Joint Center for Housing Studies

Harvard University

**Residential Building Codes, Affordability, and Health Protection:
A Risk-Tradeoff Approach**

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Abstract

Residential building codes intended to promote health and safety may produce unintended countervailing risks by adding to the cost of construction. Higher construction costs increase the price of new homes and may increase health and safety risks through "income" and "stock" effects. The income effect arises because households that purchase a new home have less income remaining for spending on other goods that contribute to health and safety. The stock effect arises because suppression of new-home construction leads to slower replacement of less safe housing units. These countervailing risks are not presently considered in code debates. We demonstrate the feasibility of estimating the approximate magnitude of countervailing risks by combining the income effect with three relatively well understood and significant home-health risks. We estimate that a code change that increases the nationwide cost of constructing and maintaining homes by $150 (0.1% of the average cost to build a single-family home) would induce offsetting risks yielding between 2 and 60 premature fatalities or, including morbidity effects, between 20 and 800 lost quality-adjusted life years (both discounted at 3%) each year the code provision remains in effect. To provide a net health benefit, the code change would need to reduce risk by at least this amount. Future research should refine these estimates, incorporate quantitative uncertainty analysis, and apply a full risk-tradeoff approach to real-world case studies of proposed code changes.

KEYWORDS: risk-tradeoff analysis, building codes, housing, health effects, QALY

Residential Building Codes, Affordability, and Health Protection: A Risk-Tradeoff Approach

by

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1. Introduction

The primary rationale for residential building codes is to protect health and safety. Nevertheless, code provisions are seldom evaluated using formal risk or benefit-cost analysis.[1] While code officials may weigh the health and safety benefits of code changes against the cost to builders to implement them, these officials lack a quantitative basis for evaluating the unintended health and safety risks that may result from making new homes less affordable. We postulate that code-induced cost increases in the long-run marginal cost of purchasing new homes exacerbate health and safety risks through two effects. One is an "income effect" that draws household income away from other health-protective investments to pay for code-related costs. The other is a "stock effect" that causes some potential buyers to delay the purchase of a new home, thereby leaving more households exposed to a larger stock of older (and riskier) dwellings.

Because code officials lack the tools to estimate income and stock effects, they cannot compare health costs to health benefits to determine whether imposition of a new code standard will generate a net health and safety benefit.[2] This paper begins the process of addressing this problem by demonstrating that it is feasible to estimate the magnitude of countervailing risks induced by adding to the costs of homebuilding. By comparing risk reductions with induced increases, code officials could give systematic weight to "affordability" concerns about new codes and quantitatively assess whether codes may inadvertently increase health risks to general or high-risk populations.

The income and stock effects we postulate are supported by a rapidly expanding

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[1] Exceptions include analyses of code provisions to reduce hurricane damage in South Florida [(24)] and of the cost-effectiveness of laws requiring residential smoke detectors [(47)].

[2] Risk analysts have long been concerned about the unintended risks that may arise from well-intentioned efforts to reduce or eliminate a hazard [(102)]. This concern may reflect the view that risk managers should behave like physicians, who are expected to consider the side effects as well as the efficacy of treatment alternatives on behalf of patients [(10,35,113)].

literature on the risk-tradeoff approach and the unintended consequences of imposing regulations.[3] This literature has shown that the economic costs of regulation may increase risk by reducing household income (51,114). The important role of income in determining family health is buttressed by a growing body of epidemiological research (13,19-21,44,98,115) and has been used by risk analysts to produce rough estimates of the statistical mortality impacts of income losses (13,14,35,49-51,111).

The risk-tradeoff literature has found that the focus of regulators on reducing risks of new products and new industrial facilities has not always been matched by a concern for the risks associated with existing products and facilities. This "new-old" distinction in risk regulation can have perverse effects. It creates economic incentives for producers and consumers to escape the costs of regulation by delaying the purchase of new products and facilities and extending the life of existing units, potentially increasing net social risk (45). Delaying the shift from old to new has resulted in tangible regulation-induced risks in a number of industries. In the pharmaceutical industry, the strict regulation of new drugs has delayed introduction of some promising therapies and curtailed research and development into new pharmaceuticals (81). In the automotive industry, as new passenger vehicles have become more expensive due to emissions, safety, and fuel-economy rules, the rate at which new vehicles replace old has fallen, slowing the penetration of the improved vehicles into the existing fleet (17,38). Regulation of new coal-fired power plants to reduce atmospheric sulfur emissions induced utilities to lengthen the life of existing coal-fired facilities which are subjected to much less stringent sulfur emission rules (2,16). In each of these industries, risk-tradeoff tools have been employed to identify and quantify the offsetting or perverse effects of regulations that lengthen the life of existing (and more risky) products and facilities, allowing a more valid comparison to regulatory benefits.

We seek to demonstrate that similar concepts and tools can be developed to assist code

officials faced with the task of setting health and safety standards for residential construction. The risk-tradeoff approach we propose represents a minimum threshold: we assume that code

[3] Early risk-tradeoff research grew out of concern that the safety of nuclear power was not appreciated adequately by opinion leaders and the public. Estimates were generated of the relative fatality risks (per unit of energy) of generating electricity from nuclear power and fossil fuels (43,46,116).

officials would not support a proposed code change unless a reasonable case is made that any unintended risks induced by the revision are less compelling than the anticipated risk reductions.[4] Once this case is made, code officials can analyze other effects of the proposed change to determine whether it should be adopted.

Estimating the countervailing risks created by adding to building costs is an important first step toward implementing a risk-tradeoff approach.[5] Implementing a full risk-tradeoff approach would require comparing the health and safety risk-reduction benefits of a code provision to both its indirect and direct countervailing risk-exacerbation costs. Direct countervailing risks depend on the specific code change. For example, energy-efficiency standards can potentially worsen indoor air quality if ventilation is excessively restricted or channeled to expose the return air to unexpected sources of pollution, and improved stairway lighting can increase electricity consumption and with it ambient air pollution from generating stations. We do not consider these direct risks but focus our calculations on the indirect risks created by adding to construction or operating costs. These risks do not depend on the specific character of the code change and so estimates can be generalized to a wide range of cases.

The remainder of the paper is organized as follows. Section 2 presents our conceptual framework and describes the two main pathways by which code-related costs may induce health risks: the "income" and "stock" effects. Section 3 provides a numerical example, in which we illustrate the income and stock effects associated with a hypothetical code-induced increase in housing costs. Conclusions and limitations are summarized in Section 4.

2. Conceptual Framework

As a starting point for analysis, we consider a proposed new code provision that, if

[4] This test applies to code provisions motivated by improving health and safety. It may of course be appropriate to adopt provisions that increase risks to human health and safety in the interests of promoting other goals (reduced environmental impact might be an example).

[5] The risk-tradeoff approach is a partial alternative to benefit-cost analysis for public health. Because it compares only the beneficial and adverse effects on health, the approach does not require information on tradeoffs between health and other goods that money can buy.

adopted, would increase construction costs and the effective price of new homes to consumers.[6] By “effective” price, we mean the net financial impact on a consumer, taking into account the present-value cost of maintaining a home as well as increments in purchase price. If a proposed code provision (e.g., an energy conservation measure) generates future savings that, in present value, exceed the initial increment in construction cost, then the effective price of housing declines. We assume that purchasing decisions are a function of this effective price, although we recognize that consumers may be more sensitive to initial costs than to future cost savings. We restrict attention to code provisions that require builders to incorporate features the market would not otherwise require. If the provision would be incorporated anyway, the provision simply ratifies market forces. For ease of analysis, we also assume that the code provision would apply uniformly across the U.S. housing market.

We assume that a code-induced increase in construction costs would increase the price of new homes by shifting the supply curve upward.[7] In making this assumption, we follow many others who reason that home builders are in a highly competitive industry that has narrowed profit margins so much that no producer could afford to absorb any portion of the code change.[8] The ultimate price increase to consumers is smaller than the increase in construction costs as builders will reduce costs by using less expensive materials or furnishings and buyers will demand fewer housing amenities. Such offsetting actions are addressed in the econometric model described below. Although our macroeconomic model, like others, cannot identify the specific changes made to offset the increased costs, it can describe the magnitude of the effect.

In the aggregate, we expect consumers to exhibit myriad responses to a code-induced price increase, depending on individual households’ tastes and incomes. Our analytical

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[6] Throughout the analysis, we consider only the costs of constructing and maintaining the structure of a home. Costs of the land are additional and any changes in land values are neglected.

[7] More stringent code provisions could also increase the cost of renovating older homes. If, as is often the case, renovations are required to come into compliance with current codes, the cost of incorporating a newly required feature into a renovation may easily exceed the cost of incorporating it in new construction. In this case, the code provision would shift demand from renovations to new construction.

[8] In terms of microeconomic theory, we assume that the residential construction sector is in long-run competitive equilibrium. Several noteworthy housing studies have adopted this assumption [(85,89)], and other studies provide empirical support for it [(104)]. An interesting study suggests that builders might pass on the full cost of the code change even absent perfect competition [(97)].

framework distinguishes two categories of consumers. First are those home buyers who would reduce other spending to pay for the price increase. The additional money can either come from housing quality, as households may still purchase a new home but with a reduction in other features (e.g., size, location, quality of materials), or from other household budgets. Members of this group would suffer an "income effect" from the code change; the income left over after paying for housing would be reduced because of the cost of the code change.

