Validation of a 20-year forecast of US childhood lead poisoning: Updated prospects for 2010

David E. Jacobs*, Rick Nevin

National Center for Healthy Housing, USA

Abstract

We forecast childhood lead poisoning and residential lead paint hazard prevalence for 1990–2010, based on a previously unvalidated model that combines national blood lead data with three different housing data sets. The housing data sets, which describe trends in housing demolition, rehabilitation, window replacement, and lead paint, are the American Housing Survey, the Residential Energy Consumption Survey, and the National Lead Paint Survey. Blood lead data are principally from the National Health and Nutrition Examination Survey. New data now make it possible to validate the midpoint of the forecast time period. For the year 2000, the model predicted 23.3 million pre-1960 housing units with lead paint hazards, compared to an empirical HUD estimate of 20.6 million units. Further, the model predicted 498,000 children with elevated blood lead levels (EBL) in 2000, compared to a CDC empirical estimate of 434,000. The model predictions were well within 95% confidence intervals of empirical estimates for both residential lead paint hazard and blood lead outcome measures. The model shows that window replacement explains a large part of the dramatic reduction in lead poisoning that occurred from 1990 to 2000. Here, the construction of the model is described and updated through 2010 using new data. Further declines in childhood lead poisoning are achievable, but the goal of eliminating children's blood lead levels $\geqslant 10\,\mu g/dL$ by 2010 is unlikely to be achieved without additional action. A window replacement policy will yield multiple benefits of lead poisoning prevention, increased home energy efficiency, decreased power plant emissions, improved housing affordability, and other previously unrecognized benefits. Finally, combining housing and health data could be applied to forecasting other housing-related diseases and injuries.
© 2006 Elsevier Inc. All rights reserved.

Keywords: Lead; Lead paint; Childhood lead poisoning; Housing; Healthy housing; Forecast methodology; Windows; Energy conservation; Health care; Policy

1. Introduction

It is well established that children under age 6 are especially vulnerable to lead exposure because their nervous systems are still developing (National Academy

---

*Corresponding author.

E-mail address: dejacobs@starpower.net (D.E. Jacobs).

of Sciences, 1993). While elevated blood lead levels (EBL) $\geqslant 10\,\mu g/dL$ are clearly associated with harmful effects on children's learning and behavior, there is currently no lower threshold for some of the observed adverse effects of lead in children (US Centers for Disease Control and Prevention, 1991, 1997). Childhood blood lead levels below $10\,\mu g/dL$ have been associated with intellectual impairment (Canfield et al., 2003). In addition, there are data suggesting that early childhood lead exposure may be associated with delinquent and criminal behavior among juveniles and young adults (Denno, 1990; Dietrich et al., 2001; Needleman et al., 1996; Nevin, 2000), although no clear dose–response relationship has been established for this effect.

The two main sources of childhood lead exposure in the United States during the 20th century were leaded gasoline

and lead paint (Agency for Toxic Substances and Disease Registry, 1988; Clark et al., 1991; Jacobs, 1995). While lead poisoning can be caused by inhalation of airborne particulate lead, ingestion of lead paint chips, and occasionally other sources, the main childhood exposure pathway is from lead-contaminated dust that settles on horizontal surfaces, such as floors and window sills, and is then ingested via normal hand-to-mouth contact (Bornschein et al., 1987; Duggan and Inskip, 1985; Lanphear et al., 1995, 1998). Before leaded gasoline was banned, children were also exposed to dust lead from settling gasoline emissions. Older homes with interior lead paint are especially likely to have lead dust hazards if the lead paint has deteriorated (Jacobs et al., 2002), but lead dust hazards may also be created by lead paint on friction and impact surfaces, such as windows, and by home renovation that disturbs lead paint without appropriate dust containment and cleanup procedures (President's Task Force on Environmental Health Risks and Safety Risks to Children, 2000).

1.1. Trends in childhood lead poisoning

The percentage of EBL children under age six fell from 88% during the Second (1976–1980) National Health and Nutrition Examination Survey (NHANES) to 9% during NHANES III phase 1 (1988–1991) (Pirkle et al., 1994). This decline revealed the public health impact of regulatory actions to remove lead from gasoline, new paint, and food and beverage can solder. But the 1988–1991 data showed that 1.7 million American children under age six still had EBLs. The sale of lead paint for residential use was banned in 1978, but a large body of research shows that lead paint hazards in older homes are now the most important remaining source of childhood lead exposure today (National Academy of Sciences, 1993, US Centers for Disease Control and Prevention, 1991, 1997; President's Task Force on Environmental Health Risks and Safety Risks to Children, 2000). By statute, the term "lead-based paint hazard" includes deteriorated lead paint $\geqslant 1 \, mg/cm^2$, as well as lead above certain levels in settled housedust and bare soil (US Environmental Protection Agency, 2001; US Department of Housing and Urban Development, 1999b).

EBL prevalence for American children under six declined to 4.4% during NHANES III phase 2 (1992–1994), but those same data showed an EBL prevalence of 16.4% among low-income children and 22% among African-American children living in houses built before 1946 (US Centers for Disease Control and Prevention, 1997). EBL prevalence for all children under age six fell further to 1.6% during the 1999–2002 NHANES (Brody et al., 2005). The ongoing decline in EBL prevalence is confirmed by CDC surveillance data (Meyer et al., 2003) that reflect blood lead tests for about 7–8% of children under age 6 in each year from 1997 to 2001 and account for a larger share of EBL children because surveillance programs target low-income areas

with older, substandard housing and higher EBL prevalence. Even within this at-risk population, EBL prevalence (as a percentage of children tested) declined from 7.66% in 1997 to 3.01% in 2001, although the disparity between low-income minority children and other children was still large (Meyer et al., 2003). Despite this progress, the 2000 national goal of eliminating blood lead levels in young children above 25 µg/dL was not achieved (Meyer et al., 2003). In short, lead poisoning, primarily but not exclusively from lead paint hazards in housing, still remains a major childhood environmental disease in the United States.

1.2. Lead paint regulatory actions

In 1992, Congress passed Title X of the Housing and Community Development Act, also known as the Residential Lead Hazard Reduction Act (Public Law 101-550; 42 USC 4851 *et seq.*). Title X authorized new programs regarding public education, standardized inspection and hazard control procedures (US Department of Housing and Urban Development, 1995), required disclosure of known lead paint hazards in most pre-1978 housing, provided funding from the US Department of Housing and Urban Development (HUD) to eliminate lead paint hazards in privately owned low-income housing, and performed other actions. Furthermore, HUD implemented lead paint regulations and released technical guidelines on lead hazard identification and control in public and Indian housing in 1990, both of which likely spurred remedial action on the part of both housing agencies and private owners during that decade. Regulatory and other efforts by some state and local governments also accelerated during the 1990s (Guthrie and McLaine, 1999). In addition, Title X prescribed lead paint hazard control activities for all federally assisted housing (not only public and Indian housing), but HUD did not issue new regulations for federally assisted housing until 1999 (US Department of Housing and Urban Development, 1999b). While all these actions likely had a positive influence, the decline in childhood lead poisoning during the 1990s cannot be explained solely by regulatory changes in assisted housing, because such housing constitutes only a small fraction of the nation's housing stock.

In 2000, the federal government released the first interagency plan on childhood lead poisoning, under the auspices of the President's Task Force on Children's Environmental Health and Safety Risks (President's Task Force on Environmental Health Risks and Safety Risks to Children, 2000). The plan included a forecast model for lead paint hazard and EBL prevalence for 1990–2010. The model is based on NHANES blood lead data combined with data on lead paint, housing demolition, window replacement, and household characteristics derived from three different housing data sets.

New data now validate the midpoint of the model forecast, with important implications for lead poisoning

prevention and for environmental health research. The model shows that a window replacement policy will yield multiple benefits, including lead poisoning prevention, increased home energy efficiency, and other benefits. (Energy-inefficient single-pane windows in older houses are especially likely to have lead paint on interior window surfaces and associated lead dust hazards.)

