

The Effect of Health Risk on Housing Values: Evidence from a Cancer Cluster

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There is a substantial literature that examines trade-offs between money and health risks. This literature has shown that estimates of marginal willingness-to-pay (MWTP) for changes in risk can be inferred from a wide variety of market situations. Much of the work has focused on mortality risks in the labor market (W. Kip Viscusi and Joseph E. Aldy, 2003), but substantial work has also looked for compensating differentials in the housing market. Significant negative effects on housing values have been found to be associated with hazardous waste sites (Ted Gayer et al., 2000), water pollution (Christopher G. Leggett and Nancy E. Bockstael, 2000), and air pollution (Kenneth Y. Chay and Michael Greenstone, 1998).

This literature has been primarily motivated by policy considerations. Policymakers have at their disposition many tools for reducing environmental health risks, including technology standards and incentive-based mechanisms, as well as water and air treatment facilities and hazardous waste remediation. The relative merits of diverse risk-reducing policies must be evaluated in terms of the value households put on risk. The efficient level of public spending for risk-reduction is reached when the sum of households' MWTP is equal to marginal cost.

Household MWTP for changes in environmental health risk is not directly observed in the market. If the level of risk varies across locations, however, and if households are mobile, then demand will be capitalized into property values. Standard assumptions about preferences

imply that houses in locations with high risks must have lower prices than equivalent houses in locations with low risks in order to attract households to these locations. These equalizing differences may be recovered by estimating a hedonic price function (Sherwin Rosen, 1974). The gradient of this function with respect to health risk is equal to household MWTP for an incremental change in risk.

In practice, hedonic price functions have proven difficult to estimate because the amenity of interest is typically not distributed randomly across locations. For example, locations with health risk due to air pollution tend also to be urban, industrial areas with particular labor market characteristics. When differences between locations are imperfectly measured and covary with health risk and housing prices, it becomes difficult to disentangle the price effects of health risks from the price effects of other locational amenities. The problem of omitted variables is compounded by an important sorting issue. Households move to locations endowed with amenities that match their preferences. When households near the amenity of interest are not representative of the population at large, it becomes difficult to interpret observed price differentials.

This paper measures the effect of health risk on housing values by exploiting a natural experiment that mitigates both econometric problems. The analysis focuses on an isolated county in Nevada where residents have recently experienced a severe increase in pediatric leukemia. Housing prices are compared before and after the increase with a nearby county acting as a control group. The variation in health risk over time makes it possible to control for unobserved differences across locations. In addition, because the leukemia cases were unanticipated there is no reason to expect sorting of households according to preferences prior to the increase. Finally, because many houses were sold repeatedly during the sample period it is possible to control for unobserved property-

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specific heterogeneity. The results provide a robust estimate of the MWTP to avoid pediatric leukemia risk. Housing prices in the affected county closely followed trends in housing prices for the control county and the rest of the state of Nevada during the period leading up to the leukemia increase. Then, beginning when eight children were diagnosed in 2000, housing prices in the affected county declined significantly. Least-squares estimates indicate that houses sold during the period of maximum risk sold for 15.6 percent less than equivalent houses not affected by the leukemia increase. Fixed effects estimates indicate a 14-percent differential. The estimated MWTP to avoid pediatric leukemia risk is used to calculate the value of a statistical case of pediatric leukemia.

I. Profile of a Cancer Cluster

Prior to 1997, Churchill County, Nevada (pop. 23,982) had no history of pediatric leukemia. Since 1997, 15 children have been diagnosed with acute lymphocytic leukemia and a sixteenth with acute myelogenous leukemia. A joint investigation by the Nevada Health Department and the U.S. Centers for Disease Control has been unable to determine the cause of the increase. No common characteristic has been identified among the case families and the cases have not been linked to occupational hazards, a certain neighborhood, or a particular water source.

Leukemia incidence of this magnitude far exceeds the population mean. The American Cancer Society estimates that 2,200 new cases of leukemia were found in children in 2003. A location with the population of Churchill County should expect to see one case of pediatric leukemia every five years. Accordingly, little attention was paid to Churchill County in 1997 or 1999 when one and two cases were confirmed, but since eight cases were diagnosed in 2000 and an additional four cases in 2001 the story has consistently made local and national news.

Rates of incidence of this magnitude are not without historical precedent. Similar clusters of pediatric leukemia have occurred in Maryvale, Arizona; Marion, Ohio; Toms River, New Jersey; and Woburn, Massachusetts. There is growing evidence in the medical literature that this clustering is widespread. E. Gilman et al. (1999), for example, find significant evidence

for clustering among the age 1–14 group using a dataset that includes 40 percent of England and Wales during the period 1984–1993. The term “cancer cluster” comes from the medical literature and is used to describe a geographic area, time period, or group of people with a greater-than-expected number of cancer cases.