While the extra cost of housing would not come solely from consumers' health budgets, we would expect the income effect to reduce the overall health of this group. Expenditures on a variety of both health-promoting and health-damaging goods rise with income, but the net effect is health-promoting as shown by the literature indicating that "Richer is Safer" [114]. Thus, a price change that left households with fewer resources to spend on non-housing goods and services would reduce the health status of those households.

The second group of consumers, unlike the first group, would respond to the price change by delaying or abandoning their plans to purchase a new home. This response would create a "stock effect:" the higher prices of new homes at the "top" of the market would filter through the existing stock by suppressing "up market" movements of households [64], causing households at the "bottom" of the market to remain in units that otherwise would have been vacated and scrapped. As described below, we model this effect by assuming that a price increase would permanently decrease the rate at which units are retired from the housing stock. Although an accelerated retirement rate might be avoided in the short run by reduced vacancy rates or an increase in residents per dwelling (crowding), we assume long-run equilibrium conditions in which price changes influence investment and maintenance rates and therefore the retirement rate. The result would be that more households would live in older and lower quality units for longer periods of time, thereby increasing their exposure to certain risk factors and changing net population health risk.

We assume that consumers do not value (or perhaps even recognize) the difference in houses because of the code change. If consumers value the feature mandated by code at least as much as it costs builders to provide, one would expect builders to incorporate this feature and the code change would only ratify market conditions. Moreover, if consumers value the change because it provides health and safety benefits, these will be included in the benefits estimate, to which the cost-induced risks we estimate are compared. Even if consumers value the feature required by the code change more than its cost, by purchasing it they necessarily reduce income available for spending on other goods, some part of which would have improved health and safety, on average.[9]

2.1. *Pathways for Cost-Induced Health Risks*

In this section, we discuss the two pathways by which code-induced increases in the costs of new home construction may increase health risk. One pathway, the "income effect," is due to marginal wealth (or investment income) reductions after new home purchases. The second pathway, the "stock effect," is related to increased occupancy of the lowest quality units that are known to be associated with greater health risks than new homes.

2.1.1. *Income and Health*

Epidemiologists and social scientists have documented an inverse relationship between income and measures of health status, including mortality risk. The association has been documented at various levels of aggregation: the individual, the household, the city or SMSA, the state, and even the country. The empirical function relating income to health risk is often curvilinear, suggesting that marginal increases in income are associated with progressively smaller reductions in health risk. Thus, the magnitude of the effect of income losses on health will depend upon who experiences the losses, with the effects being relatively greater if the income losses are experienced by lower-income families [(14,49-51)].

A statistical association between household income and health status of household members does not necessarily prove that a causal relationship exists [(95)]. Yet when a variety of potential confounders are controlled (e.g., education, social class, age, race, and gender), the inverse association between income and health status persists [(44)]. Part of the association may be explained by reverse causation--the fact that sicker people earn less income--but a variety of studies have concluded, using various empirical arguments, that reverse causation cannot account for most of the observed income-health association [(13,14,19-21)]. The degree to which

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[9] As noted by an anonymous reviewer, the code change may also provide external benefits. If there are scale economies in producing the feature, requiring all new construction to incorporate it will decrease the supply cost. Future home buyers may benefit from an information externality, since they can assume that the feature is incorporated in houses built after the effective date of the code change.

income losses are permanent (rather than transitory) may also influence the magnitude of adverse health impacts [35].

Estimates of the causal impact of income losses on mortality risk have been obtained by direct and indirect methods. The direct approaches include regression models that describe mortality risk as a function of income and other variables. Keeney uses cross-sectional and longitudinal data to estimate the relationship between income and mortality risk and interprets this function as a reduced-form statement of the causal relationship from income to mortality risk. He estimates the aggregate income loss that induces one statistical fatality as $6.5-7.2 million using cross-sectional data [49] and $11.4 million using longitudinal data [51] (assuming in both cases that costs are allocated to households in proportion to income). Chapman and Hariharan analyze panel data on older men and attempt to control for possible reverse causation by including measures of pre-existing health and adjusting for population heterogeneity. They estimate values of $12.2 million [13] and $6.4-14.0 million [14] (again assuming cost allocation proportional to income). A strength of the direct approach is that it empirically accounts for all linkages between income and health, adverse as well as beneficial. A limitation is that confounding by education and social class is difficult to eliminate.

Viscusi [111] shows that the rate at which income loss causes premature fatality is theoretically equal to the value of mortality risk reduction ("Value of Statistical Life" or VSL) divided by the marginal propensity to spend on risk reduction. The latter term is difficult to measure as mortality risk reduction is bundled with other goods (e.g., health care, food). Drawing primarily from estimates of marginal propensity to spend on health care, Viscusi suggests a range for the propensity to spend on health of about 0.3 to 0.05. Combining these values with a VSL on the order of $5 million [110] suggests a value of $15-100 million. Because it relies on estimates of VSL, the indirect approach yields values that are consistent with VSL estimates but also incorporates any biases in these estimates. In addition, the indirect approach ignores income-health linkages that are not included in the estimate of the propensity to spend on health care (e.g., larger incomes may result in safer car purchases) as well as any hazardous consumption induced by income (e.g., more income may induce more travel).

2.1.2. *Housing Characteristics and Health*

Many aspects of a home can affect risks to the health of occupants, based upon the sheer magnitude of time spent in and around the home. These factors can be categorized as related to the home's structure, size, and location. Precise quantification of the housing-health relationship is difficult, because housing characteristics are likely to have smaller effects on health than other potentially correlated factors such as smoking, diet, exercise, genetics, and access to health care.

Physical characteristics of the dwelling can influence risks of home accidents (e.g., fires and falls), exposure to contaminants (e.g., radon and lead), and protection against natural disasters (e.g., hurricanes and earthquakes). Other health risks, such as communicable-disease transmission and stress related to crowding, are influenced by the size of the structure relative to the number of occupants. Location of the dwelling may introduce risks associated with neighborhood and geographic conditions (e.g., violence, air quality, arson, and traffic crashes). Some (but not all) of these risks are associated with age of the house, which is the most common surrogate for quality of housing that is available in the literature. On some dimensions, new homes may present greater risks than older homes. For example, formaldehyde in particle board and other construction materials diffuses into the environment over several years, and so exposure is likely to be lower in an old than a new home. Overall, however, older and lower-quality units are likely to present greater risks to occupants.

Although occupants of older and lower-quality homes are more likely to have health problems, this association is not necessarily causal. Without controlling for potential confounders, this association may simply be a restatement of the association between socio-economic status and health. Moreover, health effects related to individual behaviors (e.g., smoking and alcohol consumption) may have some correlation with housing quality, but without any causal link to the structure itself. Moving a household from an old house to a new house is unlikely to change most health-related behaviors, except under extreme shifts in community norms.

Residential building codes have been continuously revised in recent decades. If prior revisions were effective in reducing health and safety risks to occupants, then lengthening the time spent in homes built to older codes will result in more health and safety risks to occupants, particularly if the units have degraded. However, we have not been able to identify any significant literature that quantifies the magnitude of the safety and health benefits of modern building codes.

Faced with severe data limitations, our approach is to quantify several specific health risks that are clearly associated with living in homes that are most likely to be retired from the market. Although these particular risks are not exhaustive, they can provide an indication of the rough magnitude of the effects that might be identified in a more comprehensive study of the causal relationship between housing quality and human health.

3. Numerical Application

For illustration, we assume that a proposed building code change increases the effective price of a new home by $150 (approximately 0.1%) under long-run competitive equilibrium conditions.[10] Given this assumption, we evaluate the health implications for the lifetimes of the population affected in a typical year following the code change (comparing equilibrium conditions and neglecting transient effects).[11] For the income effect, we assess the cohort of home buyers who incur code-related costs in this single year. For the stock effect, we assess the decline in newly constructed units in a single year and the consequent shift in the age composition of units in the stock over time. We also present the aggregate health implications for longer time periods, as this may be a more realistic representation of the total impacts of the code change under long-run equilibrium conditions. For all calculations, we assume that any transfers among intermediate homes yield no net change in risk and we neglect changes in risks that result from income transfers among parties with disparate incomes (e.g., changes in rental prices).

For both income and stock effects, human health outcomes are evaluated in quality-

[10] For changes in effective price that are small compared with the price of home ownership, our estimated effects are linear so the effects of larger or smaller changes can be readily calculated. The average price of a new housing unit, including multifamily and manufactured housing, is smaller than the $150,000 average price of a new single-family home.