This paper explains how the model was constructed; compares the forecast with empirical estimates; updates the forecast using new housing data; presents new housing data confirming that single-pane window replacement explains a large part of the 1990–2000 reduction in lead poisoning; and examines broader implications for environmental health research. To our knowledge, this is the first time that important public health trends have been accurately anticipated based on analysis of housing data. Similar analytical methods hold promise for improving our understanding of the linkage between other housing conditions and adverse health outcomes.

2. Methods and data sources

The forecast model was constructed in two main parts. The first calculated the changes in the number of housing units by year built (i.e., year of construction) and two categories of lead paint hazard risk ("high" or "low") for 1989–2010. The second part of the model linked that housing risk forecast to NHANES data to calculate the number of EBL children in each year from 1993 to 2010.

The model was first constructed in 1999. At that time, the most recent data on EBL prevalence and residential lead paint hazards were from 1992 to 1994 NHANES III (Pirkle et al., 1994) and the 1989–1990 National Lead Paint Survey (NLPS) (US Department of Housing and Urban Development, 1990), respectively. The model combined these data with housing demolition and window replacement rates derived from the 1989–1997 (five biennial) American Housing Surveys (AHS) (US Bureau of the Census and US Department of Housing and Urban Development, 2000) and the 1993 Residential Energy Consumption Survey (RECS) (US Department of Energy, Energy Information Administration, 1995).

The demolition of older houses reduces lead paint hazards and EBL prevalence because lead paint was widely used on interior and exterior surfaces and demolition eliminates such surfaces. NLPS and RECS data also showed that window replacement is a good indicator of housing rehabilitation that is likely to remove lead paint and the most severe lead dust hazards. For example, NLPS data showed that 17% of pre-1940 units had no interior lead paint in 1989 (US Department of Housing and Urban Development, 1990), and RECS data showed that 13% of pre-1940 units had all windows replaced prior to 1990 (US Department of Energy, Energy Information Administration, 1995). This suggests that most pre-1940 units without any interior lead paint in 1989 had probably removed interior lead paint through substantial rehabilitation including window replacement.

In addition to serving as an indicator of extensive rehabilitation and ongoing property maintenance, window replacement was also directly linked to reducing lead paint hazards. The NLPS showed that windows were the housing component with the highest levels of lead dust (US Department of Housing and Urban Development, 1990). Lead dust on horizontal window surfaces is also significantly correlated with children's blood lead levels (Lanphear et al., 1995). The national evaluation of the HUD lead hazard reduction grant program, a longitudinal study involving over 3000 dwellings in a dozen jurisdictions, also showed that window replacement is a common and effective hazard control strategy adopted by many local governments (National Center for Healthy Housing and University of Cincinnati Department of Environmental Health, 2004).

The effectiveness of window replacement in controlling lead dust hazards was well established when the model was developed (US Department of Housing and Urban Development and Office of Lead Hazard Control, 1999a). Preintervention median dust lead loadings in rooms treated with paint stabilization *and* window replacement were 60% higher than in rooms treated with paint stabilization and window repairs, and over three times higher than median dust lead loadings in rooms treated only with paint stabilization. Rooms that underwent window replacement had postintervention dust lead loadings that were significantly lower than dust lead loadings in rooms where window lead paint was only repaired. Rooms that underwent window replacement also had dust lead loadings significantly lower than the dust lead loadings in rooms with just paint stabilization 1 yr after intervention (US Department of Housing and Urban Development, 1999a). More recent data show dust lead loadings in units with window replacement 3 years after intervention (National Center for Healthy Housing and University of Cincinnati Department of Environmental Health, 2004) and 6 years after intervention (Wilson et al., accepted for publication) were significantly lower than in units without window replacement.

2.1. Definitions of high- and low-risk housing

Units with interior lead paint in 1989 were forecast to follow one of three paths that would determine the risk of those units having lead paint hazards through 2010. Some would undergo window replacement and ongoing property maintenance, resulting in a relatively low risk of lead paint hazards. Other units with interior lead paint would be demolished. The third path was that the units would remain occupied without window replacement, resulting in a relatively high risk of lead paint hazards over the 20-yr forecast horizon.

The term "high risk" used here should not be confused with the regulatory definition of lead paint hazards. Lead paint hazards are identified at a given housing unit at the time of its risk assessment, whereas the model forecast the risk of such hazards over a 20-yr time horizon. Intact interior lead paint, by itself, does not constitute a lead paint hazard from a regulatory standpoint. But the model defined high risk units in 1989 to include all units with interior lead paint, whether intact or deteriorated, because houses with interior lead paint had a higher risk of developing lead paint hazards (including lead dust hazards) over the 20-yr forecast horizon. Similarly, the term "low risk", as used in this model, does not necessarily mean that there is no risk; instead, it simply means that such units posed comparatively less risk than the high-risk units.

Because interior lead paint, window replacement, and demolition are all more common in older housing, the model used four housing age categories to further define risk: pre-1940, 1940–1959, 1960–1974, and post-1974. AHS data were available for each of these categories and similar housing age categories were available for NLPS and NHANES data. NHANES provided EBL prevalence data in pre-1946, 1946–1973, and post-1973 housing. The model assumed that EBL data for pre-1946 housing were representative of all pre-1940 units, because very little housing was built from 1940 to 1945 during World War II. Blood lead data for 1946–1973 housing were used to characterize EBL prevalence in 1940–1974 housing, because the two time spans are similar. NLPS data on lead paint and lead paint hazards were available for pre-1940, 1940–1959, and 1960–1977 housing. The model used the 1960–1977 NLPS data to characterize 1960–1974 housing, due to the similarity of time spans. Finally, all post-1974 housing was defined as low risk, because comparatively little lead paint was sold in the last years before it was banned in 1978 (Nevin, 2000) after initial efforts to ban lead paint for residential use began in 1971 (US Consumer Product Safety Commission, 1977). We have chosen to use the term "low-risk" for this category of housing, because there may still be some potential for exposure due to informal retailing and sale of existing lead paint stocks from store shelves following the 1978 ban on new production of residential lead-based paint.

The 1993 RECS provided data on the percentage of housing units that had all windows replaced through 1993, but window replacement data were not collected in the 1997 (or 2001) RECS. The 1995 and 1997 AHS

reported the number of units with windows and doors replaced from 1994 to 1997 and the dollar amount spent on each upgrade, but the AHS did not collect equivalent data before 1995. Therefore, the model combined RECS and AHS data to estimate the percentage of high-risk units that had most or all of their windows replaced in any given year.

2.2. Definitions of household categories for EBL forecast

The second part of the model linked the housing risk forecast with 1992–1994 NHANES data and 1997 AHS data on family income and the number of children under age 6 per occupied unit. Temporal changes in the number of EBL children were calculated for households characterized by family income, housing risk, and housing age. AHS data on family poverty-to-income ratio (PIR below 1.3) were used to characterize family income because that threshold is consistent with the NHANES data and roughly consistent with many HUD assistance programs. PIR is defined as household income divided by the level of income needed to meet the federal definition of poverty. For this model, a PIR greater than 1.3 means that a household had an income that was more than 130% of the poverty level. A decline in EBL prevalence within each housing category was forecast based on the overall decline in the percentage of housing characterized as high risk. The number of EBL children each year was then forecast for 14 different household categories (for PIR above and below 1.3 in each of seven housing risk/age categories).

2.3. Housing risk forecast

Table 1 shows the model forecast for selected years from 1989 to 2000 in seven distinct categories of housing: 3 year-built categories of high-risk (HR) housing units and 4 year-built categories of low-risk units. The parameters used to forecast changes in housing risk were the annual rates of window replacement (W) and demolition (D) in pre-1975 housing and net growth (construction minus demolition) in post-1974 housing.