The cause of leukemia is not known. Only chronic exposure to benzene, extraordinary doses of irradiation, and certain types of chemotherapy have been established as increasing the incidence of leukemia. Many environmental factors have been studied for possible association with leukemia, including petrochemicals, heavy metals, pesticides, volatile organic compounds, solvents, and consumer chemicals, but most researchers agree that definitive links with these factors have not been established.

One of the reasons epidemiologists have a difficult time identifying risk factors is that leukemia has a latency period. As a result, a child who is exposed to an environmental hazard may not become sick until many weeks, months, or even years later. This delay represents a potentially important feature of an economic analysis. Depending on the latency period, current leukemia incidence rates may or may not provide information about current health risk. These dynamics should be incorporated as clinical evidence increases our understanding of the latency period.

Initial publicity about the cluster may have led local children to be more likely to be tested for leukemia. Due to the pathology of leukemia, however, increased testing could not have affected the pattern of diagnosis. According to Martin D. Abeloff et al. (2000) the transition of acute leukemia to its active state occurs suddenly and is accompanied by the abrupt appearance of visible symptoms, so it is difficult for the disease to go undetected for an extended period of time. Also, clinical tests cannot detect leukemia before it is activated, so it is unlikely that increased frequency of testing by itself could have meaningfully accelerated incidence rates.

Pediatric leukemia is typically treated with two to three years of chemotherapy. A less common treatment for pediatric leukemia is bone-marrow transplantation, which is a difficult treatment that involves a lengthy hospital stay. Both treatments cause severe side-effects in the short term and long term. According to the American Cancer Society, five-year survival

rates for acute lymphocytic leukemia and acute myelogenous leukemia are 85 percent and 45 percent, respectively.

II. Location-Choice Model

Households are assumed to have identical preferences and choose where to live among a set of locations indexed by i . In each location there are S states of the world indexed by s . The probability of realizing state s in location i during period t is denoted π_{it}^s . All locations are endowed with equally attractive amenities and labor employment opportunities but different probabilities of realizing different states of the world. Aggregate consumption of all non-housing goods is denoted c_{it} and does not depend on the state of the world. Utility in period t is expressed in expected utility form by appealing to the expected utility theorem for state-dependent preferences.

Consider the case where there are two states of the world, "good" and "bad," which occur with probabilities $1 - \pi$ and π , where u^G and u^B are the utility functions associated with those states and $u^G(c) \geq u^B(c)$ for all values of c . Household utility in location i in period t may be expressed in the following form:

$$U(\pi_{it}, c_{it}) = (1 - \pi_{it})u^G(c_{it}) + \pi_{it}u^B(c_{it}).$$

The price of housing must equalize utility in all inhabited locations. In particular, houses in locations with high health risk must have lower prices than equivalent houses in locations with low risk in order to attract households to these locations. This compensating-differentials argument has a straightforward empirical interpretation. Controlling for all other determinants of house value, the difference in observed market price between two locations with different locational health risks is the compensating differential for risk. Following Rosen (1974), a house is described by a vector of its characteristics. In a competitive market the price-characteristic locus is determined by the equilibrium interactions of buyers and sellers. The gradient of the hedonic price function with respect to locational health risk is equal to household MWTP for an incremental change in risk.

When households are endowed with different levels of income, the implications of the frame-

work are less clear. Diminishing marginal utility of consumption implies that high-income households will require a larger compensating differential per unit risk. As a result, one expects to see high-income households moving out of dangerous locations. This sorting makes it difficult to interpret observed price differentials. The differential observed in the market will underestimate the MWTP of high-income households and overestimate the MWTP of low-income households. Other forms of household heterogeneity cause similar sorting issues.

In the next section, local health risk is estimated using local incidence rates. The state-dependent utility framework assumes that the level of risk (π_{it}) is objectively known. Estimates of health risk will take the place of π_{it} by appealing to standard subjective probability theory. Francis J. Anscombe and Robert J. Aumann (1963) showed that if preferences are state uniform ($\succeq_s = \succeq_{s'}$ for any s and s') then preferences may be expressed in expected utility form treating expectations as if they were objectively known. The measurements of health risk described in the next section are arbitrary ways of specifying these subjective probabilities.

III. Estimating Cancer Risk

Figure 1 shows four alternative measures of pediatric leukemia risk for the period January 1996 to September 2002 in Churchill County. The first measure is the cumulative number of leukemia cases. This measure is flat during the 1990s and then increases sharply in 2000 and 2001. The second measure is the cumulative number of newspaper articles in the Proquest newspaper database citing "leukemia" and "Churchill County" or "Fallon" (the county seat). This measure is flat until the middle of 2000 when the cluster began to receive media attention. The third measure is a linear spline that is zero through 1999, rises by $1/24$ each month during 2000 and 2001, and then takes the value of one. This quasi-dummy variable captures the basic pattern of the other measures and will be used for the main results.

