age of the home was known (Table 4, Model 3). The effect of asthma diagnosis on EBLLs was marginally significant, and children with an asthma diagnosis were 1.4 times more likely to have an EBLL than children without an asthma diagnosis. Additionally, the inclusion of poverty status eliminated the previous significant effect of housing tenure. Not knowing the age of the home is still significant

and in the same direction, but the effect was reduced after adding poverty status to the model. All other predictors displayed the same pattern of results as the previous model.

When race was added to the model, the association between asthma diagnosis and EBLLs was no longer significant (Table 4, Model 4). Additionally, race seems to add an independent effect over poverty status on child's lead status such that non-Hispanic black children were more likely to have EBLLs than non-Hispanic white children. All other associations showed the same pattern of results as found in the previous model. When examining model fit, each successive model was a significant improvement over the previous model, as shown by the significant likelihood ratio tests for each model.

Table 4 Binary logistic regressions of the effects of asthma diagnosis and covariates on preschool-aged children's blood lead level status for the total sample (*n* = 1855).

	Model 1		Model 2		Model 3		Model 4	
	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)
Control variables[1]								
Survey cohort	0.74	(0.63-0.86)***	0.71	(0.62-0.83)***	0.71	(0.62-0.83)***		(0.61-0.82)***
Age (ref=Age 5)								
Age 4	1.36	(0.92-2.02)	1.37	(0.92-2.05)	1.38	(0.93-2.06)	1.35	(0.90-2.02)
Age 3	1.78	(1.23-2.57)**	1.81	(1.25-2.62)**	1.89	(1.31-2.73)**	1.80	(1.24-2.63)**
Age 2	2.63	(1.81-3.82)***	2.91	(2.00-4.21)***	2.91	(1.97-4.29)***	3.00	(2.02-4.47)***
Age 1	2.84	(1.99-4.04)***	3.13	(2.17-4.51)***	3.34	(2.31-4.84)***	3.37	(2.32-4.88)***
Gender (ref=female)								
Male	1.15	(0.92-1.44)	1.21	(0.98-1.50)†	1.22	(0.99-1.52) †	1.26	(1.00-1.58) †
Insurance status (ref=no insurance)								
Have health insurance	0.75	(0.45-1.26)	0.80	(0.46-1.39)	0.88	(0.50-1.56)	0.80	(0.45-1.42)
Asthma diagnosis								
Ever had asthma	1.62	(1.15-2.28)**	1.48	(1.03-2.13)*	1.40	(0.98-2.02)†	1.28	(0.89-1.85)
Housing characteristics								
Age of home (ref: 1978 and newer)								
Pre-1978			2.48	(1.72-3.60)***	2.42	(1.68-3.51)***	2.47	(1.71-3.57)***
Don't know age of home			3.37	(2.11-5.39)***	2.68	(1.68-4.28)***	1.91	(1.21-3.01)**
Housing tenure (ref: Own)								
Rent or other arrangement			1.46	(1.08-1.98)*	1.15	(0.83-1.59)	0.97	(0.70-1.37)
Sociodemographic factors								
Poverty status (ref=Above threshold)								
Below threshold					2.30	(1.67-3.15)***	2.16	(1.59-2.93)***
Race (ref=non-Hispanic white)								
Non-Hispanic black							2.76	(2.09-3.64)***
χ^2	76.80***		68.07***		26.70***		50.94***	
AIC_{min}=2132.23	2082.23		2005.00		1959.60		1940.94	

† P ≤ 0.10 * P ≤ 0.05; ** P ≤ 0.01; *** P ≤ 0.001

[1]Analyses were also adjusted for survey cohort (not shown in table).

The outcomes for the stratified models are different depending on whether children live in homes that are owned compared to children who live in homes that are rented or under some other arrangement (Table 5). The primary finding is that asthma diagnosis significantly predicted elevated blood lead levels for children who live in homes that are rented or under some other arrangement. Asthma diagnosis did not predict EBLLs for children living in homes that are owned. The pattern of results based on housing tenure was also slightly different for the age of the child and for health insurance status. For children living in rented homes, children ages 1 and 2 were more likely to have EBLLs than older children. For children living in homes that are owned, the finding was similar but children age 3 were also significantly more likely to have EBLLs than older children. Additionally, health insurance status was a significant predictor of EBLLs but only for children in homes that were owned. Children with some form of health insurance were less likely to have EBLLs than children without health insurance. All patterns were very similar as shown in the models with the full sample. In both types of housing tenure, children living in homes older than 1978 and where the housing age was unknown were more likely to have EBLLs than children living in newer homes. Children were more likely to have EBLLs if they were below 200% of the poverty threshold compared to children at or above the poverty threshold and non-Hispanic black children were more likely to have EBLLs than non-Hispanic white children.