NLPS data on units with and without interior lead paint were used to calculate the number of pre-1975 high- and low-risk units, respectively, in 1989. AHS data were used to estimate the total number of post-1974 units. The forecast increase in post-1974 housing units reflects a constant net growth (new construction minus demolition) of 3.7% per year, which is the average of 1989–1997 AHS data. The forecast decline in pre-1975 high-risk housing is due to the combined effects of demolition and window replacement. A forecast increase in pre-1975 low-risk housing reflected low-risk unit demolition that is more than offset by an increase in low-risk

units due to window replacement in high-risk units, because window replacement moves high-risk units into the low-risk category.

The equations and definitions used to generate Table 1 are as follows:

1989 high-risk (HR) units
$$= \text{Pre-1975 units with interior lead paint (from HUD 1990),} \quad (1)$$

1989 low risk (LR) units
$$= \text{Pre-75 units without interior lead paint (from HUD 1990)}$$
$$+ \text{post-74 units (from AHS).} \quad (2)$$

For each year after 1989 (separate calculation by year built):

HR housing in Year$_{t+1}$
$$= \text{HR housing in Year}_t - D_{\text{HR}} - W_{\text{HR}}; \text{ where} \quad (3)$$

$$D_{\text{HR}} = (\text{high risk demolition rate, } D\%)$$
$$\times (\text{HR housing in Year}_t), \quad (4)$$

$$W_{\text{HR}} = (\text{high risk window replacement rate, } W\%)$$
$$\times (\text{HR housing in year}_t). \quad (5)$$

Pre-1975 LR housing in Year$_{t+1}$
$$= \text{LR housing in Year}_t - D_{\text{LR}} + W_{\text{HR}}; \text{ where} \quad (6)$$

$$D_{\text{LR}} = (\text{low-risk demolition rate, } D\%)$$
$$\times (\text{LR housing in year}_t), \quad (7)$$

$$W_{\text{HR}} = \text{high-risk window replacement}$$
$$\text{units, calculated above for same age of housing.} \quad (8)$$

Post-74 housing in year$_{t+1}$
$$= 1.037 \times (\text{post-74 housing in year}_t). \quad (9)$$

2.4. Demolition rates by housing risk category

The model used a demolition rate for all low-risk housing of 0.4% per year, regardless of year built, which is slightly below the average 0.5% demolition rate reported by 1989–1997 AHS data for all 1960–1974 units. This slightly lower rate was used because many of these low-risk units had been substantially rehabilitated. Demolition rates for each age category of high-risk units were then calculated using a weighted average of high- and

Table 1
High- and low-risk housing forecast through 2000

	Annual rate of change			Housing units (millions)				
	Window replacement %	Demolition %	Total %	1989	1993	1994	1999	2000
High-risk units (year of construction)								
Pre-1940	−1.85	−0.95	−2.80	17.3	15.4	15.0	13.0	12.6
1940–1959	−1.85	−0.80	−2.65	14.4	13.0	12.6	11.0	10.7
1960–1974	−1.50	−0.60	−2.10	12.5	11.5	11.2	10.1	9.9
Total high-risk units				**44.2**	**39.9**	**38.8**	**34.1**	**33.2**
Low-risk units								
Pre-1940		−0.4		3.5	4.7	5.0	6.2	6.4
1940–1959		−0.4		6.5	7.4	7.6	8.6	8.7
1960–1974		−0.4		13.0	13.5	13.6	14.2	14.3
Post-1974		+3.7 (net)		26.5	30.7	31.8	30.7	31.8
Total low-risk units				**49.5**	**56.3**	**58.0**	**67.1**	**69.0**
Percentage high-risk				**47.2%**	**41.5%**	**40.1%**	**33.7%**	**32.5%**

Table 2
Derivation of high- and low-risk demolition rates

HR% = 1989 high-risk percentage of all housing, by age of housing
LR% = 1989 low-risk percentage of all housing, by age of housing (= 1-HR%)
D%-All = Overall demolition rate, by age of housing
D%-All = (HR% × D%$_{HR}$) + (LR% × D%$_{LR}$); where
  D%$_{HR}$ = high-risk demolition rate, by age of housing
  D%$_{LR}$ = low-risk demolition rate, by age of housing = 0.4%

1960–1974	1940–1959	Pre-1940
HR% = 49%	HR% = 69%	HR% = 83%
LR% = 51%	LR% = 31%	LR% = 17%
D%-All = 0.5%	D%-All = 0.68%	D%-All = 0.86%
D%$_{LR}$ = 0.4%	D%$_{LR}$ = 0.4%	D%$_{LR}$ = 0.4%
(0.49 × D%$_{HR}$) + (0.51 × 0.4) = 0.5	(0.69 × D%$_{HR}$) + (0.31 × 0.4) = 0.68	(0.83 × D%$_{HR}$) + (0.17 × 0.4) = 0.86
Therefore, D%$_{HR}$ = 0.6%	Therefore, D%$_{HR}$ = 0.8%	Therefore, D%$_{HR}$ = 0.95%

Table 3
Derivation of window replacement rates in high- and low-risk housing

HR% = 1989 high-risk percentage of all housing, by age of housing
LR% = 1989 low-risk percent of all housing, by age of housing (= 1-HR%)
W%-All = Overall window replacement rate, by age of housing
W%-All = (HR% × W%$_{HR}$) + (LR% × W%$_{LR}$); where
  W%$_{HR}$ = high-risk window replacement rate, by age of housing
  W%$_{LR}$ = low-risk window replacement rate, by age of housing

1960–1974	1940–1959	Pre-1940
HR% = 49%	HR% = 69%	HR% = 83%
LR% = 51%	LR% = 31%	LR% = 17%
W%-All = 1.25%	W%-All = 1.6%	W-All = 1.6%
W%$_{LR}$ = 1.0%	W%$_{LR}$ = 1.05%	W%$_{HR}$ = 1.85%
(0.49 × W%$_{HR}$) + (0.51 × 1.0) = 1.25	(0.69 × W%$_{HR}$) + (0.31 × 1.05) = 1.6	(0.83 × W%$_{HR}$) + (0.17 × 0.4) = 1.6
Therefore, W%$_{HR}$ = 1.5%	Therefore, W%$_{HR}$ = 1.85%	Therefore, W%$_{LR}$ = 0.40%

low-risk demolition rates, such that the overall demolition rate equaled the rate provided in the AHS. Table 2 shows the weighted average calculations used.

2.5. Window replacement rates by housing risk

RECS and AHS data showed that the window replacement rate during the 1990s was 1% per year in units built during the 1970s (President's Task Force on Environmental Health Risks and Safety Risks to Children, 2000). The model applied this 1% replacement rate to all low-risk 1960–1974 housing. The window replacement rate in low-risk 1940–1959 housing was assumed to be 1.05% (slightly above the rate for 1970s housing), and the model also assumed that the window replacement rate would be similar in all high-risk pre-1960 units. These assumptions were combined with overall window replacement rates from the AHS and RECS data to calculate low- and high-risk rates, by age of construction, where the weight for high-risk housing was the "1989 high-risk %" value, by age of housing. Table 3 shows the weighted average calculations used to derive window replacement rates, by year built, for high- and low-risk housing.

Only the high-risk window replacement rates derived from this analysis were used in further analysis, because window replacement in low-risk units would not change the assignment to the low-risk category.

2.6. Forecast for EBL children

Table 4 shows the 1992–1994 NHANES data on EBL prevalence by age of housing and PIR.