The fourth measure of risk is generated using a Bayesian learning process. Suppose that households in location i draw health outcomes each period from a Bernoulli distribution with parameter π_i where, as above, π_i is the annual

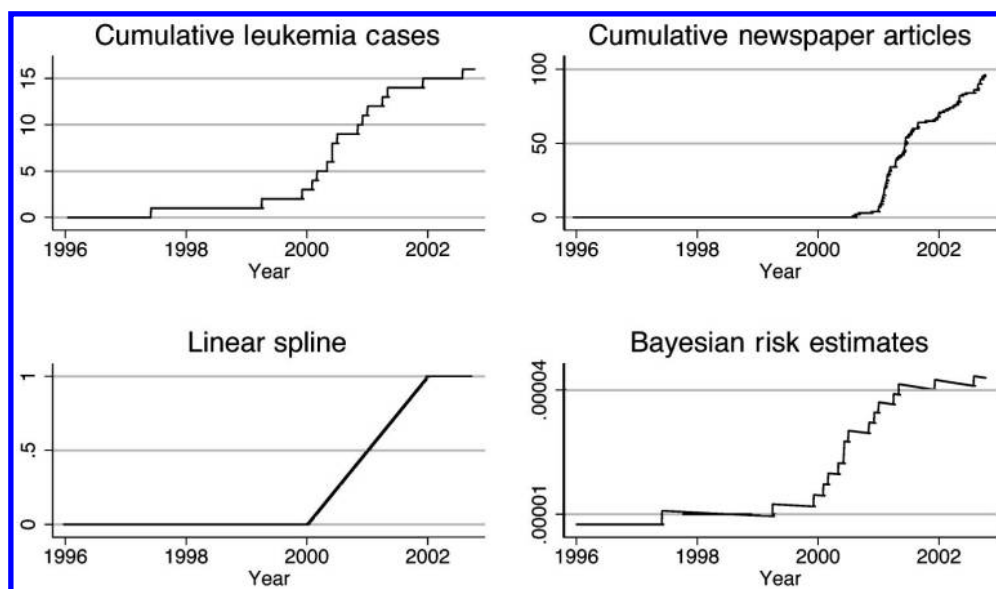


FIGURE 1. INCIDENCE RATES INCREASE AFTER 1999:
ALTERNATIVE MEASUREMENTS OF PEDIATRIC LEUKEMIA RISK FOR CHURCHILL COUNTY, NEVADA

probability of realizing the unfavorable state. Households do not know π_i . By observing draws from the distribution, however, they are able to make inference. Their beliefs about this true level of risk are described by a second distribution. Morris H. DeGroot (1970) derives a closed-form solution for the updating mechanism with a Beta distribution for beliefs. The mean of the beliefs distribution represents the perceived level of annual pediatric leukemia risk.

To derive the Bayesian estimates of risk, a prior distribution is assumed for January 1, 1997, and then updated daily using the diagnosis pattern observed in Churchill County. A natural candidate for the mean of the prior is the average national incidence rate for pediatric leukemia. It is important to consider, however, the possibility that the baseline level of risk for Churchill County could be different from that of the national mean. Because the county does not have any superfund sites or a history of extensive mining or heavy industry, households may have believed they faced lower risk than in other locations. Because links between the environment and leukemia are so poorly understood, however, it is difficult to know on what basis to compare locations. The choice of the variance of the prior is equivalent to choosing

the number of observed outcomes before 1997. The more outcomes the household observes, the lower the variance of the prior. For the standard prior, the mean is set equal to the average national incidence rate and the variance is constructed to reflect the cumulative population of Churchill County since 1970.

Two caveats are in order. First, all four measures of health risk ignore the possibility that household risk perceptions may depend upon leukemia rates in nearby locations. The next section describes an adjacent county that will be used as a control group in the estimation. The Nevada State Health Department reports that residents of the control county have not experienced increased pediatric leukemia rates. Their close proximity to the highly publicized cases in Churchill County, however, may have caused them to increase their own perceptions of risk. If this is the case, the estimated difference in risk between the two counties will be overstated. Second, several other forms of cancer, including adult leukemia, non-Hodgkin's lymphoma, and brain cancer, are widely believed to be linked to environmental factors. The Nevada State Health Registry has been analyzed and residents of Churchill County have not exhibited increased rates of incidence for these or any other form of cancer. Never-

theless, households in Churchill County may believe that whatever is responsible for the increase of pediatric leukemia has put them at increased risk for other health risks as well. If this is the case, the estimated difference in risk between the two counties will be understated.