[11] Long-run competitive equilibrium implies that net housing demand is not substantially depressed from baseline, and that changes in housing starts are largely related to the decline in unit retirement. Our analysis compares two equilibrium conditions, with and without the code change, for a one year cohort of potential new home buyers. Calculations can be extrapolated readily to the effective lifetime of a code, given estimates of the duration of code efficacy and proper discounting of effects. Transient effects of code changes are neglected in the analysis, as modeling these would require consideration of buyers' expectations about future code changes and home builders' responses.

adjusted life years (QALYs). The QALY is a standard metric that is widely used in evaluation of medical and public-health interventions [(33)]. Unlike statistical lives saved and other alternatives, it combines effects on both mortality and morbidity. Each life year is rated on a scale from 0 (death) to 1 (perfect health), with years assigned intermediate values based on subjective assessments of well-being.[12] No standard set of health quality weights exists, so we use weights from a variety of sources as appropriate for each health effect we consider. For healthy life years, we follow standard practice in assigning weights less than one to older ages to reflect the greater prevalence of chronic health impairments in those age groups [(25)]. Also following standard cost-effectiveness analysis techniques [(33)], we discount future year QALYs (at 3% per year) and report the present value of the entire stream of lifetime QALY decrements (denoted "$QALY_{PV}$").[13]

3.1. *Market Characteristics and Implications*

To estimate the effects of an effective-price increase on demand for new housing, we rely on a macroeconomic model of the residential housing stock [(86)]. The model uses aggregate U.S. data to estimate the change that such a price increase would prompt, both in demand for homes and in retirement rates (which is not conventionally estimated in the literature). The effects on new-home demand and retirement rate determine the allocation of potential home buyers between those who buy a new home despite the price increase (and suffer the income effect) and those who delay purchasing a new home (and thereby increase the number of households remaining in the existing stock).

The economic model provides an internally consistent representation of the effects of the code change on housing cost, quality, and age distribution, and yields estimates that are

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[12] The QALY can be interpreted as a von Neumann-Morgenstern utility function for health and longevity if certain plausible assumptions are satisfied (risk neutrality on length of life and a willingness to trade the same fraction of longevity to alleviate a specified health impairment, regardless of expected longevity) [(9,83)]. Although the available evidence suggests these assumptions are not always satisfied, they serve as reasonable modeling assumptions [(8,60,67,70,100)].

[13] As an example, the average 45-year old in the United States has a life expectancy of 43 years. A multiattribute utility analysis of perceived health status and activity limitation from the National Health Interview Study [(25)] estimated that the average health-related quality of life (QoL) ranges from 0.88 at age 45 to 0.51 at age 85. Weighting life years by these QoL weights yields an expected value of approximately 30 QALYs, or 16 $QALY_{PV}$ if future years are discounted at 3%. The average $QALY_{PV}$ remaining over the U.S. population is about 18.1.

broadly consistent with other available information. However, the model outputs should be interpreted cautiously, as the model was newly developed for this work and the specification has not been exhaustively tested.

The model describes demand for housing, differentiating between the number of units (KU_t) and the average value per unit (VPU_t) in year t. The total value of the housing stock in year t (KV_t) is, by definition, the product of these terms. New and existing homes are close substitutes, and so their prices are determined by the cost of new construction and other factors. The effective price or opportunity cost of home ownership can be characterized as a rental value ($RENT_t$), because an individual may be viewed as buying a home and renting it to herself. The opportunity cost of housing depends on the construction cost of new homes, tax and interest rates, and other factors, and is modeled as:

$$RENT_t = (C_t / P_t) \{(\rho_t + \mu_t)(1 - \tau_t) - \pi_t + \gamma\} \qquad (1)$$

where C_t is the nominal construction cost per quality-adjusted unit of owner-occupied housing, P_t is the general price level, τ_t is the marginal tax rate on homeowners, ρ_t is the nominal mortgage interest rate, μ_t is the property tax rate, π_t is the rate of housing price inflation, and γ is the fixed rate at which the value per unit of housing depreciates.[14]

The annual change in the stock of housing units is equal to the number of new units completed (HC_t) less the fraction of the existing stock (KU_{t-1}) that is demolished or abandoned:

$$\Delta KU_t = HC_t - \delta_t KU_{t-1} \qquad (2)$$

where the net retirement rate δ_t incorporates demolition and abandonment as well as rehabilitation of previously abandoned units and conversion of buildings from non-residential to residential use. The number of units completed is related to the number started in the current and preceding year by

$$HC_t = 0.8\, HS_t + 0.2\, HS_{t-1} \qquad (3)$$

(the coefficients are based on monthly Census Bureau data).

The net retirement rate responds to economic factors. In particular, the rate at which households trade up from existing units in favor of new ones is likely to be faster in periods

[14] The leverage ratio does not enter equation (1) because we assume the opportunity cost of the equity-financed portion of the housing stock equals the after-tax mortgage rate.

when households enjoy the prospect of secure income and employment and housing costs are low; in less favorable circumstances, households retain and/or renovate existing units. We estimate this effect by modeling the retirement rate δ_t as a function of unemployment and the effective cost of housing, obtaining (t-statistics in parenthesis):

$$\delta_t = 0.02901 - 0.00748 \ln(U_t/U^*_t) - 0.01083 \ln(RENT_t) - 0.00948\, D79_t \qquad (4)$$

$$(2.1938) \quad (-2.0430) \qquad\qquad (-1.9945) \qquad\qquad (-2.3405)$$

Sample: 1968-1996 $\quad R^2 = 0.409 \quad$ Std Err = 0.0039 $\quad$ Durbin-Watson = 2.12

where U_t is the civilian unemployment rate, U^*_t is the Non-Accelerating Inflation Rate of Unemployment (NAIRU), and $D79_t$ is a dummy variable equal to one for 1979 and later years to control for a redefinition of housing stock that occurred then. The retirement rate was constructed by subtracting net changes in the housing stock from the flow of gross additions and dividing by the stock at the beginning of the year, using Census Bureau data. It is sensitive to fluctuations in these variables and averages 0.175% over the sample period (1968-1996). The coefficient on RENT (which incorporates the mortgage interest rate) suggests that a 1% increase in ownership costs decreases the retirement rate by 0.01%.

Housing starts are jointly determined by the need to replace retired units and to accommodate demand for growth in the stock. The demand for housing depends on the number of households that would be expected to form, income per household, and the effective price of housing. The household formation variable ($HHADJ_t$) is constructed by adjusting for changes in the age distribution of the population but holding constant the age-specific probability of forming a household. Changes in household formation in response to economic factors (e.g., children remaining longer in their parents' homes) are reflected in the estimated coefficients on income per household (Y_t) and $RENT_t$. Incorporating predicted values of the retirement rate from equation (4) and allowing for lags in the response of housing starts to changes in prices and income, we estimate the following equation (t-statistics in parentheses):

$$HS_t/KU_{t-1} - \delta_t = 1.05094\ \Delta\ln(HHADJ)_t + 0.09268\ \Delta\ln(Y)_t + 0.05129\ \Delta\ln(Y)_{t-1} \quad (5)$$

$$(6.3786) \qquad (3.1813) \qquad (1.7734)$$

$$- 0.02102\ \Delta\ln(RENT)_t - 0.01274\ \Delta\ln(RENT)_{t-1} + 0.76127\ u_{t-1}$$

$$(-2.5403) \qquad (-1.5456) \qquad (5.3070)$$

Sample: 1970-1996 $R^2 = 0.8232$ Std Err = 0.0030 Durbin-Watson = 1.86

The coefficient on the log of the number of households (adjusted for changes in population) is not significantly different from one, which is consistent with the expectation that the number of housing units grows in proportion to households. Current and lagged income and price effects are statistically significant. The estimated long-run income elasticity is about 0.144 (= 0.09268 + 0.05129) and the long-run price elasticity is about -0.034 (= -0.02102 - 0.01274). The estimated price elasticity implies that a 10% increase in effective price (roughly a one percentage point increase in the after-tax mortgage rate) reduces starts by 250,000 in the first year, which is reasonable to a bit high in historical context.

Finally, the average value per unit is modeled as a function of income per household and effective price. The value encompasses the quantity and quality of fixtures as well as the number of rooms and square footage of a house; if construction costs rise, households purchasing new homes may respond by seeking units in less expensive locations and/or units that are slightly smaller or contain less expensive materials and fixtures. Similarly, although owners of existing units cannot instantaneously adjust the value of their units, they may reduce it over time by deferring maintenance or planned upgrades. Accordingly, we model the response including a factor representing the speed of adjustment between the desired and current average value, obtaining (t-statistics in parentheses):

$$\Delta\ln(VPU)_t = 0.7023 + 0.1552\ \ln(Y)_t - 0.02072\ \ln(RENT)_t - 0.1733\ \ln(VPU)_{t-1} \quad (6)$$

$$(4.8696) \quad (4.9795) \qquad (-4.2106) \qquad (-4.8958)$$

Sample: 1967-1996 $R^2 = 0.573$ Std Err = 0.0042 Durbin-Watson = 2.13

The implied long-run elasticity of value per unit with respect to income per household is 0.896 (= 0.1552/0.1733). Combined with the income elasticity for housing units estimated in equation (5), the overall income elasticity for the value of the housing stock is 1.04 (= 0.896 + 0.144), a value that is near the high end of previous estimates.[15]

[15] Ermisch et al.[(26)] report a consensus that the income elasticity of demand for housing in Britain is "in

The long-run price elasticity of the value per unit is -0.120 (= -0.02072/0.1733). Thus, a 1% increase in effective cost ultimately reduces the average value per unit by about 0.12%. This is substantially larger than the elasticity of units (-0.034) estimated in equation (5), which implies the effect on average quality is 3-4 times larger than the effect on number of units, related both to a shift in the age distribution of the stock and reduced investment in existing units. The overall price elasticity of the real value of the housing stock is the sum of these values, -0.154. This value is somewhat smaller than most estimates in the literature, which suggests our method may underestimate the effects of a cost increase.[16] The speed of adjustment, which represents the rate at which the gap between the actual and desired average value is closed (not the rate at which the average value changes), is 17% per year.