The model combined these NHANES estimates with the housing risk forecast to develop EBL prevalence estimates by age of housing, PIR, and housing risk category. This analysis assumed that EBL prevalence in pre-1974 low-risk units is equal to the EBL prevalence in post-1974 units. NHANES EBL prevalence estimates for pre-1974 housing reflect a weighted average of the prevalence in low- and high-risk housing, where the weights reflect the percentage of housing in each year-built category that was characterized as high-risk housing in 1994. The weighting factors were derived as follows:

$$\text{Pre-1940 HR\%} = 75\% \ (15 \text{ million out of } 20 \text{ million units}), \tag{10}$$

$$\text{Pre-1940 LR\%} = (1 - \text{Pre-1940 HR\%}) = 25\%, \tag{11}$$

$$\begin{aligned} &1940-1974\,\text{HR\%} \\ &= 53\% \ (24 \text{ million out of } 45 \text{ million units}), \end{aligned} \tag{12}$$

$$\begin{aligned} &1940-1974\,\text{LR\%} \\ &= (1 - 1940-1974\,\text{HR\%}) = 47\%. \end{aligned} \tag{13}$$

These weighting factors were used to derive EBL prevalence estimates for distinct EBL risk categories, characterized by family PIR, age of housing, and housing risk, defined as follows:

$$X1 = \text{EBL prevalence for children with PIR under 1.3 in low-risk housing} = 4.33\%, \quad (14)$$

$$X2 = \text{EBL prevalence for children with PIR above 1.3 in low-risk housing} = 0.22\%, \quad (15)$$

$$X3 = \text{BL prevalence for children with PIR under 1.3 in high-risk pre-40 housing,} \quad (16)$$

$$X4 = \text{EBL prevalence for children with PIR above 1.3 in high-risk pre-40 housing,} \quad (17)$$

$$X5 = \text{EBL prevalence for children with PIR under 1.3 in high-risk } 1940-74 \text{ housing,} \quad (18)$$

$$X6 = \text{EBL prevalence for children with PIR above 1.3 in high-risk } 1940-74 \text{ housing,} \quad (19)$$

$$X7 = \text{EBL prevalence for children with PIR under 1.3 in all pre-40 housing} = 16.37\%, \quad (20)$$

$$X8 = \text{EBL prevalence for children with PIR above 1.3 in all pre-40 housing} = 3.19\%, \quad (21)$$

$$X9 = \text{EBL prevalence for children with PIR under 1.3 in all } 1940-1974 \text{ housing} = 7.25\%, \quad (22)$$

$$X10 = \text{EBL prevalence for children with PIR above 1.3 in all } 1940-1974 \text{ housing} = 2.24\%. \quad (23)$$

The values for X1 (4.33%) and X2 (0.22%) reflect NHANES data for post-1973 housing, and values for X3–X6 were derived from the

NHANES data for X7–X10 as follows:

$$\text{Pre-1940 LR\%} \times X1 + \text{Pre-1940 HR\%} \times X3 = X7$$
$$\Rightarrow 0.25 \times 4.33 + 0.75 \times X3 = 16.37$$
$$\Rightarrow X3 = (16.37 - (0.25 \times 4.33))/0.75 = 20.38\%, \quad (24)$$

$$\text{Pre-1940 LR\%} \times X2 + \text{Pre-1940 HR\%} \times X4 = X8$$
$$\Rightarrow 0.25 \times 0.22 + 0.75 \times X4 = 3.19 = X8$$
$$\Rightarrow X4 = (3.19 - (0.25 \times 0.22))/0.75 = 4.18\%, \quad (25)$$

$$1940-1974 \text{ LR\%} \times X1 + 1940-1974 \text{ HR\%} \times X5 = X9$$
$$\Rightarrow 0.47 \times 4.33 + 0.53 \times X5 = 7.25$$
$$\Rightarrow X5 = (7.25 - (0.47 \times 4.33))/0.53 = 9.84\%, \quad (26)$$

$$1940-1974 \text{ LR\%} \times X2 + 1940-1974 \text{ HR\%} \times X5 = X10$$
$$\Rightarrow 0.47 \times 0.22 + 0.53 \times X6 = 2.24$$
$$\Rightarrow X6 = (2.24 - (0.47 \times 0.22))/0.53 = 4.00\%. \quad (27)$$

These calculations indicate an EBL prevalence of about 4% for children with PIR below 1.3 in low-risk housing (X1) and for children with PIR above 1.3 in high-risk housing (X4 and X6). The EBL prevalence for children with PIR above 1.3 in low-risk housing is only 0.22%. The EBL prevalence is much higher for children with PIR below 1.3 in high-risk housing: 20.38% for children in pre-40 housing and 9.84% for children in 1940–1974 housing.

Table 5 illustrates how the second part of the model linked the housing risk forecast with the EBL prevalence estimates by family PIR, age of housing, and housing risk to forecast 1993–2000 changes in EBL prevalence for young children in six household categories. The EBL prevalence estimates derived above, from 1992 to 1994 NHANES data, were entered for 1993. EBL declines were then forecast based on the assumption that EBL prevalence (EBL%) within each of the six categories would decline at a rate proportionate to the decline in the percentage of all housing units characterized as high-risk in that category.

The equation used in Table 5 is as follows:

$$\text{EBL\% in Year}_{t+1} = (\text{EBL\% in Year}_t) \times (\text{HR\% in Year}_{t+1}/\text{HR\% in Year}_t), \quad (28)$$

where HR% in Year$_t$ = (high-risk housing units in Year$_t$)/(all housing units in Year$_t$) from Table 1 HR% in Year$_{t+1}$ = (high-risk housing units in Year$_{t+1}$)/(all housing units in Year$_{t+1}$) from Table 1.

While the direct benefit of the decline in high-risk housing was reflected in the declining percentage of children living in high-risk units, the indirect benefit was reflected in the forecast decline in EBL prevalence within each housing risk category. This indirect benefit would reflect declining neighborhood lead paint hazards (e.g., deteriorating exterior lead paint) and reduced lead paint hazard exposure in other residential units visited by children (including units where child care is provided), both of which can increase the blood lead levels of children who have no lead paint in

Table 4
Prevalence of children under age 6 with blood lead levels $\geqslant 10\,\mu g/dL$ by poverty to income ratio (PIR) and housing year of construction

	Pre-1946 (%)	1946–1973 (%)	Post-1973 (%)
PIR $\leqslant 1.3$	16.37	7.25	4.33
PIR > 1.3	3.19	2.24	0.22

Table 5
Model forecast for prevalence of high-risk housing and childhood blood lead level $\geqslant 10\,\mu g/dL$ for selected years between 1993 and 2000

	1993 (%)	1994 (%)	1999 (%)	2000 (%)
Percent of housing that is high-risk (HR%)				
Pre-1940	77	75	68	66
1940–1959	64	62	56	55
1960–1974	46	45	42	41
Elevated blood lead prevalence (%) by housing age and poverty/income ratio				
Poverty/income ratio >1.3, high risk, Pre-1940	4.19	4.1	3.4	3.3
Poverty/income ratio >1.3, high-risk, 1940–1974	3.96	3.8	3.2	3.1
Poverty/income ratio >1.3, low-risk	0.22	0.2	0.2	0.2
Poverty/income ratio <1.3, high-risk, Pre-1940	20.38	19.7	16.6	16.0
Poverty/income ratio <1.3, high-risk, 1940–1974	9.84	9.5	8.0	7.7
Poverty/income ratio <1.3, low-risk	4.33	4.2	3.5	3.4

Table 6
Model forecast for number of children <6 with blood lead levels ⩾10 µg/dL by age of housing and poverty/income ratio (PIR)

Housing category	Number of children <6 per housing unit		Year			
			1993	1994	1999	2000
		Poverty/income ratio >1.3 (%)	Thousands of children PIR > 1.3			
High-Risk pre-1940	0.214	67.0	89	85	64	60
1940–1959	0.216	66.0	71	68	51	48
1960–1974	0.199	67.3	64	60	44	41
Low-risk pre-1940	0.214	67.0	1	1	2	2
1940–1959	0.216	66.0	2	2	2	2
1960–1974	0.199	67.3	4	4	3	3
Post-1974	0.249	77.7	14	14	13	13
		Poverty/income ratio <1.3 (%)	Thousands of children PIR < 1.3			
High-risk pre-1940	0.214	33.0	313	278	152	143
1940–1959	0.216	34.0	104	96	65	61
1960–1974	0.199	32.7	88	81	53	50
Low risk pre-1940	0.214	33.0	20	19	15	15
1940–1959	0.216	34.0	26	25	22	22
1960–1974	0.199	32.7	45	43	32	31
Post-1974	0.249	22.3	82	80	75	75
All children <6 with blood lead levels ⩾10 µg/dL (thousands)			925	857	593	565

Columns may not sum to totals due to rounding.

their own homes. This forecast methodology implicitly assumed that eliminating all high-risk housing would also end childhood EBL caused by exposure to lead paint hazards.