IV. Housing Sales Records

The Churchill County assessor in Fallon, Nevada, and the Lyon County assessor in Yerington, Nevada, provided a record of all sales of single-family residences between 1990 and 2002.¹ These offices maintain a record of all private and commercial property sales within the boundaries of their respective counties. Lyon County was chosen to act as a control on the basis of median income and median house value. Lyon County lies immediately to the west of Churchill County. Table 1 provides a comparison of the two counties prior to the increased leukemia incidence which includes housing, demographic, and labor-market characteristics. For Lyon County to be a valid control group it must be unaffected by the cancer cluster. As mentioned in the previous section, however, even though their leukemia rates have not increased, residents of Lyon County may perceive higher leukemia risk because of their geographic proximity to the cases in Churchill County. The leukemia cases may also have affected Lyon County indirectly by causing households to move from Churchill County to Lyon County. Either of these effects could bias the estimated differential for risk.

One way to assess the validity of Lyon County as a control is to compare housing prices in Lyon County to housing prices in the rest of Nevada. Property-level sales records are not available for the entire state of Nevada. However, the Office of Federal Housing Enterprise Oversight (OFHEO) publishes the Conventional Mortgage Home Price Index (HPI) each quarter using mortgage transactions for single-family houses. The HPI is not available for Churchill County or Lyon County but the

property-level sales records make it possible to construct these indices, using the same generalized least squares first-differences methodology that the OFHEO uses to construct the HPI for the state of Nevada. See William Stephens et al. (1995) for a detailed description of the HPI methodology.

Figure 2 shows biannual house-price indices for Churchill County, Lyon County, and the state of Nevada. The indices reflect nominal sales prices for single-family residences with the average value for the period 1990–1999 normalized to 100 for each index. The dashed lines indicate a ninety-fifth percentile confidence interval around the index for Churchill County. Beginning in the first half of 2000 and continuing until the end of the sample period, the index for Churchill County is significantly below the indices for Lyon County and Nevada. Housing prices in Lyon County follow housing prices in Nevada during the period of the cancer cluster. This lends support to the use of Lyon County as a control. Figure 3 shows the percentage difference between the Churchill County HPI and the Nevada HPI. During the 1990s the Churchill County HPI fluctuates around the Nevada HPI. Beginning in 2000 the index for Churchill County diverges.

The basic contribution of this paper is to connect the increase in leukemia incidence with the decrease in housing prices. The framework described in the following section controls for unobserved time-invariant differences across locations and unobserved time effects. The analysis does not, however, rule out the possibility that county characteristics other than leukemia incidence may have changed simultaneously. In particular, a severe county-specific downturn in the labor market could provide an alternate explanation for the observed decline in housing prices. County-level employment data from the Bureau of Economic Analysis make it possible to evaluate this possibility. During the 1998–2001 period Churchill County employment levels decreased moderately in some sectors but total employment increased. The two hardest-hit sectors were agriculture, which went from 6.3 percent to 5.6 percent of total employment, and government, which went from 23.8 percent to 22.4 percent. County-level unemployment data from the Bureau of Labor Statistics reveal that from 1998 to 2002 annual unemployment rates in Churchill County were

¹ Three percent of sales were excluded from the analysis due to missing values or miscoding: construction year missing (208); sales price missing (76); interior floor space missing (32); multiple parcel sales (26); duplicate records (12); and date of sale miscoded (1). The number of excluded observations is shown in parentheses.

TABLE 1—COMPARING THE TREATMENT AND CONTROL COUNTIES

	Churchill (<i>n</i> = 2495)	Lyon (<i>n</i> = 3683)
Housing characteristics:		
Mean sales price	\$116,060 (52,791)	\$119,723 (55,060)
Mean lot size (acres)	1.42 (3.97)	1.16 (6.21)
Mean interior floor space (square feet)	1493 (461)	1480 (438)
Mean building age (years)	16.9 (20.8)	10.8 (15.6)
Mean class (range 1–5)	1.75 (.59)	2.16 (.76)
Demographic characteristics:		
Population	23,982	34,501
Persons per square mile	4.9	17.3
Percentage under 18	28.9	27.1
Percentage over 65	11.9	13.7
Percentage white	84.2	88.6
Percentage high-school graduates	85.1	81.5
Percentage college graduates	16.7	11.3
Homeownership rate	65.8	75.8
Percentage multi-unit	11.7	8.1
Percentage below poverty	8.7	10.4
Median household income	\$40,808	\$40,699
Labor market characteristics:		
Percentage employed in services	28.6	25.8
Percentage employed in government	23.8	12.1
Percentage employed in trade	18.6	18.0
Percentage employed in F.I.R.E.*	8.6	7.2
Percentage employed in agriculture	6.3	8.2
Percentage employed in construction	5.8	9.6
Percentage employed in manufacturing	5.6	14.1
Percentage employed in utilities	2.2	3.3
Percentage employed in mining	0.5	1.7
Percentage of labor force unemployed	6.2	7.4

Notes: The housing characteristics are for single-family residences sold during the 1990–1998 period. Standard deviations are in parentheses. Sales prices have been deflated to reflect year 2000 prices using the Nevada Real Estate Price Index. Demographic characteristics are from the 2000 Census. Percentage multi-unit refers to the percentage of housing units in multi-unit structures. Median household income is for 1999. Employment statistics come from the Bureau of Economic Analysis for 1998.