Table 5 Binary logistic regressions stratified by housing tenure for the effects of asthma diagnosis and covariates on preschool-aged children's blood lead level status for the total sample (*n* = 1855).

	Rent or some other arrangement (*n* = 983)							
	Model 1		Model 2		Model 3		Model 4	
	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)
Control variables								
Survey cohort	0.73	(0.62-0.85)***	0.71	(0.61-0.82)***	0.71	(0.62-0.82)***	0.70	(0.61-0.81)***
Age (ref=Age 5)								
Age 4	1.18	(0.72-1.91)	1.14	(0.70-1.87)	1.08	(0.65-1.79)	0.96	(0.58-1.59)
Age 3	1.45	(0.90-2.33)	1.37	(0.83-2.26)	1.32	(0.80-2.15)	1.20	(0.75-1.91)
Age 2	2.35	(1.59-3.49)***	2.33	(1.56-3.48)***	2.15	(1.40-3.29)**	2.21	(1.40-3.48)**
Age 1	3.00	(1.91-4.71)***	2.98	(1.80-4.93)***	2.95	(1.77-4.90)***	2.84	(1.70-4.77)***
Gender (ref=female)								
Male	1.12	(0.87-1.46)	1.18	(0.89-1.56)	1.20	(0.91-1.59)	1.23	(0.93-1.64)
Insurance status (ref=no insurance)								
Have health insurance	1.70	(0.90-3.21)	1.87	(0.98-3.58)†	1.67	(0.81-3.43)	1.30	(0.65-2.59)
Asthma diagnosis								
Ever had asthma	1.46	(1.05-2.04)*	1.38	(1.00-1.91)†	1.28	(0.92-1.78)	1.18	(0.83-1.69)
Housing characteristics								
Age of home (ref=1978 and newer)								
Pre-1978			3.02	(1.68-5.42)***	3.02	(1.66-5.49)***	3.29	(1.81-5.98)***
Don’t know age of home			3.90	(2.17-7.02)***	3.19	(1.81-5.63)***	2.28	(1.32-3.92)**
Sociodemographic factors								
Poverty status (ref=Above threshold)								
Below threshold					2.44	(1.40-4.23)**	2.21	(1.29-3.78)**
Race (ref=non-Hispanic white)								
Non-Hispanic black							2.99	(2.11-4.24)***
χ^2	63.08***		32.70***		10.71**		38.85***	
AIC_{min}=1359.43	1327.91		1273.10		1248.96		1213.06	

† P ≤ 0.10; *P ≤ 0.05; ** P ≤ 0.01; *** P ≤ 0.001

Table 5 (continued) Binary logistic regressions stratified by housing tenure for the effects of asthma diagnosis and covariates on preschool-aged children's blood lead level status for the total sample (n = 1855).

	Own (n = 872)							
	Model 1		Model 2		Model 3		Model 4	
	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)
Control variables								
Survey cohort	0.71	(0.59-0.86)**	0.70	(0.57-0.85)***	0.70	(0.57-0.85)**	0.69	(0.57-0.84)***
Age (ref=Age 5)								
Age 4	1.78	(0.89-3.54)	1.76	(0.91-3.43)	1.85	(0.94-3.64)†	1.91	(0.97-3.74)†
Age 3	2.36	(1.21-4.58)*	2.47	(1.27-4.79)*	2.73	(1.41-5.30)**	2.70	(1.38-5.29)**
Age 2	3.48	(1.95-6.20)***	3.82	(2.16-6.75)***	4.00	(2.20-7.29)***	4.12	(2.26-7.51)***
Age 1	3.31	(2.03-5.39)***	3.67	(2.26-5.98)***	4.08	(2.50-6.66)***	4.21	(2.59-6.86)***
Gender (ref=female)								
Male	1.21	(0.86-1.71)	1.30	(0.93-1.82)	1.29	(0.91-1.83)	1.31	(0.92-1.86)
Insurance status (ref=no insurance)								
Have health insurance	0.42	(0.21-0.87)*	0.40	(0.21-0.80)*	0.52	(0.25-1.05)†	0.53	(0.26-1.08)†
Asthma diagnosis								
Ever had asthma	1.47	(0.81-2.69)	1.52	(0.82-2.81)	1.51	(0.81-2.81)	1.39	(0.74-2.62)
Housing characteristics								
Age of home (ref=1978 and newer)								
Pre-1978			2.33	(1.56-3.48)***	2.26	(1.51-3.39)***	2.24	(1.51-3.33)***
Don’t know age of home			3.57	(1.68-7.62)**	2.48	(1.10-5.60)*	1.88	(0.79-4.48)
Sociodemographic factors								
Poverty status (ref=Above threshold)								
Below threshold					2.13	(1.51-3.03)***	2.08	(1.49-2.92)***
Race (ref=non-Hispanic white)								
Non-Hispanic black							2.41	(1.58-3.67)***
χ^2	44.85***		26.13***		17.90***		16.10***	
AIC_{min}=833.19	799.56		784.50		770.42		765.81	