Table 6 illustrates how the model then combined the housing risk and EBL prevalence forecasts with AHS data on the average number of children under age six per unit and percentage with PIR above and below 1.3 (by age of housing) to forecast the number of EBL children in the 14 household categories, by housing risk, year built, and family income. The equations used in Table 6 are as follows:

EBL children with PIR < 1.3 in each of seven housing risk/age

categories = (Forecast units, from Table 1)

$\times$ (Children < 6 per unit) $\times$ (% PIR < 1.3) $\times$ (EBL%),   (29)

EBL children with PIR > 1.3 in each of seven housing risk/age

categories = (Forecast units, from Table 1)

$\times$ (Children < 6 per unit) $\times$ (% PIR > 1.3) $\times$ (EBL%).   (30)

To summarize, the forecast number of EBL children in each year was derived from the forecast number of housing units in each of the seven housing risk/age categories from Table 1; the forecast EBL prevalence for each of the six EBL risk categories in Table 5; and AHS data on the average number of children per housing unit and the percentage of children with PIR < 1.3 in each category. The total number of EBL children for each year was forecast by summing the 14 different household categories for children with PIR above and below 1.3 within each of the seven housing risk/age categories (Table 6).

3. Results

3.1. Validation of estimate of housing units with lead paint hazards

The model forecast a decline in high-risk housing from 44.2 million units in 1989 to 33.3 million in 2000, with window replacement and demolition accounting for 70% and 30% of this decline, respectively. The model forecast an increase in low-risk units from 49.5 million units in 1989 to 69 million in 2000. Window replacement in high-risk units accounted for almost 75% of the growth in low-risk units and net growth in post-1974 units accounted for the remaining 25%. The decline in high-risk units and the growth in low-risk units had a combined effect of reducing the percentage of all housing characterized as high-risk from 47.2% in 1989 to 32.5% in 2000 (Table 1).

The NSLAH (completed in 2000) revealed that the initial model was especially accurate in anticipating the extent of lead paint hazards in pre-1960 units, but less accurate for 1960–1978 units (Table 7). This disparity likely is due to the much smaller sample size of the NLPS, which had only 284 housing units, while the 2000 NSLAH had 831 units. A comparison of NLPS and NSLAH data (Table 8) suggests that the percentage of pre-1940 homes with interior lead paint declined over the 1990s from 83% to 79%; the percentage of 1940–1959 homes with interior lead paint declined from 69% to 46%; and the percentage of 1960–1974 homes with interior lead paint fell from 49% to just 16%. The decline in the percentage of pre-1960 homes with interior lead paint could be explained largely by housing demolition and rehabilitation, but the large decline in 1960–1978 units with interior lead paint is more likely a reflection of estimation error in the NLPS.

The 1960–1978 data from the larger NSLAH sample are also more consistent with historical data on the sale of

Table 7

Comparison of forecast for high-risk housing in 2000 with empirical estimate of number of housing units with lead paint hazards by year of construction (millions of housing units)

	Pre-1940	1940–1959	1960–1974/1978
Model forecast for 2000	12.6	10.7	9.9
Empirical estimate for 1999–2000 (from Jacobs et al., 2002)	11.8	8.8	2.3
95% confidence interval for empirical estimate	10.0–13.6	6.7–10.9	1.4–3.2

Table 8

Percentage of housing units with interior lead paint 1990–2000

	Pre-1940 (%)	1940–1959 (%)	1960–1978 (%)
1990 percentage of housing units with interior lead paint	83	69	49
2000 percentage of housing units with interior lead paint	79	46	16

Table 9

Total weight of lead paint in housing and lead paint production by housing age of construction (thousands of tons)

	Pre-1940	1940–1959	1960–1978
Total lead in house paint	255	75	45
White lead produced for paint	843	128	33
Percent of historic white lead use	30%	59%	136%

Table 10

Trend in number of children per occupied housing unit and percentage with poverty/income ratio < 1.3 by housing age from 1997 to 2001

Housing age	Number of children < 6/unit		%Poverty/income ratio < 1.3	
	1997	2001	1997	2001
Pre-1940	0.214	0.204	33.0%	33.0%
1940–1959	0.216	0.207	34.0%	27.0%
1960–1974	0.199	0.180	32.7%	28.5%
Post-1974	0.249	0.227	22.3%	17.4%

white lead (lead carbonate), the most common form of lead used in the production of lead paint. The NLPS data showed that total lead remaining in paint in 1960–1978 housing in 1989 was 36% *greater* than the total amount of white lead used in paint from 1960 to 1980, which of course is highly unlikely (Table 9) (President's Task Force, 2000).

3.2. Validation of estimate of EBL children

At the time when the model was first completed (1999), the 1997 AHS had the most recent data available to calculate the number of children under age 6 per occupied unit and the percentage of children with PIR below 1.3 (both by year of construction). The forecast assumed that these values would remain constant through 2010. However, more recent 2001 AHS data show that both the average number of children per occupied unit and the percentage with PIR < 1.3 actually declined from 1997 to 2001 (Table 10).

Finally, Table 11 compares the original and updated model forecasts for EBL children with 1999–2000 NHANES data. The original model forecast 565,000 EBL children in 2000, based on 1997 AHS data. But using the more recent 2001 AHS data on children per unit and percentage with PIR < 1.3 yields a lower forecast of 498,000 EBL children in 2000. This revised forecast is more consistent with the NHANES mean estimate of 430,000 EBL children in 1999–2000.

3.3. Lead dust and lead paint on interior window surfaces

In 2000, lead dust hazards (the most common pathway of childhood lead exposure) were present in 61% of homes with deteriorated interior lead paint; 33% of homes with interior lead paint in good condition; and only 6% of homes with no interior lead paint (Jacobs et al., 2002). NSLAH data also confirm the NLPS finding that windows are the housing component where lead paint is most likely to be found. To better characterize the relationship between lead dust hazards and lead paint on windows, NSLAH data for pre-1978 homes were divided into five distinct categories related to deteriorated interior lead paint:

1. Deteriorated interior lead paint *only* on window surfaces.
2. Deteriorated interior lead paint *only* on nonwindow surfaces.
3. Deteriorated interior lead paint on window *and* non-window surfaces.
4. *No* deteriorated interior lead paint, but lead paint on interior window surfaces.
5. *No* deteriorated interior lead paint, *and* no lead paint on interior window surfaces.

Table 11
Original and revised 2000 forecast for children with blood lead levels $\geqslant 10\,\mu g/dL$ compared to empirical estimate, by poverty/income ratio and housing category

Housing category	Original forecast for 2000 (thousands of children)			Revised forecast for 2000 (thousands of children)		
	Poverty/income ratio <1.3	Poverty/income ratio >1.3	Total	Poverty/income ratio <1.3	Poverty/income ratio >1.3	Total
High-risk: Pre-1940	143	60	203	136	57	193
1940–1959	61	48	109	46	50	97
1960–1974	50	41	91	39	40	79
High risk: total	253	148	402	222	147	369
Low-risk Pre-1940	15	2	17	15	2	16
1940–1959	22	2	24	17	2	19
1960–1974	31	3	35	25	3	28
Post-1974	75	13	88	53	13	66
Low-risk: total	143	20	164	109	20	129
Total	397	169	<u>565</u>	331	167	<u>498</u>
1999–2000 empirical Estimate (from National Health and Nutrition Examination Survey (NHANES), Meyer et al., 2003)				<u>434</u>		
1999–2000 empirical estimate, 95% CI				189–846		

Columns may not sum to totals due to rounding error.