* F.I.R.E. includes finance, insurance, and real estate.

6.3 percent, 8.6 percent, 8.1 percent, 8.8 percent, and 6.4 percent. It is difficult to make definitive statements with the available data but there appears to be little evidence to support the explanation that the decline in housing prices is due to a labor market disturbance.

V. Estimation Strategy

Sales prices in logs are regressed on a vector of housing characteristics, X , and the linear spline, $RISK$, or some alternative measurement of local health risk:

$$PRICE_{jct} = \beta_1 X_{jct} + \beta_2 RISK_{ct} + \eta_{ct} + \varepsilon_{jct}.$$

Here j indexes individual houses, c indexes county, and t indexes time. Observable housing characteristics include lot size (acres), interior floor space (square feet [100s]), building age (years), and overall condition (class), as well as county, year, and month dummies. Class is a discrete variable (1–5) assigned by the assessor at the time of sale to reflect the overall condition of the property. The county dummy controls for county-specific fixed effects. The year dummies

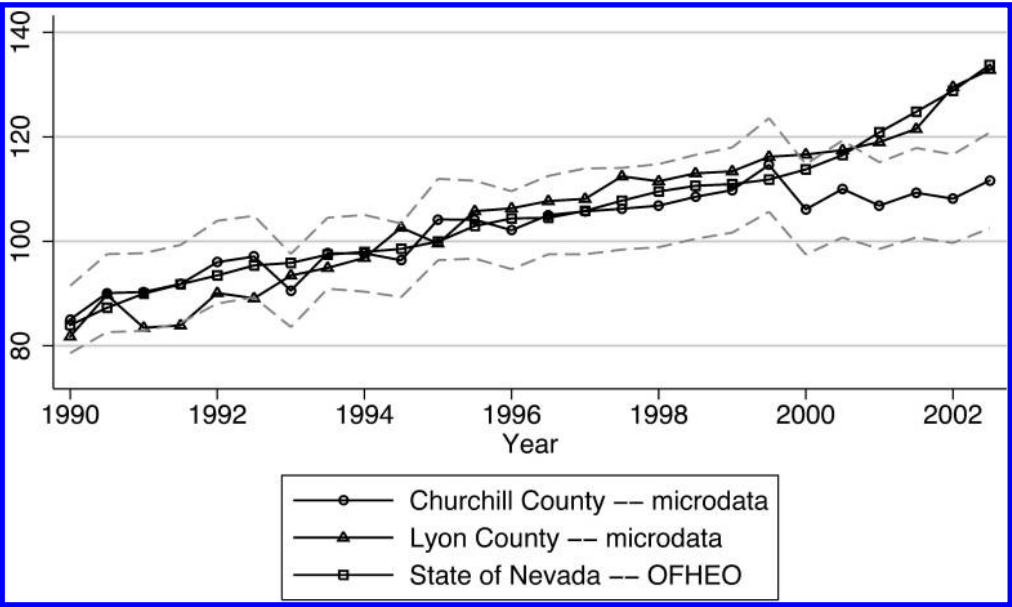


FIGURE 2. ASSESSING THE VALIDITY OF THE COUNTERFACTUAL:
HOUSE-PRICE INDICES FOR SINGLE-FAMILY RESIDENCES, 1990–2002

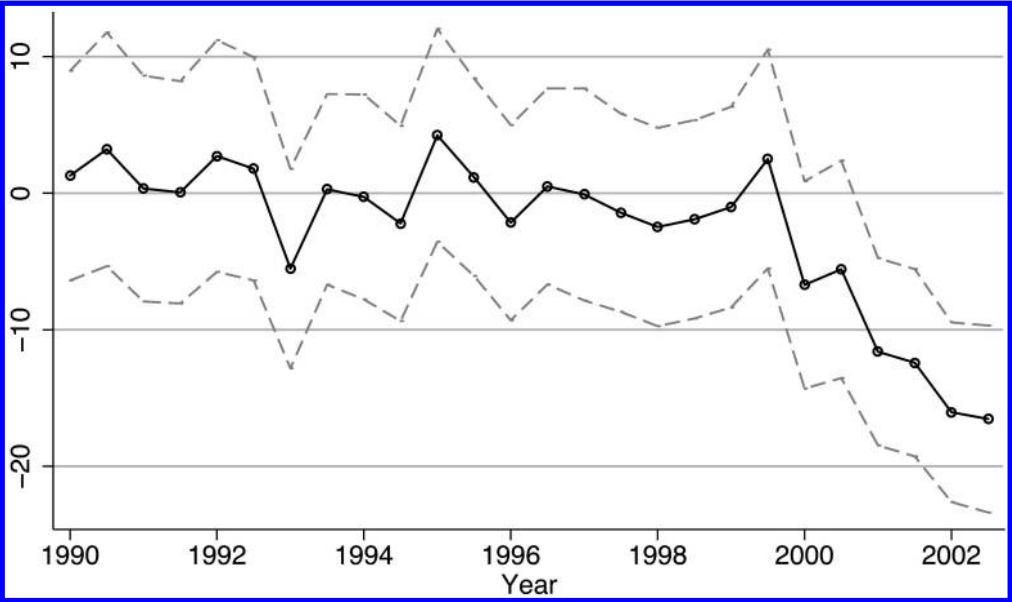


FIGURE 3. FALL IN HOUSING PRICES AFTER 1999:
PERCENTAGE DIFFERENCE BETWEEN CHURCHILL COUNTY HPI AND NEVADA HPI

control for unobserved state-level trends and the month dummies control for seasonal effects. The coefficient of interest, β_2 , is the percentage

effect of a unit change in *RISK* on property values. The variance matrix is estimated taking into

account that there are unobserved factors η_{ct} that cause prices to vary from month to month in each county. The correction procedure described by Brent R. Moulton (1986) allows each county-month group to have a different and unrestricted covariance structure but assumes that errors are uncorrelated across groups. Least-squares estimation is consistent if the explanatory variables are exogenous conditional on η_{ct} :

$$E\{\varepsilon_{jct}|X_{jct}, RISK_{ct}, \eta_{ct}\} = 0.$$

After including these covariates there remains large unexplained variation in house prices because many of the characteristics that determine the value of a house are unobserved. Much of the variation may be explained by unobserved factors that characterize particular properties like geographical features, neighborhood characteristics, and design amenities. The specification with a property-specific fixed effect, a_j , may be expressed as follows:

$$PRICE_{jct} = \beta_1 X_{jct} + \beta_2 RISK_{ct} + a_j + \eta_{ct} + \xi_{jct}.$$

An appealing feature of the housing sales records is that it is possible to link individual houses across years. By comparing the sales price of these houses at different points in time the fixed-effects estimator controls for property-specific heterogeneity and serves as an important test of the robustness of the results. The within transformation eliminates the property-specific fixed effect, a_j , along with all time-invariant regressors but does not eliminate the county-month effect, η_{ct} . Thus the variance-covariance matrix for the fixed effects estimates will also be corrected for intragroup correlation. Fixed effects estimation is consistent if the explanatory variables are strictly exogenous conditional on a_j and η_{ct} :

$$E\{\xi_{jct}|X_{jct}, RISK_{ct}, a_j, \eta_{ct}\} = 0 \quad \forall \tau.$$

VI. Results

Table 2 reports difference-in-difference estimates of the equalizing differential for risk.

TABLE 2—DIFFERENCE-IN-DIFFERENCE ESTIMATOR: MEAN LOG SALES PRICE BEFORE AND DURING LEUKEMIA INCREASE

	1990–1999	2000–2002	Difference
Churchill County	11.587 (0.408) $n = 2800$	11.550 (0.407) $n = 796$	−0.037
Lyon County	11.627 (0.403) $n = 4323$	11.667 (0.342) $n = 2285$	0.040
Relative difference			−0.077 (0.019)

Sales prices throughout have been deflated to reflect year 2000 prices using the Nevada HPI. Between the 1990–1999 period and the 2000–2002 period the mean log sales price of houses in Churchill County decreased by 0.037 while prices in Lyon County increased by 0.040. The period of increased perceived health risk is associated with a difference-in-difference of −0.077. The coefficient is significant at the 1-percent level. The difference-in-difference methodology provides a baseline estimate of the equalizing differential of interest but it does not control for changes in the composition of the housing stock. On average the houses sold in Churchill County between 2000 and 2002 had considerably larger lots and more floor space than houses sold previously. The regression-based estimates take this compositional change into account.