The U.S. housing stock consists of about 115 million homes and construction costs average about $150,000. Our model implies that a $150 (0.1%) increase in construction costs will permanently reduce the retirement rate by 0.0011%, about 1,350 homes per year. The decline in retirement implies a corresponding reduction in housing construction and a consequent increase in the average age of the housing stock. Thus, the long-run equilibrium scenario implies that 1,350 households would forgo new home purchases in a single year and an additional 1,350 households would remain in units that would have otherwise been retired that year. The matching of households and units is determined by market "filtering." The incremental households remaining in the additional retained units are not necessarily the same households that would have purchased new homes.

The cost increase also yields a transient reduction in the demand for new units of 0.0034%, or about 4,000 homes.[17] We assume that this net unit reduction, which can be linked to a reduction in vacancy rates, household growth, or second home purchases, would have no significant effects on health. In addition, the average value per unit falls 0.012%, for an overall price elasticity effect of about $18. This implies that new home buyers would respond

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the region of 0.8 to 1.0" (p. 66). A previous survey of the U.S., Mayo [(65)], reports values between 0.36 and 0.87.

[16] Rapaport [(88)] estimates a comparable price elasticity, -0.12, but Ermisch et al.[(26)] report estimates of -0.40 to -0.80, and Mayo[(65)] reports price elasticities of -0.67 to -0.76.

[17] The transient effect is spread over several years, but most of the effect is realized within two years. The 4,000 unit reduction in construction does not reduce the stock of houses but simply reduces the rate of growth, which is driven by population and income growth.

to the $150 cost increase by compromising on location, size, quality of fixtures, or other attributes, ultimately spending $132 (= $150 - $18) more than they otherwise would have.

We next estimate the characteristics of the incremental retained units. To do so, we construct a profile of traits using American Housing Survey (AHS) data from 1993 and 1995, using as a base of analysis occupied units that were retired from the stock between 1993 and 1995. As reported in Table I, the 171,000 occupied homes retired from the housing inventory between 1993 and 1995 were generally older, renter-occupied, and in substandard condition.

To determine the resulting difference in life years spent in new and existing housing, we build a simple dynamic model in which housing unit retirement is a function of age of the unit. We divide homes into four age classes (0-13, 14-33, 34-53, 54+ years) representing 21%, 37%, 21%, and 21% of the current stock, respectively (1993 AHS data). Baseline annual retirement rates of (0, 0.0006, 0.0011, 0.0016) are estimated by comparing 1993 and 1995 AHS data. Given these rates, we assume that the age category of 0-13 years (with no retirements) is representative of the stock of higher quality new units. We assume that 1.1 million households are added per year, with 1997 baseline market characteristics. To determine the reduced number of life-years spent in new units by a single-year cohort, we reduce the total retirement rate by 0.0011% by lowering all age-specific retirement rates by 1.6% from baseline for a single year, and analyze the corresponding age distribution over time. Under this assumption, a single-year reduction of 1,350 new units balanced by reduced retirements results in 13,400 fewer present-value life years spent in new units, which are presumed to be distributed among units that would have otherwise been retired from the market.

The estimated effect of increased construction prices on the incremental exposure of households to the existing residential stock depends on a number of factors, such as choices about model specification and the data used for estimation. We approximate the effect of these uncertainties using a simple uncertainty factor of five, based loosely on some of the larger price elasticities cited above and the estimated quality effect that is 3-4 times larger than the quantity effect. Incorporating this factor symmetrically suggests that the number of additional present-value life years spent in existing units ranges between about 3,000 and 70,000.

3.2. *Income Effect*

Estimates of the effect of reduced income on higher mortality risk compare annual income with mortality rates. Consequently, we derive an effective change in annual disposable income that results from the increased cost of new housing. For the approximately 1.1 million less 1,350 households who would purchase new homes despite the cost increase, we assume that the $132 increase in effective price calculated above would reduce investment income or increase mortgage payments throughout the lifetime of the household. Assuming a real return to investment of 3%, this implies a permanent annual household income loss of $3.96. Due to the discounting of future years, the findings are not substantially different if a limited mortgage payment schedule or investment horizon is assumed.

The demographic characteristics of these households are taken as those of households owning units less than 5 years old, as reported in the 1993 AHS data. These households have an average household size of 2.8, with a mean household income of $52,000. An estimated 55% of household members are between the ages of 25 and 64, and 88% of heads of household are white. We assume all household members are of the same race as the head of household.

We estimate the income effect using Keeney's model [(51)], which relates annual mortality risk to family income, race, and gender using the equation

$$r(x) = a\, e^{-b x} + d \qquad (7)$$

where r(x) is the annual mortality probability and x is annual household income in thousands of dollars. Keeney estimated model parameters using data for white and black adults aged 25-64; lacking better information, we apply it to all age groups and use the parameters for blacks for all non-white households.[18] Simulated increases in annual mortality risk range between 2×10^{-13} and 3×10^{-6}, with an average value of 2×10^{-7}. Aggregating across the population yields a present value of 22 statistical fatalities (0.7 statistical fatalities per year).

To calculate $QALY_{PV}$ reductions, baseline annual mortality rates were taken from life table data [(75)] assuming constant mortality rates through 5-year age categories and a constant rate for all individuals older than 85 [(29)]. Using standard values for healthy life years

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[18] Estimated model parameters (a, b, d) are white males: (0.00926, 0.0450, 0.00422), black males: (0.0122, 0.0541, 0.00614), white females: (0.0039, 0.0708, 0.00277), black females: (0.00701, 0.0698, 0.00343).

remaining for different age groups [25], the per capita $QALY_{PV}$ reduction averages 7 x 10^{-5} with values ranging between 2 x 10^{-11} and 1 x 10^{-3}. Aggregating across all households yields a population total of 212 $QALY_{PV}$. In other words, reducing the income of approximately 1.1 million households by about $4 per year for the remainder of their lives would result in a loss of about 210 quality-adjusted life years, discounted appropriately.

Keeney's estimates imply that a cost of $11.4 million (allocated in proportion to income) induces one premature fatality. Other direct estimates range from $6.5 million to $12.2 million [13,14,49] while Viscusi's [111] indirect estimates are on the order of $15-100 million. This suggests alternative estimates of the income effect between one-tenth to twice as large (21-420 $QALY_{PV}$, 2-44 premature fatalities).

3.3. *Stock Effect*

Households occupying units at the bottom of the market are exposed to elevated levels of many risk factors. Table II depicts a number of pathways by which the condition of the structure can be quantitatively linked to the health of occupants or, through air emissions, of others. Outcomes associated strictly with the location of the structure or that do not have a quantifiable endpoint associated with housing quality (e.g., crowding) are not considered.

For some of the pathways, such as radon exposure or exacerbation of asthma or allergies by indoor air pollutants, the overall mortality risk is high but the association is more strongly related to source strengths or air exchange rates than housing quality. We focus on three pathways with a strong causal link to the quality of the home and with significant mortality or morbidity outcomes--energy consumption, fires, and lead in paint. Restricting consideration to these three factors is likely to underestimate the total stock effect but demonstrates the methodology for including additional risks and suggests the order of magnitude expected from a more comprehensive analysis. Within these categories, only direct health impacts estimable as QALYs are considered. Other outcomes, such as property damage from fires and climatic impacts of energy consumption are omitted. For each category, we provide a plausible central value as well as rough estimates of upper and lower bounds. These uncertainty intervals are conditioned on the central estimate of the housing-market effect. Uncertainty about the magnitude of the housing-market effect is subsequently combined with uncertainties about the specific pathways to yield an overall uncertainty range.

3.3.1. *Mortality from Energy Use*

Homes that consume more energy are responsible for greater pollutant emissions, both from residential combustion sources and from power plants producing electricity for the home. This can lead to adverse health impacts to the large population outside the household, an attribute that distinguishes energy consumption from the other health risks. Older homes with higher retirement rates tend to consume more energy than new homes, but the question remains as to how much of this difference is associated with characteristics of the household (such as income or household size) and how much is associated with the structure itself.

To account for these factors, we estimate multivariate regression models from 1993 Residential Energy Consumption Survey (RECS) microdata, stratifying energy consumption by end use and fuel type (Appendix 1). We assume that, although retrofitting occurs, characteristics such as insulation and central air conditioning are causally linked to the age of the structure and therefore omit them from the regression equations. We further assume that the retained homes are in the same geographic regions as the avoided housing starts, that they are of the same size and type, and that household composition and appliance ownership are not affected.