Table 12
Window sill dust lead and interior lead paint condition

	Housing units with dust lead hazards		Median windowsill dust lead loading ($\mu g/ft^2$ in units with dust hazards)
	Number (millions)	Prevalence (row %)	
Housing units with deteriorated interior lead paint			
On windows and other interior surfaces	1.0	78	924
Only on windows	2.4	58	926
Only on interior surfaces other than windows	1.5	52	889
Homes without deteriorated interior lead paint			
With intact lead paint on interior window surfaces	4.7	40	725
Without intact lead paint on interior window surfaces	5.4	12	360

Within each of these categories, the prevalence of lead dust hazards and the median interior windowsill lead dust loading in units with dust hazards were calculated (Table 12). These data provide several insights into why window replacement rates proved to be such an accurate way of predicting lead paint hazards, particularly in pre-1960 housing. First, about 70% of units with deteriorated interior lead paint have deteriorated lead paint on interior window surfaces (3.4 million out of 4.9 million). Indeed, half of these units have deteriorated interior lead paint *only* on window surfaces. Second, the prevalence and the severity (median lead loading) of dust lead hazards are greatest in units with deteriorated lead paint on interior window surfaces.

Table 12 also shows that the prevalence and the severity of dust lead hazards in houses with intact lead paint on interior window surfaces and *no* deteriorated interior lead paint is almost as great as the prevalence and severity of lead dust hazards in units with deteriorated interior lead paint *only* on nonwindow surfaces. Furthermore, the total number of houses in this category with lead dust hazards (4.7 million) is almost as great as the number of homes with dust hazards that can be explained by deteriorated interior lead paint.

Finally, Table 12 shows that homes with *no* deteriorated interior lead paint and *no* lead paint on interior window surfaces actually account for about one-third of all units with lead dust hazards. But the prevalence of dust hazards in this category is much lower (12%), and the median lead dust loading on windowsills in these units ($360\,\mu g/ft^2$) is substantially lower. Dust lead hazards in these units could come from exterior lead paint, prior renovation work that
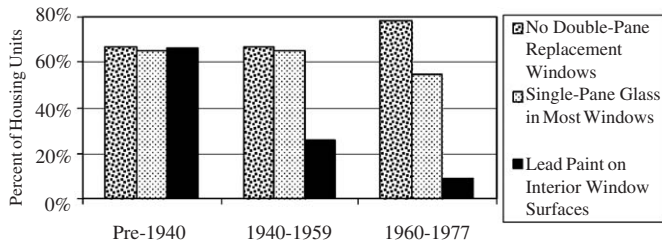
Fig. 1. Single-plane windows vs. lead paint on interior window surfaces.

Table 13
Percentage of nondisaster upgrade expenditures used for window replacement

	1997 American housing survey estimate (%)	2001 American housing survey estimate (%)
All assisted upgrades	9.1	13.3
Assisted upgrades in pre-1960 housing units	7.2	15.3
All unassisted upgrades	8.5	8.7
Unassisted upgrades in pre-1960 housing units	7.8	8.5

removed lead paint without adequate cleanup, and/or track-in from lead-contaminated soil or other exterior sources. (Houses with *no* deteriorated interior lead paint and *no* lead paint on interior window surfaces were characterized as low-risk houses in the Task Force model, with a low EBL prevalence that is consistent with relatively low dust lead loadings.)

3.4. Single-pane windows and windows with interior lead paint

Fig. 1 shows that single-pane windows in older homes are an indicator of lead paint on interior window surfaces (and thus an indicator of higher dust lead hazard prevalence and severity).

The NSLAH data show that about two-thirds of pre-1940 homes, 25% of 1940–1959 homes, and 10% of 1960–1977 homes have lead paint (intact or deteriorated) on interior window surfaces. The RECS and AHS data show that about two-thirds of pre-1960 homes had single-pane glass in most windows and *no* double-pane replacement windows at the time of the 1998–2000 NSLAH. Double-pane windows were not used in new home construction before 1960 (Fisette, 2003), so pre-1960 houses with double-pane glass in most windows have already had most or all original windows replaced. RECS data also show that almost all double-pane replacement windows were installed after the 1978 ban on lead paint (US Department of Energy, Energy Information Administration, 1995), so houses with double-pane windows are highly unlikely to have lead paint on interior window surfaces. Conversely, single-pane window replacement in older homes also effectively targets homes with lead paint on interior window surfaces. Lead paint was used on most original windows in pre-1940 construction, so almost all pre-1940 homes with single-pane windows today are also likely to have lead paint on interior window surfaces. Homes with lead paint on interior window surfaces also appear to account for about 40% of 1940–1959 homes with single-pane windows.

4. Discussion

Both the original and revised model forecasts are well within the 1999–2000 NHANES 95% confidence interval

estimate of 189,000–846,000. Although this is a large confidence interval due to NHANES sample size limitations (*n* = approximately 800 children aged 1–6 yr), the model forecast trend is also consistent with blood lead surveillance data reported to the CDC (Meyer et al., 2003). The model forecast that the total number of EBL children would decline by 27.2% over these years, while the actual number of EBL children reported to CDC from surveillance data declined by 35.5% from 1997 to 2001 (Meyer et al., 2003), which is a reasonably good agreement between the model and the surveillance data.

In addition, AHS data for 2001 suggest that the overall window replacement rate in pre-1975 housing has increased substantially over the 1989–2000 replacement rates used in the original Task Force model (Table 13).

Fig. 2 shows that the model, updated to reflect the 2001 AHS data on children per unit and percentage below 130% of poverty, plus the 20% increase in window replacement rates for 2000–2010 (relative to 1989–2000 rates), now forecasts that market trends for window replacement and demolition alone would reduce the number of EBL children to 292,000 by 2010, not counting other efforts to reduce childhood lead exposure. The new HUD rule will also protect more children living in federally assisted housing, but 250,000 children would still be at risk by the end of the decade unless further action is taken. In addition, there could be other recent housing stock changes that would be expected to affect the 2010 forecast. For example, it is possible that the effect of Hurricane Katrina and other weather-related events will accelerate the rate of demolition and substantial rehabilitation of older housing, at least in some areas of the country. As we near 2010, updating the model with newer empirical estimates will be needed, underscoring the need for continued surveillance of both high-risk children and housing.

The validation of the Task Force model suggests that the additional action needed should include a "lead-safe window replacement" initiative, which would yield multiple benefits of childhood lead poisoning prevention, increased home energy efficiency, reduced air pollution and carbon emission caused by power plant emissions, and improved housing affordability (Nevin and Jacobs, 2006).
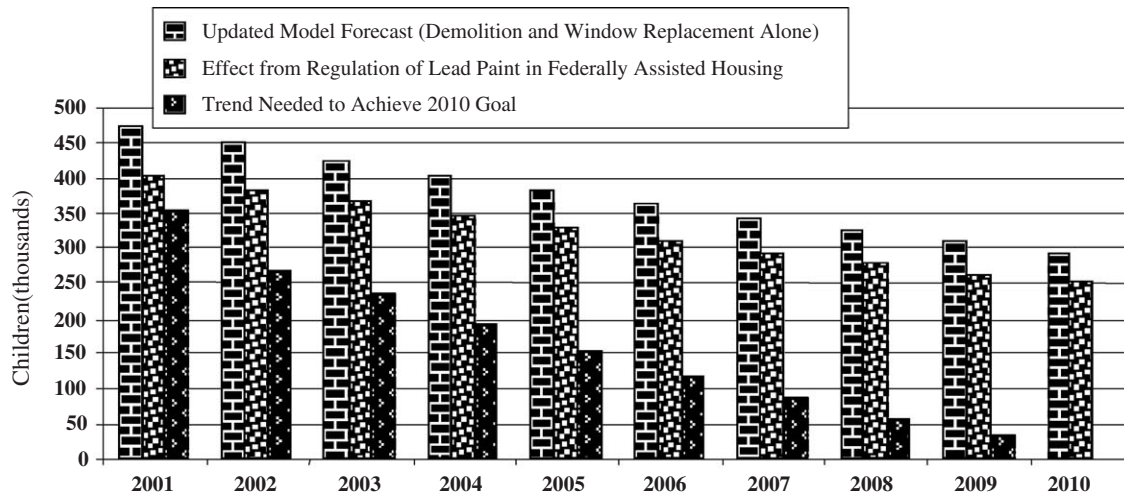
Fig. 2. Number of children with blood lead levels $\geqslant 10\,\mu g/dL$ from 2000 to 2010.