† $P \leq 0.10$; *$P \leq 0.05$; ** $P \leq 0.01$; *** $P \leq 0.001$

Discussion

This study examined comorbidity of lead and asthma and the association of these conditions with sociodemographic and housing characteristics. The first question was whether lead poisoning and asthma were comorbid conditions among preschool-aged children. The primary finding was that EBLLs and asthma diagnosis were associated for the full sample of children. The second question asked was whether racial and/or socioeconomic disparities were evident for both lead poisoning and asthma. There were common risk factors for lead and asthma separately such that children who were non-Hispanic black, low socioeconomic status, and lived in homes that were rented and the age of the home was unknown were at higher risk for both. Where risks of lead and asthma showed different patterns included age, health insurance status, gender, and age of the home (when age was known). Children at risk for lead tended to be younger and live in older housing while children at risk for asthma tended to be older, male, have health insurance, and live in newer housing.

The final question asked whether housing characteristics of where children lived had effects on lead and asthma. Age of housing was not associated with comorbidity as expected, due to the different patterns of associations between age of housing and EBLLs compared to the association between age of housing and asthma diagnosis. In the former comparison, housing that was older than 1978 or where age of housing was unknown was associated with higher rates of asthma among children. This was in the expected direction based on previous literature regarding routes of lead exposure for preschool-aged children and age of housing (Jacobs et al., 2002; Levin et al., 2008). For children who spend more of their time in the home environment, their primary exposure may be housing in which there is lead paint, specifically creating hazards through chipping paint and lead dust within the household. Houses built prior to 1978 have the

highest risk of containing lead-based paint, with risk of exposure increasing with the age of the home (Environmental Protection Agency, 2014; Jacobs et al., 2002). Age of housing was not associated with asthma diagnosis in the same way. Instead, asthma diagnosis was higher for children in newer homes compared to homes built prior to 1978 although asthma rates were also high among children where age of housing was unknown. Rates of lead, asthma, and the comorbid condition were all higher among children who lived in homes where the age was unknown.

For cases in which the age of housing was unknown, the majority of respondents also reported that they did not own their home. It could be argued that housing tenure is a proxy for material circumstances that are related to income or wealth, but there is also evidence that housing tenure may be more directly related to health because rented homes are more likely to be of lower quality and contain associated environmental hazards than homes that are owned (Ellaway & Macintyre, 1998; Leventhal & Newman, 2010). The common thread for comorbidity of lead and asthma, therefore, may still be the condition of housing but not necessarily linked directly to the age of the home. More direct measurements of housing hazards (e.g., chipping paint, dampness, and mold) would be useful in determining how housing quality is directly related to health outcomes in children, but the current data does not contain these items. Housing tenure may be a suitable proxy for quality of housing. When the models were stratified by housing tenure, it was evident that ever having an asthma diagnosis predicted an elevated blood lead level but only for children living in rented homes. This association was not significant for children living in homes that were owned.

In the full sample, after controlling for housing characteristics, poverty status further reduced but did not completely eliminate the association between lead and asthma.

Socioeconomic disparities have been shown in the literature for both lead poisoning (Pamuk et al., 1998) and asthma (Gold & Wright, 2005). Additionally, poverty status reduced the effect of unknown age of housing. This would indicate that socioeconomic status may partially explain the association between lead and asthma as well as explaining the effect of unknown age of housing. In the current analyses, there was a higher proportion of children living in homes where age of housing was unknown as well as living in homes that were not owned. It may be that these homes are in greater disrepair as compared to homes that are owned. Low-income children are more likely to live in homes that are not owned and are disadvantaged by a number of measures of housing quality (e.g., mold, dampness, crowding) (Evans, 2004, 2006). In the stratified models, there was no longer a significant association between asthma diagnosis and EBLL for children living in rented homes once poverty status was entered into the model.