Thus, for our central estimate, we assume that new and retained homes will differ only in age and its associated structural characteristics. Given the median retained home age of 44 years and depending on fuel type, the retained home would have 33-57% greater space heating energy consumption, 28% lower space cooling energy consumption, and 0-4% lower energy consumption for all other purposes, when compared to new homes. To determine the resulting fuel consumption in retained units, we assume baseline new home energy consumption to be that of post-1990 units in RECS and derive energy consumption patterns of retained units from cross-tabulations of RECS data by the age distribution in AHS. In the aggregate, an additional life-year spent in a retained home is estimated to reduce electricity consumption by 5 million BTU, but increase direct natural gas consumption by 29 million BTU and fuel oil consumption by 16 million BTU. We assume the electricity is produced with the national average fossil fuel mix, a common assumption for changes in residential electricity consumption [(4)]. Assuming a power plant efficiency of 35% and a residential annual fuel utilization efficiency (AFUE) of 80%, the difference in energy consumption between new and retained homes corresponds to 0.4 fewer tons of coal, 144 more gallons of fuel oil, and 35 more MCF of natural gas consumed.

To estimate health impacts, we use a damage function model created for power plants, in which changes in fuel combustion are translated into pollutant emissions, concentration changes at a number of receptors are estimated using dispersion models and chemical conversion equations, and health outcomes are estimated from epidemiological studies on the primary and secondary pollutants. Although there are fundamental differences in dispersion patterns and exposed populations for residential combustion and power plants, mass balance notions suggest this approach provides a reasonable approximation of aggregate impacts.

We estimate health effects using the EXMOD software [(90)]. Created to estimate externalities of New York State power plants, EXMOD is calibrated to a population with density comparable to the U.S. average (29.3 vs. 30.9 people/km^2) and typically yields results in the middle of the range of other studies [(27,79)]. QALY estimates are based on mortality and chronic bronchitis outcomes. A quality of life (QoL) reduction of 23% of age-specific health quality is estimated for adults with chronic bronchitis (median age of 56), or 2.5 $QALY_{PV}$ per case[19] [(37)]. Default characteristics of natural gas and fuel oil power plants are assumed [(90)].

Estimated changes in health outcomes per unit of fuel combustion are reported in Table III. Combining these with energy consumption changes implies that each additional life-year spent in a retained home corresponds to an estimated 2.1 x 10^{-5} $QALY_{PV}$ lost in the national population. Multiplying by 13,400 additional present value life-years in retained homes, the central estimate for the energy component of the stock effect from a single-year cohort totals 0.3 $QALY_{PV}$.

The complexity of the impact assessment models preclude formal uncertainty analysis here. As a crude estimate, we judge that our impact calculation can potentially differ by two orders of magnitude, depending on the fuel source for electricity, population density, variation in meteorological conditions, and dose-response functions. The impact would be reduced by a factor of 6 if we assume (consistent with AHS data) that new homes are on average larger than the homes that would be retired. Thus, our approximate bounds on the energy estimate

[19] Consistent with this apparently large decrement in health-related quality of life, willingness to pay to avoid a statistical case of chronic bronchitis has been estimated as $1 million (mean) and $0.5 million (median) [(112)].

are 0.0005-30 $QALY_{PV}$.

3.3.2. *Mortality and Morbidity from Lead*

Inhalation and ingestion of lead in household dust appear to be causally associated with a range of adverse health effects. Exposed children may suffer a permanent cognitive deficit, and adults risk hypertension, stroke, and heart attack. A major source of lead in house dust is deteriorating lead-based paint, which is far more prevalent in older homes; an estimated 83% of pre-1980 housing has lead-based paint, which is minimal in newer units [(108)]. In addition, 22% of retained units have at least 1 ft^2 of peeling paint and retained units can have higher soil-lead concentrations due to exterior lead-based paint and other outdoor sources.

We estimate exposure from measurements taken and models derived for the HUD National Survey [(108)]. For our bounding analysis, we consider retained units to have either median (lower bound) or 95th percentile (upper bound) lead concentrations or loadings for their age category, with the 75th percentile used for the central estimate. For all estimates, we consider changes in interior dust only; although soil lead concentrations are higher in older units, this is largely due to outdoor sources that are presumably unrelated to the structure.

The choice of concentration (μg/g) or loading (μg/ft^2) is based on the terms in the Integrated Exposure, Uptake, and Biokinetic Model for Lead in Children (IEUBK), a model that evaluates the relationship between lead exposures and blood lead levels (PbB) in children [(107)]. We use the median estimates from 1960-1979 units without lead paint to approximate new units, since no measurements were taken in newer units. Under these assumptions, retained units have floor dust-lead loadings of 29 μg/ft^2 (range: 11-113) and window sill dust-lead loadings of 464 μg/ft^2 (range: 55-3240). New units have floor dust-lead loadings of 3 μg/ft^2 and window sill dust-lead loadings of 8 μg/ft^2.

We estimate the effects of these differences on PbB by applying the IEUBK model to HUD National Survey data (Appendix 2) [(6)]. Assuming fair/poor paint quality in 22% of retained units and 0% of new units and identical rates of pica, we find PbB differences of 1.4 μg/dL (range: 0.8-2.2). For comparison, a literature review estimated the relationship between PbB and dust lead to be 5 μg/dL/1000 ppm [(18)], which would imply a 2.6 μg/dL difference in

PbB using the 75th percentile difference in dust-lead concentrations between retained and new units. We also note that NHANES III Phase II data show a 1.8 μg/dL difference between geometric mean concentrations in children in pre-1946 and post-1973 housing, categories that would likely differ less than retained and new units. Since our estimates do not consider soil lead concentration differences, which are incorporated in these other estimates, our estimates appear to be reasonable. Including soil lead differences would lead to a central estimate about twice as large, 2.5 μg/dL.

The effect of PbB on IQ is estimated by a recent meta-analysis, which finds a reduction of 2.6 IQ points for school-age children given an increase in PbB from 10 to 20 μg/dL. The trend is linear with no evidence of a threshold [93]. Reductions in IQ can affect individual quality of life, reduce future income, and increase educational costs. To estimate QALYs, we consider the increased probability of mental retardation (IQ < 70), as estimated in the EPA model and implemented in EXMOD (Appendix 2) [1]. The function is stepwise linear and assumes that IQ decrements occur following 3-4 years of exposure for children aged 7 and younger. To approximate the effect from one incremental year of exposure, we assume that the effect is linear across the 3-4 year period and divide by 3.5. For an upper bound estimate, we assume that the complete effect occurs within the year. As an order-of-magnitude approximation of a lower bound, we simply assume the effect to be a factor of 10 below the total effect.

For severely reduced cognitive ability, we approximate the impact as a 13% QoL reduction from baseline at all ages, using a standard assessment instrument (HUI Mark II) and a moderately high level of cognitive impairment with no associated physical impairments [80]. There is also evidence of reduced life expectancy associated with decreased mental capacity [69] or learning disabilities [66]. As an upper bound estimate (given the number of comorbid physical maladies associated with mental retardation), we assume the lifespan reduction to be completely related to the retardation and use the population-based standardized mortality ratio estimates associated with learning disabilities. We assume no lifespan reduction for the lower bound and use the mean of these values for our central estimate. Combining the quality and longevity effects yields total losses of 5.0 $QALY_{PV}$ (range: 3.2-6.7) per case.

Given that the average household in retained homes has approximately 0.6 children age 7 or younger, an additional life-year in a retained home leads to an estimated reduction of

4×10^{-4} $QALY_{PV}$ (range: 3×10^{-5} - 2×10^{-3}). The aggregate loss associated with the increased cases of mental retardation is approximately 6 $QALY_{PV}$ (range: 0.4-26). With the inclusion of soil lead, the loss would be 10 $QALY_{PV}$ (range: 0.8-70).

Since the IEUBK model is constructed for children, we base our PbB estimates for adults on a different biokinetic model [11]. This model uses a biokinetic slope factor of 0.375 μg/dL PbB per μg/day lead uptake and assumes a typical dust ingestion rate of 20 mg/day and 8% absorption of dust lead. For the interior dust concentration estimates, we use an average of floor and window sill concentrations (weighted by the IEUBK coefficients). The estimated PbB difference between adults in retained and new units is 0.8 μg/dL (range: 0.2-1.4). This value is comparable to the NHANES III geometric mean difference of 0.7 μg/dL between adults 40 years or older in pre-1946 and post-1973 homes. As for children, we assume that adult health risks from lead depend only on current lead exposure, which may underestimate impacts associated with accumulated bone lead.

Adult PbB is related to both systolic and diastolic blood pressure. To avoid double-counting, we consider only systolic blood pressure (BP_s), which is a stronger predictor of cardiovascular events and mortality [68,87,101]. The relationship between PbB and BP_s can be modeled as log-linear with no apparent threshold [42,82]. A recent meta-analysis [99] estimates that doubling PbB corresponds to a 1.2 mm Hg increase in BP_s (95% CI: 0.2-2.2) for men and a 0.8 mm Hg increase (95% CI: 0.2-1.5) for women.

We consider three distinct outcomes associated with increases in blood pressure: non-fatal myocardial infarctions (MI), non-fatal strokes, and all-cause mortality (including fatal MI and strokes). Baseline BP_s by age is derived from NHANES III data.