Table 3 reports least-squares and fixed-effects regression estimates for the linear spline. Specification (1) includes the coefficients for lot size, floor space, and building age. Coefficients for these characteristics are in the expected direction and comparable in magnitude to those found in Katherine A. Kiel and Katherine T. McClain (1995) and Janet E. Kohlhase (1991). The coefficient for the linear spline indicates that houses sold during the period of maximum risk in Churchill County sold for 12.3 percent less than equivalent houses not affected by the cluster. In specification (2) the differential increases to −15.6 percent after including county, class, time, and month dummies. The county dummy controls for county-specific amenities and indicates a time-invariant premium for Churchill County. The fixed effects estimate indicates a −14-percent differential. It would appear that controlling for observable characteristics in the pooled cross-section captures

TABLE 3—THE EFFECT OF HEALTH RISK ON HOUSING VALUES

	OLS (1)	OLS (2)	FE
Leukemia risk (linear spline)	−0.123 (0.013)	−0.156 (0.017)	−0.140 (0.015)
Lot size (acres)	0.011 (0.002)	0.012 (0.002)	—
Lot size squared	−1.88E-05 (3.20E-06)	−2.02E-05 (3.18E-06)	—
Floor space (square feet, 100s)	0.054 (0.001)	0.044 (0.001)	—
Building age (years)	−0.009 (0.001)	−0.006 (0.001)	—
Building age squared	3.57E-05 (8.61E-06)	1.20E-05 (8.42E-06)	—
Churchill County dummy	—	0.068 (0.009)	—
Class dummies	no	yes	—
Year dummies	no	yes	yes
Month dummies	no	yes	yes
<i>n</i>	10204	10204	4922
<i>R</i> ²	0.60	0.63	0.05

Notes: The sample consists of sales of single-family residences from 1990 to 2002 from both counties. The dependent variable is sales price in logs. The linear spline is zero through 1999, rises by 1/24 each month during 2000 and 2001, and then takes the value of one. For the control county the linear spline is equal to zero for all periods. Standard errors are corrected for heteroskedasticity and correlated errors within county-month groups.

much of the same property-specific heterogeneity controlled for in the panel.

For alternative measures of leukemia risk the results are of similar magnitude to those reported in Table 3. When the number of cumulative leukemia cases is used as the measurement of risk, the estimates indicate that the period of maximum risk is associated with a 15.6-percent (OLS) and 14.1-percent (FE) decrease in sales price. For the cumulative number of newspaper articles the differentials are 17.5 percent and 16.8 percent. For the Bayesian estimates of risk the differentials are 15.0 percent and 13.6 percent. These coefficients are of similar statistical significance as the coefficients described above for the linear spline, and the overall fit of the model does not appear to be better with one regressor over the others. Table 4 reports equalizing differentials for risk for small, medium, large, and very large houses. The coefficients are derived from eight separate regressions each including all of the observable characteristics described in Table 3. The observed differential is stable across segments of the housing market ranging from 14.1 percent to

TABLE 4—COMPARING DIFFERENTIALS FOR DIFFERENT-SIZED HOMES

	OLS	FE
Small homes	−0.147	−0.159
<1,250 square feet	(.043)	(.040)
	<i>n</i> = 2817	<i>n</i> = 1469
Medium homes	−0.165	−0.166
1,250–1,500 square feet	(.025)	(.026)
	<i>n</i> = 3010	<i>n</i> = 1496
Large homes	−0.153	−0.120
1,500–2,000 square feet	(.021)	(.020)
	<i>n</i> = 3147	<i>n</i> = 1371
Very large homes	−0.141	−0.126
>2,000 square feet	(.039)	(.047)
	<i>n</i> = 1230	<i>n</i> = 586

Notes: The OLS specification includes housing characteristics, class dummies, time dummies, monthly dummies, and a county dummy. The FE specification includes time and monthly dummies. The mean 1990–1999 sales prices for Churchill County for the four groups are \$84,600, \$109,000, \$135,900, and \$183,100, respectively. Standard errors are corrected for heteroskedasticity and correlated errors within county-month groups.

TABLE 5—LIFETIME ESTIMATES OF RISK AND THE STATISTICAL VALUE OF PEDIATRIC LEUKEMIA

	Standard Prior	Low Mean Prior	High Mean Prior	Low Variance Prior	High Variance Prior
Risk estimate 1997	2.59 (1.71)	1.29 (1.21)	5.18 (2.42)	2.59 (1.25)	2.59 (2.53)
Risk estimate 2002	14.5 (3.48)	13.6 (3.37)	16.4 (3.71)	9.82 (2.23)	22.6 (5.64)
VPL-least squares	\$5.55 (0.60)	\$5.39 (0.58)	\$5.88 (0.64)	\$9.20 (0.99)	\$3.28 (0.36)
VPL-fixed effects	\$5.00 (0.46)	\$4.88 (0.44)	\$5.26 (0.48)	\$8.29 (0.75)	\$2.97 (0.28)

Notes: The first two rows of the table report estimated lifetime pediatric leukemia risk per 10,000 individuals as of January 1, 1997, and January 1, 2002, with the standard deviation of the beliefs distribution in parenthesis. The second two rows report the value of a statistical case of pediatric leukemia (VPL) in millions of U.S. dollars (2000) with standard errors in parenthesis. The VPL estimates are derived from ten separate regressions. The OLS specification includes housing characteristics, class dummies, time dummies, monthly dummies, and a county dummy. The FE specification includes time and monthly dummies. Standard errors are corrected for heteroskedasticity and correlated errors within county-month groups.