Adding race to the model after including poverty status and housing characteristics completely removed the association between lead and asthma. Although race and socioeconomic status are associated, they are not interchangeable. In the current sample and in the existing literature, black children are at the highest risks for both lead poisoning and asthma diagnosis, especially when they are low-income. Children from both low socioeconomic and black neighborhoods face a higher proportion of environmental hazards related to child health, and low-income, black children may be at the largest disadvantage due to patterns of residential segregation that result in concentrated poverty (Oyana & Margai, 2007; Vivier et al., 2011; Williams & Collins, 2001; Williams, Sternthal, & Wright, 2009). In turn, this could lead to low-income, black children living areas in which there are higher proportions of older or substandard housing in addition to employment limitations for parents.

Alternative mechanisms for environmental exposure

Although the current study was focused on the age of the home as the primary predictor of lead and asthma, one alternative is that lead and asthma comorbidity may be determined by other individual- or neighborhood-level factors. Recently, more attention has been given to occupational exposures to lead. Children may be exposed to risk factors for both lead and asthma at multiple levels. At the individual level, they may be exposed to lead and respiratory irritants via their parents' occupation (Levin et al., 2008; Tong & Lu, 2001; Vivier et al., 2011). Parents may be bringing in lead and other particles into the home from their clothes.

Additionally, children may be exposed to lead and respiratory irritants if their neighborhoods are situated in areas of worse air quality and pollution. Asthma prevalence has been found to be associated with nitrogen dioxide, a marker of traffic-related air pollution, as well as lead, carbon monoxide, particulate matter, and other pollutants (Brauer et al., 2007; Jerrett et al., 2008; Kim, 2004; McConnell et al., 2006). A recent study in the U.S. confirmed an association between levels of lead present in the air and children's individual lead levels such that higher content of lead in the air predicted children having lead levels above 10 µg/dL (Brink et al., 2013). According to the U.S. Environmental Protection Agency, national lead emissions in the air are mostly the result of mobile (e.g., on-road vehicles and non-road equipment) and industrial processes (e.g., cement and chemical manufacturing, mining). Proximity to major roads and to particular industrial sites may lead to comorbid conditions of lead poisoning and asthma. These types of environmental exposures may also be worse in areas already disadvantaged by concentrated poverty and segregation.

Air pollution is higher among neighborhoods in closer proximity to roadways or industrial complexes, and these neighborhoods are more likely to be low-income and minority (Chakraborty & Zandbergen, 2007). In a study of air pollution exposure among school-aged

children in Orange County, Florida, Chakraborty and Zandbergen (2007) identified racial disparities in the exposure to air pollution for children, both at home and at school. Exposure was identified as proximity to a number of sources, including industrial and other smaller stationary facilities and major roads. Both black and Hispanic children were disproportionately exposed to pollutants in their home and school neighborhoods, with the former carrying the highest risk of exposure.

Taken together, there is evidence in the literature of both individual- and neighborhood-level factors in the exposure to lead and asthma risk factors for children. Many authors have argued for a multi-level approach to environmental health outcomes (Soobader, Cubbin, Gee, Rosenbaum, & Laurenson, 2006; Williams et al., 2009; Wright & Subramanian, 2007). As previously discussed, there are obvious social patterns to the development of both lead poisoning and asthma in that low-income and black children are disproportionately at risk. Developing a model that incorporates both individual-level risk factors (e.g., sociodemographic characteristics, quality of home) and higher-level neighborhood risk factors (e.g., residential segregation, occupation opportunities) may yield a better understanding of the racial/ethnic and socioeconomic disparities in lead, asthma, and their comorbid condition. Furthermore, this is an area of research that can be expanded by the use of geospatial techniques in order to look at spatial patterns of housing quality or proximity to roadways and industrial sites that may increase the likelihood of lead poisoning and asthma diagnosis among disadvantaged populations.

Limitations

One limitation of the current study is the lack specific risk factors in the NHANES data that is generally found to be associated with both lead poisoning and asthma, including measures of parental occupation, housing quality, and specific points of exposure to lead and allergens

(e.g., dust levels, carpeting, and clay pots). If these were available, more direct measures of the associations between environmental hazards and lead poisoning and asthma could be constructed. A related limitation for this kind of study is the availability of data on both asthma and lead while including multiple characteristics that may predict comorbidity. At the individual level, NHANES is the only data set that includes both measured lead levels and asthma diagnosis for children. However, the public-use data file is limited in that geospatially-based predictors of health cannot be addressed, although some geoidentifiers are available in the restricted data set. In general, there is very little availability of data that allows a connection between environmental factors and health outcomes, especially for any kind of spatial analysis (Maantay & McLafferty, 2011).