For non-fatal MI, we use the logistic regression function generated from the Pooling Project to estimate the risk of MI associated with increased BP_s, including age as a covariate [84] (Appendix 2). This function is estimated for white males 40-59 years of age, but some evidence exists of a relationship for both women and older populations [101,106]. For our central estimate, we assume the relationship can be extrapolated to all males over age 40. The lower bound considers only males age 40-59 and the upper bound considers all individuals over age 40. MI fatality rates by age are taken from the Worcester Heart Attack Study [34] and range between 3% for individuals younger than 55 to 23% for individuals older than 75.

Life expectancy reductions are calculated assuming that annual mortality risk in each year following an MI is equal to the population baseline rate plus 1.5% [10]. The QoL value for non-fatal MI is 0.75, roughly equivalent to mildly impaired or symptomatic heart disease (QoL values of 0.70 and 0.82, respectively [80]). Combined with the estimated 0.2 males over age 40 per retained household, an added life-year in a retained home corresponds to a loss of 7×10^{-5} $QALY_{PV}$ (range: 2×10^{-6} - 4×10^{-4}) and the total non-fatal MI component is estimated as 0.9 $QALY_{PV}$ (range: 0.02-5.9).

The incidence of stroke is modeled as a function of BP_s using multivariate regressions from an analysis of the Framingham study, which assessed the probability of stroke in adults age 55-84 as a function of gender and subject characteristics [117]. We substitute Framingham study population means for all covariates except age and BP_s (Appendix 2). The stroke-BP_s relationship is applied to all adults age 40 and over, based on studies finding a positive relationship for all adults [62].

Life expectancy reductions are based on constant annual survival rates among stroke victims in different age categories [7], and the QoL reduction post-stroke is assumed to be 20% [3]. An additional life-year in a retained home yields a 3×10^{-5} loss in $QALY_{PV}$ (range: 2×10^{-6} - 9×10^{-5}) due to strokes, for a total of 0.4 $QALY_{PV}$ (range: 0.02-1.2).

Finally, a logistic regression function was estimated from a table relating all-cause mortality to BP_s, controlling for age, race, income, and a number of behavioral factors [101] (Appendix 2). Although this function was generated for males age 35-64, similar rates were seen in women as well. We consider male-only as the lower bound, and all adults over 40 for both the central and upper bound estimates. The average annual mortality risk increase from the estimated increase in BP_s is found to be 3×10^{-5} (range: 2×10^{-6} - 9×10^{-5}). Incorporating the average $QALY_{PV}$ remaining for each age category and the number of adults over 40 per household yields a loss of 2×10^{-4} $QALY_{PV}$ (range: 4×10^{-6} - 5×10^{-4}) per additional life-year in a retained unit, for a total of 2.1 $QALY_{PV}$ (range: 0.05-6.3). Thus, the total impact from all lead pathways is estimated as 9 $QALY_{PV}$ (range: 0.5-39).

3.3.3. *Mortality from Fire*

The mortality risk from house fires is clearly higher in older homes, with survey data indicating a mortality rate of 5.2×10^{-5} in pre-1970 units and 9.0×10^{-6} per unit in homes built between 1981 and 1986 [73]. However, as with the energy pathway above, a portion of this difference can be related to characteristics of the household (e.g., smoking status, alcohol consumption) that would presumably not be altered by housing market shifts. We take the observed difference as an upper bound estimate, and determine our central estimate of the increased fire risk in retained homes by focusing on the two potential routes of influence.

First, the retained older homes can have building materials, space heaters, faulty wiring, or other characteristics that might lead to a greater risk of a fire starting. The probability of an electrical system fire (8% of all residential fires) is over 5 times greater in homes older than 40 years than in homes less than 10 years old [96]. In addition, 15% of residential fires can be attributed to home heating, including fireplaces, fixed-area heaters, central heaters, and portable heaters [40]. Given the distribution of heating sources in AHS and the fire incidence attributed to each heating source by Hall [40], the risk of heating source fire in retained homes is 5.8×10^{-4}, versus 3.5×10^{-4} fires per year in post-1990 homes. Finally, although the incidence of fires might differ by building quality or materials, this factor could not be quantified. Fire risk associated with building materials would not be anticipated to be significant, given the similar composition of old and new homes in RECS (23% brick and 38% wood in pre-1940, 22% brick and 33% wood in post-1990).

Older homes might also have fewer functional smoke detectors, greater structural inadequacy, or lesser ease of exit, which might lead to greater mortality risks per fire that occurs. A study in North Carolina found the fatality rate per fire to be 100% greater in homes 20 years or older than in newer homes [91]. However, this estimate did not control for a number of correlated factors that can also influence fire fatality rates. Focusing on smoke detectors, although studies have shown that presence of a smoke detector is associated with a 40-50% lower mortality risk per fire [39], a multiple regression model found that this efficacy is reduced to 9% when factors such as household income and smoking status are controlled [32]. Given that 94% of new homes and 75% of retained homes have operational smoke detectors [28], this would imply a fatality rate per fire only 2% higher in retained homes. To bridge the large gulf between these estimates, we apply the percentage attenuation from the smoke detector study to the overall fatality estimate, yielding an approximately 20% higher fatality rate per fire in retained units compared to new units. Although this is a crude estimate, it is likely reasonable given the above bounding estimates. We use the 2% figure for our lower bound estimate.

For our QALY estimate, we use the 1995 figures of 3,600 annual civilian fire deaths in 425,500 residential fires [(48)]. Given evidence of a "U-shaped" fire mortality risk function versus age [(5)], the population average for $QALY_{PV}$ remaining is used. Using the above figures and assuming constant fire risk outside of electrical or heating fires, our central estimate for the annual number of fires per household is 5×10^{-3} in retained homes and 4×10^{-3} in post-1990 homes, with a calculated death rate of 1×10^{-2} per fire in retained homes and 8×10^{-3} in new homes. Thus, an additional life-year in a retained unit corresponds to 2×10^{-4} $QALY_{PV}$ (range: 1×10^{-4} - 8×10^{-4}) due to increased fire mortality, for a total of 3 $QALY_{PV}$ (range: 1-10).

3.4. *Summary of Numerical Application and Uncertainties*

As summarized in Table IV, a code change that increases the effective price of new homes by 0.1% is estimated to yield indirect health effects on the order of 20-800 $QALY_{PV}$, with a central estimate of 225 $QALY_{PV}$. If the cost increase is permanent, increasing the cost and reducing the retirement rate in perpetuity, the resulting health effect would total about 7,700 $QALY_{PV}$. Similar calculations can be made for other potential time scales, such as 10 years (2,000 $QALY_{PV}$) and 20 years (3,400 total $QALY_{PV}$).[20] Our calculations suggest that the income effect predominates, accounting for about 210 of the total 225 $QALY_{PV}$ per annual cohort. About half the estimated stock effect (6/13) results from the effect of lead on children's cognitive development, with fire and the effects of lead on all-cause mortality accounting for most of the rest of the effect. Although we have quantified only a few of the pathways by which decreased construction of new homes could influence health, we believe we have included the most significant pathways.

These results demonstrate that it is possible to estimate the magnitude of adverse health effects that may be induced by code changes which increase housing costs. As is often true in risk analysis, the calculations are elaborate and rely on a variety of data sources and

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[20] To give a sense of the interpretation of these magnitudes, the QALY increase associated with a 45-year old quitting smoking has been estimated as approximately 2 $QALY_{PV}$ [(30)]. Thus, the effect of a 0.1% price increase on 1.1 million households (225 $QALY_{PV}$) purchasing new homes in one year is approximately the same as if about 110 fewer 45-year old smokers quit in a single year.

untested assumptions. We review the sensitivity of the numerical results to several of these assumptions, including the discount rate, the estimated relationship between income and health, and the use of age as a proxy for housing quality.

The discount rate used to compare present and future health effects and the interest rate used to convert the increased housing price into an annual income decrement have a moderate effect on the results. We used a value of 3% for both rates; using 5% for both rates, the income effect consists of an identical number of statistical fatalities (since the greater annual income loss is offset by the greater discount rate) but a reduction in associated $QALY_{PV}$ from 212 to 198 (because the lost future years of life are discounted more heavily). The statistical fatalities due to the stock effect are virtually unchanged, and the lost $QALY_{PV}$ declines from 12 to 7 (again, because of stronger discounting of future losses). In sum, using a 5% rate reduces the estimated lost $QALY_{PV}$ from 225 to 205.

The income effect accounts for the bulk of our health effect. The study we used yields results that are broadly consistent with this literature. The possibility that reductions in disposable income would, on average, increase health risks is extremely plausible, but the development of reliable empirical estimates of the magnitude of the effect is challenging. The available literature suggests that the effect is larger for lower-income households and for men, so the incidence of increased costs by income group and gender may be important. The effects of lost income on morbidity were not considered, but would increase the total health risk. Other factors that contribute uncertainty to the estimate include assumptions regarding decreased investment income versus increased interest-bearing debt and the allocation of income within households.

The stock effect was estimated on the assumption that changes in the age of homes affect health, but that reductions in the average quality have no significant effect. Because the effect of the simulated increase in construction costs on the average value per unit is 3-4 times larger than the effect on the number of new homes, this omission could be significant. We also neglected any effect of the net reduction in units in the housing stock, assuming these would be reflected in fewer second homes or lower vacancy rates. If the reduction in total units were to increase crowding, there could be additional health losses [31,57-59,63].