Window replacement, combined with control of other lead-based paint hazards, has been shown to reduce both dust lead and children's blood lead levels (National Center for Healthy Housing and University of Cincinnati Department of Environmental Health, 2004). This relationship should be confirmed with additional research.

The validation of the Task Force model also suggests that a more systematic effort to combine housing and health interventions could further help protect the population from other diseases related in part to housing condition. Although home weatherization is sometimes associated with increased indoor air pollution and mold and moisture problems, occupants of properly weatherized homes report reduced incidence of colds, flu, allergies, headaches, and nausea, while a control group showed no change over the same period (Berry et al., 1997). A large randomized trial of housing insulation treatments in New Zealand showed significant improvements in children's days off school, adult's days off work, self-rated general health, reduced respiratory symptoms, and reduced visits to physician's offices and hospitals (Howden-Chapman et al., 2005). Some of these health benefits may be directly related to energy efficiency improvements that reduce drafts and improve temperature consistency, but weatherization programs also routinely repair combustion equipment and exhaust ventilation systems to reduce carbon monoxide poisoning risks and other health hazards. Leaking air ducts reduce home energy efficiency and also cause moisture problems, which are associated with mold-induced illness and the distribution of indoor air pollution throughout a home.

Substandard housing conditions have been linked to a large number of adverse health outcomes (Breysse et al., 2004; Jacobs, 2005; Krieger and Higgins, 2002; Matte and Jacobs, 2000). For example, dust mites, mold, cockroach, and other allergen-producing organisms in the home environment are triggers for asthma, especially in children. The specialized cleanup required to remove lead dust

hazards, such as using a high efficiency particulate air (HEPA) vacuum cleaner with wet cleaning, is similar to cleanup techniques used to reduce allergens in dust. Such cleaning, together with other coordinated housing and medical interventions, has achieved statistically significant improvements in asthma in a large inner-city cohort of children in seven cities (Morgan et al., 2004). Integrating these hazard reduction protocols could address both lead dust hazards and the most common triggers for asthma simultaneously.

Further research on the relationship between housing condition and health outcome is needed. One important research opportunity is to integrate housing and community data into the planned National Children's Study. For example, combining American Housing Survey data for the specific cities to be included in the study would be essential.

While the energy efficiency benefits of window replacement, duct sealing and other weatherization activities are well established (Nevin and Watson, 1998; Nevin et al., 1999), the related health benefits, especially those associated with chronic disease morbidity and mortality, are only beginning to be fully understood. The experience with lead poisoning, which clearly shows the benefits of housing-based health interventions, can serve as a model in addressing other housing-related health problems. Currently, housing-related health problems are largely ignored in housing markets and are not reflected in housing value and price. This contributes to inefficient cost shifting between housing and health care sectors of the economy, substandard housing and inadequate health care (Jacobs, 2005).

Better data on costs and market value impacts from these upgrades could also inform mortgage underwriters about default risks. If a lead-safe window replacement initiative were expanded to address other healthy home energy efficiency improvements, an evaluation that tracks costs, health benefits, energy savings, and other benefits from bundled home upgrade strategies would be essential

in order to enable the market to properly value health investments in housing.

5. Conclusion

The 1999 model has now been validated with empirical estimates. Trends in housing demolition, window replacement, abatement, regulatory and other initiatives, and demographic patterns all help explain the dramatic reduction in childhood lead poisoning that occurred from 1990 to 2000. Yet without additional action, the nation is unlikely to meet its goal of eliminating childhood blood lead levels above $10 \mu g/dL$, just as it failed to meet the 2000 goal of eliminating childhood blood lead levels above $25 \mu g/dL$. The actions needed are well known, the disease is entirely preventable, and it has persisted for far too long. Furthermore, a focused window replacement policy can yield multiple benefits of lead poisoning prevention, home energy efficiency, reduced air pollution, improved housing affordability, and other benefits. Finally, modeling housing, demographic, and disease data holds great promise in recognizing, forecasting and preventing other housing-related diseases and injuries.

References

Agency for Toxic Substances and Disease Registry, 1988. The Nature and Extent of Lead Poisoning in Children in the United States: A Report to Congress. US Department of Health and Human Services, Atlanta, GA.

Berry, L.G., Brown, M.A., Kinney, L.F., 1997. Progress Report of the National Weatherization Assistance Program. Oak Ridge National Laboratory.

Bornschein, R.L., Succop, P., Kraft, K.M., Clark, S., Peace, B., Hammond, P., 1987. Exterior surface dust lead, interior house dust and childhood lead exposure in an urban environment. In: Hemphill, D.D. (Ed.), Trace Substances in Environmental Health, 20. Proceedings of the University of Missouri's 20th Annual Conference, June 1986, University of Missouri, Columbia, MO.

Breysse, P., Farr, N., Galke, W., Lanphear, B.P., Morley, R.L., Bergofsky, L., 2004. The relationship between housing and health. Environ. Health Perspect. 112, 1583–1588.

Brody, D.J., Brown, M.J., Jones, R.L., Jacobs, D.E., Ashley, P.J., Homa, D., Schwemberger, J.G., Mosby, J.E., Doa, M.J., 2005. Blood lead levels—United States, 1999–2002. Centers for Disease Control and Prevention. Morb. Mort. Wkly. Rep. 54, 513–516.

Canfield, R.L., Henderson, C.R., Cory-Slechta, D., Cox, C., Jusko, T.A., Lanphear, B.P., 2003. Intellectual impairment in children with blood lead levels below $10 \mu g/dL$. New Engl. J. Med. 348, 1517–1522.

Clark, S., Bornschein, R.L., Succop, P., Roda, S., Peace, B., 1991. Urban lead exposures of children in Cincinnati, Ohio. J. Chem. Speciat. Bioavailab. 3 (3/4), 163–171.

Denno, D.W., 1990. Biology and Violence. Cambridge University Press, New York.

Dietrich, K.N., Ris, M.D., Succop, P.A., Berger, O.G., Bornschein, R.L., 2001. Early exposure to lead and juvenile delinquency. Neurotoxicol. Teratol. 23, 511–518.

Duggan, M.J., Inskip, M., 1985. Childhood exposure to lead in surface dust and soil: a community health problem. Public Health Rev. 13, 1–54.

Fisette, P., 2003. Windows: understanding energy efficient performance, building materials and wood technology. Department of Natural Resources Conservation, College of Natural Resources and the Environment, University of Massachusetts, Amherst. Available at: http://www.umass.edu/bmatwt/publications/articles/windows_understanding_energy_efficient_performance.html

Guthrie, A., McLaine, P., 1999. Another link in the chain. Alliance for Healthy Housing and National Center for Healthy Housing. Available at: http://www.centerforhealthyhousing.org/html/state_ph.html

Howden-Chapman, P., Matheson, A., Crane, J., Viggers, H., Cunningham, M., Blakely, T., O'Dea, D., Cunningham, C., Woodward, A., Saville-Smith, K., Baker, M., Waipara, N., Kennedy, M., Davies, G., 2005. Retrofitting houses with insulation to reduce health inequalities: a community-based randomized trial. In: Proceedings of the Second WHO International Housing and Health Symposium, September 29–October 1, 2004. World Health Organization European Centre for Environment and Health (Bonn Office), November 2005, pp. 51–59. http://www.euro.who.int/Housing/Activities/20060113_1

Jacobs, D.E., 1995. Lead paint as a major source of childhood lead poisoning: a review of the evidence. In: Michael E. B., Allen Iske, S.D. (Eds.), Lead in Paint, Soil and Dust: Health Risks, Exposure Studies, Control Measures and Quality Assurance. ASTM STP 1226, American Society for Testing and Materials, Philadelphia.