Policy implications

Traditionally, lead and asthma interventions have focused on adjusting risk factors within the home environment. Interventions have been developed under the umbrella of "healthy housing" which focuses on risk factors for child health within the indoor home setting. For lead poisoning, some of the common interventions include education regarding cleaning of paint chips and dust, personal hygiene and clothes cleaning for certain occupation types, nutrition education, and lead remediation and abatement (Harvey, 2002). Similarly, interventions for asthma include the use of integrated pest management, home cleaning techniques, and education about routes of exposure for common allergens (U.S. Department of Health and Human Services, 2007). Although these types of interventions may show some success, they are not addressing the broader distal factors that produce housing and health inequalities.

On a broader level, it has been suggested to use a market-based approach to educate both the home inhabitants as well as landlords and homebuilders in the association between housing

and health with additional information regarding cost effectiveness for the latter group (Breysse et al., 2004). Including other stakeholders, such as medical professionals and government agencies, in the conversation regarding the high priority of housing hazards and health may lead to better coordination of efforts.

Additionally, improved monitoring of lead levels among patients and at the aggregate level is necessary in order to better understand the prevalence of lead poisoning among children. Currently, lead testing of children is mostly limited to those on Medicaid, since it is a requirement of well-child checkups. However, there may be a number of children who are not on Medicaid but still may be at a disadvantage for exposure to lead and are not being tested by a medical professional. Lead poisoning, especially at very low levels, is very asymptomatic and may be missed until later in life when developmental and cognitive consequences are more dramatic (Chiodo et al., 2004). One proposal is to expand testing of lead to well-child checkups for all preschool-aged children. The CDC has long recommended universal screening of preschool-aged children, especially emphasizing screening among children who are low-income and/or live in substandard housing (CDC, 1997; Wengrovitz & Brown, 2007). Additionally, based on national data showing that Medicaid-eligible children had higher rates of lead, lead testing is required at 9 months and 2 years for Medicaid-eligible children. However, since both national and local patterns are no longer seeing the higher lead rates among Medicaid-eligible children (Wengrovitz & Brown, 2007), it may be better to target all children based on particular risk factors (e.g., living in old housing, parents with particular occupations, environmental lead proximity) of all preschool children rather than implementing Medicaid eligibility as a primary risk factor for lead. This would require commitment from multiple community stakeholders that may include local lead surveillance programs, children's primary care physicians, and parents. It

may also be useful to include preschool programs and school settings as potential sites for lead screening using lead capillary tests (i.e., a test that is cheaper and easier to administer than a venous draw) in order to improve early identification of children at risk for lead poisoning.

Monitoring of lead data also needs to improve at county, state, and national levels. Monitoring of area-level rates of elevated lead levels is handled at the county level and reported to the state. Additionally, although the CDC receives data from some states, they are not legislatively mandated to collect data pertaining to lead poisoning (personal communication, 3/7/14). Additionally, the data that is provided is generally outdated (usually more than three years old) and does not contain updated data regarding lead levels at the new threshold. By providing better monitoring of lead levels among patients as well at population-levels, rates of lead poisoning can be addressed sooner.

Nationally, rates of lead poisoning are relatively low (Wheeler & Brown, 2013), but it is possible that rates are actually underestimated due to the chosen measurement cutoff. Based on recent NHANES data, 3% of children have lead levels at or above 5 µg/dL, but the current NHANES data shows that about 30% of children have lead levels at or above 2 µg/dL. CDC recently changed the reference level of lead from 10 µg/dL to 5 µg/dL, which is a step in the right direction. However, there is research showing that even lower levels (as low as 2 µg/dL) have shown negative long-term effects for children's cognitive and behavioral functioning (Bellinger, 2004; Canfield et al., 2003; Chiodo et al., 2004). Additionally, although the reason is not yet known, there is may be an indication that children's lead levels are more associated with asthma symptoms at lower levels (Joseph et al., 2005). On a related note, most health outcome monitoring seems to be limited to a single health measure. However, monitoring of multiple conditions together (here, lead levels and asthma) may assist to improve the general state of

knowledge regarding why these conditions may be comorbid and what factors may be related to both. It may also shed light on the relationship between asthma and lead at varying lead levels.

Generally, there is little research regarding social inequities and environmental health, except in the case of lead poisoning (Northridge, Stover, Rosenthal, & Sherard, 2003). Future research needs to explore potential comorbid conditions as they are impacted by environmental hazards at multiple levels (e.g., individual, neighborhood, county). In this way, researchers can better understand how environmental hazards impact different diseases along common pathways, and how these pathways may be socially patterned by race and poverty.

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