The stock effect is estimated on the basis of only three pathways. Omitted pathways would likely increase the total effect. The estimates corresponding to each of the included pathways are subject to a variety of uncertainties. The energy component depends on local conditions and results could differ by at least two orders of magnitude, depending on the fuel source for electricity, population density, and meteorological conditions governing local emissions. In addition, there are uncertainties about the share of the age-related difference in energy use attributable to the structure as well as the dose-response functions relating air pollutant levels to health effects. For lead, assumptions about the pattern and duration of exposure and its relationship with blood lead concentration changes add some uncertainty, and assessing only the cognitive impairment threshold of mental retardation may lead to inaccuracies in determining the impacts of marginal IQ reductions. For cardiovascular impacts, assumptions about the age and gender cutoffs, the temporal relationships between events, and the ability to extrapolate functions along these categories contributes to the uncertainty. Finally, for fire risk, common low-quality house attributes such as inadequate exit routing and poor building condition could increase risks, and the estimate does not consider possible behavioral changes with housing unit. For all outcomes, the quality of life estimates are crude and may suffer from lack of comparability due to differences in populations and study designs.

Despite their limitations, the calculations demonstrate that tangible values can be placed on the health losses induced by code-related costs. Our initial estimate is that a national code change that increased housing costs $150 would annually induce a loss of 2 to 60 (present value) premature fatalities. Including morbidity, the loss is estimated as about 20 to 800 $QALY_{PV}$, the majority accruing (via the income effect) within a cohort of 1.1 million new home buyers. For changes in construction costs that are small compared with the price of housing, our calculations are proportional to the price change so these estimates can be readily extrapolated.

4. Conclusion

We have presented an analytic approach to account for "affordability" considerations in the development of new residential building codes aimed at protecting the health and safety of residents. Unlike benefit-cost analysis, which requires measuring the health and safety benefits of a proposed code change in dollars, we propose a risk-tradeoff approach that allows code officials to compare the countervailing health and safety risks of code changes with estimates of their benefits in comparable health units [61].

The risk-tradeoff test--which implies a code change is beneficial if the health and safety benefits of the change (net of any direct countervailing risks) outweigh the health risks created by the income and stock effects--is less stringent than a conventional benefit-cost test [111]. Code changes that fail the risk-tradeoff test would also fail a benefit-cost test (unless they provide non-health benefits that exceed the health costs). The difference arises because benefit-cost analysis, but not risk-tradeoff analysis, accounts for other benefits in addition to health and safety that are foregone by increasing construction costs. Households bearing the income effect would have spent some of that money on other goods they value in addition to health, and households affected by the stock effect would have valued the other non-health amenities of living in a newer home.

Our primary purpose in this article has been to develop a conceptual risk-tradeoff approach for evaluating code provisions and to provide an illustrative numerical application to demonstrate the feasibility of the approach and the possible magnitude and distribution of the impacts. Our approach builds on previous risk-analytic literature by applying risk-tradeoff concepts and tools to the housing construction industry. Nevertheless, it has some important limitations.

First, we have not identified and quantified all of the uncertainties associated with our numerical estimates. In a complete application, a number of stages of the analysis would have significant uncertainties. An application would require estimation of the magnitude of construction cost increases associated with a proposed code change, the possibility of cost savings (or increases) over the life of the dwelling, the incidence of costs incurred by builders and consumers, the response of home buyers to the effective price increase, the magnitude of the income-health relationship, the effects of codes on the housing retirement rate, and the incremental health and safety risks associated with living in units at the bottom of the housing market. It would require a far more complex model to quantify all of these uncertainties and express their cumulative impact on our numerical estimates. A serious uncertainty analysis would also be required to determine with any confidence whether the income effect is indeed larger than the stock effect, as our illustrative estimates suggest.

Second, we have not applied the risk-tradeoff approach in any real-world case studies of proposed code changes. Specific cases would allow us to quantify the risk-reductions anticipated from adopting the code provision, for comparison with the countervailing risks considered here. Conducting such case studies would be useful for evaluating the feasibility and utility of the risk-tradeoff approach. It would also be useful to conduct such case studies in collaboration with code officials, insurers, builders, and other stakeholders committed to bringing more analytic rigor to the code-making process.

Finally, we were disappointed with the quality of the epidemiological literature relating housing characteristics to health status. Given that the majority of most people's life-hours are spent in homes, residential pathways may be an important source of exposure to a number of risk factors. We recommend that health research organizations fund large, prospective studies of the long-term impact of housing characteristics on human health. Such studies would also be useful for the purposes of quantifying the risks of existing design features as well as the benefits of existing codes.

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Table I: Characteristics of Occupied New and Retained Housing Units

Characteristic of unit	*Retained units*[a]	*New units*[b]
Median age	44 years	2 years
Geographic location	49% in South 20% in Midwest 18% in Northeast 13% in West	41% in South 22% in Midwest 12% in Northeast 26% in West
% in urban setting	36%	17%
% of units rented	83%	20%
Quality of unit	67% adequate 20% moderately inadequate 13% severely inadequate	95% adequate 3% moderately inadequate 1% severely inadequate
% of units with cracks/holes in wall or ceiling	33%	1%
% of units with peeling paint over 1 square foot	22%	0.3%
Heating fuel distribution	50% natural gas 18% electric 14% fuel oil	48% natural gas 44% electric 4% fuel oil
Mean household income	$10,000	$52,000
Household size (est. sq. ft.)	1100	1800
Number of people	3.1	2.8
Number of children age 0-7	0.6	0.4

Source: American Housing Survey, 1993 and 1995.
a. Units removed from stock by disinvestment or structural deficiencies; removals by fire and natural disaster are excluded.
b. Units five or fewer years old.

Table II: Health risks in the home hypothesized to be related to the condition of the structure, ranked by the baseline mortality risk estimate. Note that not all deaths in each category occur in the home or as a result of the home.

Mortality risk	*Est. total deaths/year in United States*	*Other major health impacts*	*Authors' judgement of strength of causal link with structure*	*Sources*
Radon	7000-15,000		Weak	(109)
Residential energy consumption	**800-19,000**[a]	**Chronic bronchitis** **Hospital admissions**	**Strong**	**(23, 27, 90)**
Falls at home	7000	Fractures Restricted activities	Moderate	(76)
Asthma/allergies	5000	Asthma attacks Doctor visits	Weak	(71)
Residential fires	**3600**	**Burns**	**Strong**	**(48)**
Heat	1000		Moderate	(72)
Hypothermia	700		Moderate-weak	(74)
Tornadoes	400		Moderate	(92)
Hurricanes	40		Weak	(24)
Earthquakes	10-40		Moderate	(103)
Lead paint	**NQ**[b]	**IQ decrement** **Hypertension**	**Strong**	**(93, 94)**

[a] Approximated from total residential energy consumption of 16.6 quads (6.7 primary, 9.9 for electricity) and estimated mortality rate from fossil fuel electricity generation from two externality studies of coal, oil, and natural gas. Indoor air impacts are not included.
[b] NQ = direct mortality impact not quantified

Table III: Central estimates of health effects associated with power plant combustion of natural gas or fuel oil.

	Particulate matter (primary and secondary)	*Ozone*	*Estimated $QALY_{PV}$ lost/case*	*Expected $QALY_{PV}$/ unit fuel*
Coal (Cases/ton) [1]				
Mortality, > 65	1.0×10^{-6}	-	5.8	5.8×10^{-6}
Mortality, < 65	8.8×10^{-8}	-	19.8	1.7×10^{-6}
Mortality, all ages	-	6.0×10^{-7}	18.1	1.1×10^{-5}
Chronic bronchitis (> 25)	4.6×10^{-6}	-	2.5	1.2×10^{-5}
TOTAL				3.0×10^{-5}
Natural gas (Cases/MCF) [2]				
Mortality, > 65	1.0×10^{-8}	-	5.8	5.8×10^{-8}
Mortality, < 65	9.1×10^{-10}	-	19.8	1.8×10^{-8}
Mortality, all ages	-	1.1×10^{-8}	18.1	2.0×10^{-7}
Chronic bronchitis (> 25)	4.7×10^{-8}	-	2.5	1.2×10^{-7}
TOTAL				3.9×10^{-7}
Fuel oil (Cases/gallon) [3]				
Mortality, > 65	5.1×10^{-9}	-	5.8	3.0×10^{-8}
Mortality, < 65	4.4×10^{-10}	-	19.8	8.7×10^{-9}
Mortality, all ages	-	2.0×10^{-9}	18.1	3.6×10^{-8}
Chronic bronchitis (> 25)	2.3×10^{-8}	-	2.5	5.8×10^{-8}
TOTAL				1.3×10^{-7}

[1] Given fuel heat content = 13,100 BTU/lb, heat rate = 9856 BTU/kWh
[2] Given fuel heat content = 21,824 BTU/lb, heat rate = 9400 BTU/kWh
[3] Given fuel heat content = 19,000 BTU/lb, heat rate = 9400 BTU/kWh

Table IV: Summary of health risks from evaluated pathways (numbers may not sum due to rounding).