Jacobs, D.E., 2005. Housing and health: challenges and opportunities. Proceedings of the Second WHO International Housing and Health Symposium. September 29–October 1, 2004, Vilnius, Lithuania. World Health Organization European Centre for Environment and Health, Bonn Office, November 2005, pp. 35–50. http://www.euro.who.int/Housing/Activities/20060113_1

Jacobs, D.E., Clickner, R., Zhou, J., Viet, S., Marker, D.A., Rogers, J.W., Broene, P., Zeldin, D.C., 2002. The prevalence of lead-based paint hazards in US housing. Environ. Health Perspect. 110, A599–A606.

Krieger, J., Higgins, D.L., 2002. Housing and health: time again for public health action. Am. J. Public Health 92 (5), 758–768.

Lanphear, B.P., Emond, M., Jacobs, D.E., Weitzman, M., Tanner, M., Yakir, B., Eberly, S., 1995. A side-by-side comparison of dust collection methods for sampling lead-contaminated house dust. Environ. Res. 68, 114–123.

Lanphear, B.P., Matte, T.D., Rogers, J., Clickner, R., Dietz, B., Bornschein, R.L., Succop, P., Mahaffey, K.R., Dixon, S., Galke, W., Rabinowitz, M., Farfel, M., Rhode, C., Schwartz, J., Ashley, P., Jacobs, D.E., 1998. The contribution of lead-contaminated house dust and residential soil to children's blood lead levels: a pooled analysis of 12 epidemiological studies. Environ. Res. 79, 51–68.

Matte, T.D., Jacobs, D.E., 2000. Housing and health—current issues and implications for research and programs. J. Urban Health 77 (1), 7–25.

Meyer, P., Pivetz, T., Dignam, T., et al., 2003. Surveillance for elevated blood lead levels among children 1997–2001. Morb. Mort. Wkly. Rep. 52, SS-10.

Morgan, W.J., Crain, E.F., Gruchalla, R.S., O'Connor, G.T., Kattan, M., Evans, R., Stout, J., Malindzak, G., Smartt, E., Plaut, M., Walter, M., Vaughn, B., Herman, M., 2004. Results of a home-based environmental intervention among urban children with asthma. New Engl. J. Med. 351 (11), 1068–1080.

National Academy of Sciences, 1993. Measuring Lead Exposure in Infants, Children, and Other Sensitive Populations, Board on Environmental Studies and Toxicology. Commission on Life Sciences, National Academy Press, Washington, DC.

National Center for Healthy Housing and University of Cincinnati Department of Environmental Health, 2004. National Center for Healthy Housing, Evaluation of the HUD Lead Hazard Control Grant Program. Final Report, May 1, 2004, Available at: http://www.centerforhealthyhousing.org/HUD_National_Evaluation_Final_Report.pdf

Needleman, H.L., Riess, J.A., Tobin, M.J., Biesecker, G.E., Greenhouse, J.B., 1996. Bone lead levels and delinquent behavior. J. Am. Med. Assoc. 257, 363–369.

Nevin, R., 2000. How lead exposure relates to temporal changes in IQ, violent crime, and unwed pregnancy. Environ. Res. Sect. A 83, 1–22.

Nevin, R., Jacobs, D.E., 2006. Windows of opportunity: lead poisoning prevention, housing affordability and energy conservation. Housing Policy Debate (accepted).

Nevin, R., Watson, G., 1998. Evidence of rational market valuations for home energy efficiency. Appraisal J., October.

Nevin, R., Gazan, H., Bender, C., 1999. More evidence of rational market values for home energy efficiency. Appraisal J., October.

Pirkle, J.L., Brody, D.J., Gunter, E.W., Kramer, R.A., Paschal, D.C., Flegal, K.M., Matte, T.D., 1994. The decline in blood lead levels in the United States. J. Am. Med. Assoc 272, 284–291.

President's Task Force on Environmental Health Risks and Safety Risks to Children, 2000. Eliminating childhood lead poisoning: a federal strategy targeting lead paint hazards (report and appendix). US Department of Housing and Urban Development and US Environmental Protection Agency, Washington, DC, February. Available at: http://www.hud.gov/offices/lead/reports/fedstrategy.cfm, accessed 12 July 2005.

US Bureau of the Census and US Department of Housing and Urban Development, 2000. American Housing Survey (AHS), ICF Consulting Analysis of biannual AHS data for 1989–2001. American Housing Survey, 1997. Available at: http://www.huduser.org/intercept.asp?loc=http://www.census.gov/prod/99pubs/h150-97.pdf, accessed 1 September 2004.

US Centers for Disease Control and Prevention, 1991. Preventing lead poisoning in young children: a statement by the centers for disease control. Report 99-2230, US Department of Health and Human Services, Atlanta GA.

US Centers for Disease Control and Prevention, 1997. Blood lead levels—United States, 1991–1994. Morb. Mort. Wkly. Rep. 46(7), February 21.

US Consumer Product Safety Commission, 1977. Ban of lead-containing paint and certain consumer products bearing lead-containing paint. 16 CFR 1303. Fed. Reg. 42, 44199.

US Department of Energy, Energy Information Administration, 1995. 1993 Housing Characteristics from the 1993 Residential Energy Consumption Survey (RECS), June.

US Department of Housing and Urban Development, 1990. Comprehensive and workable plan for the abatement of lead-based paint in privately owned housing. HUD-PDR 1295, Report to Congress, HUD Office of Policy Development and Research, Washington, DC. Available at: http://www.hud.gov/utilities/intercept.cfm?/offices/lead/reports/plan1990.pdf, accessed 1 September 2004.

US Department of Housing and Urban Development, 1995. Guidelines for the Evaluation and Control of Lead-Based Paint Hazards in Housing. HUD 1539-LBP, Washington, DC. Available at: http://www.hud.gov/offices/lead/guidelines/hudguidelines/index.cfm, accessed 22 April. 2005.

US Department of Housing and Urban Development, Office of Lead Hazard Control, 1999a. Economic Assessment of the Final Rule on Lead-Based Paint: Requirements for Notification. Evaluation and Reduction of Lead-Based Paint Hazards in Federally Owned Residential Property and Housing Receiving Federal Assistance, Washington, DC, September 7. Available at: http://www.hud.gov/utilities/intercept.cfm?/offices/lead/leadsaferule/completeRIA1012.pdf, accessed 1 September 2004.

US Department of Housing and Urban Development, 1999b. 24 CFR Part 35: Requirements for Notification. Evaluation and Reduction of Lead-Based Paint Hazards in Federally Owned Residential Property and Housing Receiving Federal Assistance, September 15, 1999. Available at: http://www.hud.gov/offices/lead/leadsaferule/index.cfm, accessed 1 September 2004.

US Environmental Protection Agency, 2001. 40 CFR Part 745, Lead; Identification of Dangerous Levels of Lead. Final Rule, Available at: http://www.epa.gov/lead/leadhaz.htm, accessed 1 September 2004.

Wilson, J., Pivetz, T., Ashley, P., Jacobs, D.E., Strauss, W., Menkedick, J., Dixon, S., Tsai, H.C., Brown, V. Evaluation of HUD-funded lead hazard control treatments at six years post-intervention, Environ. Res. (accepted for publication).