Category	*PV statistical fatalities*	*$QALY_{PV}$ lost*	*People at elevated risk per year*
Income effect	22 (2-44)	212 (21-420)	3.1 million
Stock effect, three pathways	0.4 (0.1-2.9)	13 (1.5-79)	
Energy		0.3 (0.0005-30)	≈ 250 million
Lead: IQ	-	6 (0.4-26)	800
non-fatal MI	-	0.9 (0.02-5.9)	300
non-fatal stroke	-	0.4 (0.02-1.2)	600
all-cause mortality		2.1 (0.05-6.3)	600
Fire		3 (1-10)	3,800
Housing market uncertainty	0.2-5.0	0.2-5.0	
TOTAL	22 (2-60)	225 (20-800)	

Notes: The stock effect intervals are conditioned on the central estimate of the housing market effect. The housing-market uncertainty factor accounts for uncertainty in the number of households remaining in older units and is combined with the stock effect intervals to calculate the uncertainty ranges for the total effect.
Using a 5% discount rate to calculate present values and 5% rate of return to amortize the wealth effect of higher housing costs yields:
Income Effect: 22 (2-44) statistical fatalities and 198 (20-400) $QALY_{PV}$ lost;
Stock Effect: 0.3 (0.1-2.5) statistical fatalities and 7 (1.2-48) $QALY_{PV}$ lost;
Total: 22 (2-44) statistical fatalities and 205 (21-440) $QALY_{PV}$ lost.

Appendix 1: Stock Effect Risk Calculations, Energy Consumption

Define:
Elecheat = electric space heating consumption (MBTU)
NGheat = natural gas space heating consumption (MBTU)
FOheat = fuel oil space heating consumption (MBTU)
AC = electricity consumption for space cooling (MBTU)
Elecoth = electricity consumption for non-space conditioning (MBTU)
NGoth = natural gas consumption for non-space conditioning (MBTU)
FOoth = fuel oil consumption for non-space conditioning (MBTU)
Age = Age of home, 1993 base
HDD65 = Heating degree days, 65 degree base
CDD65 = Cooling degree days, 65 degree base
Sqft = Size of home, in ft^2
Applnum = number of major appliances owned (stove, microwave, refrigerator, freezer, washer, dryer, dishwasher, humidifier, dehumidifier)
Income = annual household income
HHsize = number of people in household
Eleccost = cost of electricity, $/MWh
NGcost = cost of natural gas, $/100 MCF
FOcost = cost of fuel oil, $/MBTU
Detach = 1 if home is single-family detached, 0 otherwise
Own = 1 if home is owned, 0 otherwise
Urban = 1 if home is in a city or town, 0 if in a suburb or rural area
Northeast = 1 if home is in Northeast, 0 otherwise
Midwest = 1 if home is in Midwest, 0 otherwise
South = 1 if home is in South, 0 otherwise

The following regressions were estimated using 1993 Residential Energy Consumption Survey microdata, with variables included using stepwise selection based on Mallows' C_p statistic. Regressions are estimated using only households with non-zero energy consumption within the energy category.

Variable	Ln(Elecheat)	Ln(NGheat)	Ln(FOheat)	Ln(AC)	Ln(Elecoth)	Ln(NGoth)	Ln(FOoth)
Intercept	3.89 (0.29)	0.55 (0.20)	0.24 (0.67)	1.29 (0.28)	10.5 (0.18)	10.1 (0.40)	9.22 (0.20)
Age	0.010 (0.0007)	0.0080 (0.0004)	0.0065 (0.0008)	-0.0075 (0.0005)	-0.0008 (0.0003)	-	-
Ln(HDD65)	0.55 (0.013)	0.69 (0.014)	0.73 (0.053)	-	-0.037 (0.010)	0.085 (0.018)	-
Ln(CDD65)	-	0.10 (0.014)	0.14 (0.036)	0.69 (0.021)	-	-	-
Ln(Sqft)	0.29 (0.028)	0.47 (0.016)	0.38 (0.037)	0.30 (0.025)	0.14 (0.013)	0.13 (0.021)	-
Ln(Income)	-	-	-	0.11 (0.013)	-	-	0.046 (0.020)
Ln(HHsize)	0.083 (0.019)	-	-	0.21 (0.019)	0.38 (0.010)	0.51 (0.018)	0.52 (0.031)
Ln(Eleccost)	-0.44 (0.043)	-	-	-0.39 (0.047)	-0.54 (0.024)	-	-
Ln(NGcost)	-	-	-	0.016 (0.0031)	-	-0.32 (0.056)	-
Ln(FOcost)	-	-	-	-	-	-	-
Applnum	0.056 (0.0082)	-	0.030 (0.011)	0.055 (0.0076)	0.12 (0.0041)	-	-
Detach	0.25 (0.028)	0.19 (0.020)	0.17 (0.045)	-0.0021 (0.027)	-	-	-
Own	0.24 (0.030)	0.14 (0.020)	0.21 (0.045)	0.13 (0.028)	0.058 (0.015)	-0.070 (0.024)	-
Urban	0.085 (0.023)	-	-	-	0.083 (0.012)	-	-
Northeast	0.23 (0.047)	-	0.20 (0.036)	-	0.10 (0.021)	-	-
Midwest	0.21 (0.039)	-	-	0.25 (0.029)	0.065 (0.019)	0.062 (0.029)	-0.41 (0.13)
South	0.26 (0.026)	0.070 (0.022)	-	0.45 (0.035)	0.24 (0.016)	0.14 (0.025)	-
R^2	0.77	0.65	0.50	0.56	0.55	0.24	0.50

Appendix 2: Stock Effect Risk Calculations, Lead Consumption

Define:
BPS = systolic blood pressure (mm Hg)
BPS_{new} = systolic blood pressure, new home exposure assumed (mm Hg)
$BPS_{retained}$ = systolic blood pressure, retained home exposure assumed (mm Hg)
PbB_{new} = blood lead concentration, new home exposure assumed (μg/dL)
PbB_{bottom} = blood lead concentration, retained home exposure assumed (μg/dL)
PbB = blood lead concentration (μg/dL)
PbFloor = floor dust-lead loading (μg/ft^2)
PbWindow = window sill dust-lead loading (μg/ft^2)
PbSoil = soil-lead concentration (μg/g)
Pntpica = 0 if no lead-based paint, paint in good condition, or no pica; 1 if lead-based paint present in fair/poor condition and no pica; 2 if lead-based paint present in fair/poor condition and pica;
ΔPr(IQ < 70) = change in risk of mental retardation, given 3.5 years exposure to lead
ΔPr(MI) = change in annual risk of MI associated with change in blood pressure
ΔPr(stroke) = change in annual risk of stroke associated with change in blood pressure
ΔPr(all-cause death) = change in annual risk of all-cause death associated with change in blood pressure
a = age of individual

$$\Delta BPS, \text{men} = 1.7*[\ln(PbB_{retained}) - \ln(PbB_{new})]$$
$$\Delta BPS, \text{women} = 1.2*[\ln(PbB_{retained}) - \ln(PbB_{new})]$$

$$\ln(PbB) = 0.65 + 0.032 \ln(PbFloor) + 0.050 \ln(PbWindow) + 0.094 \ln(PbSoil) + 0.256\, Pntpica$$

$$\Delta \Pr(IQ < 70) = \begin{cases} 0.000204(\text{PbB}) + 0.00360, & \text{PbB} \le 5.0 \\ 0.000488(\text{PbB}) + 0.00218, & 5.0 < \text{PbB} \le 7.5 \\ 0.001068(\text{PbB}) - 0.00217, & 7.5 < \text{PbB} \le 10.0 \\ 0.001044(\text{PbB}) - 0.00193, & 10.0 < \text{PbB} \le 12.5 \\ 0.000976(\text{PbB}) - 0.00108, & 12.5 < \text{PbB} \le 15.0 \end{cases}$$

$$\Pr(fatal\ MI \mid MI) = \begin{cases} 0.03, & a < 55 \\ 0.07, & 55 \le a \le 64 \\ 0.16, & 65 \le a \le 74 \\ 0.23, & a > 75 \end{cases}$$

$$\Delta \Pr(stroke,\ male) = (0.9948)\, e^{-5.2529 + 0.0505a + 0.014\, BPSnew} - (0.9948)\, e^{-5.2529 + 0.0505a + 0.014\, BPSretained}$$

$$\Delta \Pr(stroke,\ female) = (0.9977)\, e^{-7.1559 + 0.0657a + 0.0197\, BPSnew} - (0.9977)\, e^{-7.1559 + 0.0657a + 0.0197 BPSretained}$$

$$\Pr(fatal\ stroke \mid stroke) = \begin{cases} 0.26, & a < 65 \\ 0.24, & 65 \le a \le 74 \\ 0.32, & 75 \le a \le 84 \\ 0.48, & a \ge 85 \end{cases}$$

$$\Delta \Pr(cardiovascular\ death) = (1 + e^{-(-7.5414 + 0.0176\, BPS_{retained})})^{-1} - (1 + e^{-(-7.5414 + 0.0176\, BPS_{new})})^{-1}$$