16.5 percent (OLS) and from 12 percent to 16.6 percent (FE).

VII. Statistical Value of Pediatric Leukemia

This section uses the Bayesian estimates of risk to estimate the MWTP for change in lifetime pediatric leukemia risk. This tradeoff is used to calculate the value of a statistical case of pediatric leukemia (VPL). First articulated by Gary Fromm (1965) and later described by Thomas C. Schelling (1968) and Richard H. Thaler and Rosen (1975), the statistical value (or “inferred value”) of a health risk is the total amount of compensation a group would require to face one expected unfavorable outcome from within their group. The statistical value of a health risk is derived by dividing MWTP by the risk increment.²

² Inferred values would appear to provide a common metric for comparing human health valuations across contexts. However, it is important not to overstate the generality of this transformation. Several models including Michael Jones-Lee (1974) and Milton C. Weinstein et al. (1980) predict that MWTP for a reduction in health risk is increasing in the base level of risk. The gradient of the hedonic price function gives the MWTP evaluated at one particular level of risk. It does not provide information about how MWTP changes with the level of risk or about willingness-to-pay for non-marginal changes. In practice this implies that caution should be used in comparing estimates of MWTP across contexts with different base levels of risk.

The price of a house should capitalize the present discounted value of all future pediatric leukemia risk associated with living there. Accordingly, to calculate MWTP it is appropriate to use a measure of lifetime risk. The Bayesian estimates are used to derive lifetime risk by assuming that at every point in time the perceived level of risk in all future years is equal to the perceived current level of risk. Households are assumed to be infinitely lived and, following David M. Cutler and Elizabeth Richardson (1997), future risk is discounted at 3 percent annually. For a 5-percent discount rate the VPL estimates are larger by a factor of 1.63.

Table 5 reports the estimates of lifetime risk and the VPL. Using the standard prior, lifetime pediatric leukemia risk increases from 2.59 to 14.5 per 10,000 individuals between January 1, 1997, and January 1, 2002. This corresponds to a level of risk that rises from the national average to about six times the national average. To test the robustness of the results, lifetime pediatric risk is constructed for four alternative priors: (i) mean equal to one half the national average, (ii) mean equal to twice the national average, (iii) variance that reflects the cumulative county population since 1950, and (iv) variance that reflects the cumulative county population since 1990. Depending on the choice of prior, the estimated change in perceived lifetime risk ranges from 7.23 to 20 per 10,000 individuals. The estimates of risk are particu-

larly sensitive to the choice of the variance of the prior. The more outcomes the household observes, the lower the variance of the prior, and the smaller the effect individual leukemia cases have on perceptions of risk.

To generate the estimate of the VPL, this measure of lifetime pediatric leukemia risk is assigned to house sales by the date of sale and included in the price regression. The risk estimates for the control county are set equal to the January 1, 1997, prior. The price regression indicates that for the standard prior household MWTP is 1.22 (OLS) and 1.10 (FE) percent of house-sales price per 1 in 10,000 change in lifetime risk. The VPL estimates are calculated by multiplying the MWTP estimates by mean house price and dividing by the average number of members per household in Churchill County as reported in the 2000 Census (2.64). This second adjustment is necessary because the observed differential reflects household MWTP whereas the risk estimates are calculated per individual.

For the standard prior the least squares estimate indicates a VPL of \$5.6 million. The fixed effects estimate indicates a VPL of \$5 million. Point estimates for alternative priors range from \$3 million to \$9.2 million. These estimates are comparable in size to previous estimates in the literature for the inferred value of cancer and mortality risk. In the study most similar to this one, Gayer et al. (2000) derive value-of-cancer estimates ranging from \$4.3 million to \$5 million from the effect of local superfund sites on housing prices after the release of site risk assessments. This range is consistent with estimates of the statistical value of life measured in a broad range of contexts. See Viscusi and Aldy (2003) for a recent survey. Estimates from mortality risks in the labor market typically range from \$4 million to \$9 million. All estimates are expressed in year 2000 prices.

These estimates provide some of the first market-based estimates of the value of health for children. One of the reasons limited empirical evidence is available is because wage-risk studies are impossible for individuals who are not in the labor market. Considering the housing market effects of environmental health risks that predominantly affect particular age groups may be one approach for refining age-based measures of the value of health. The VPL estimates may be interpreted as the statistical value of the

life of a child only if housing decisions were made on the expectation that pediatric leukemia is terminal. More generally, value-of-cancer estimates reflect MWTP to avoid mortality risks and MWTP to avoid all other consequences of cancer. Both sources of valuation are important for assessing the cost-effectiveness of environmental regulations. Analyses that consider only mortality risks will underestimate the value of cancer risk reductions.

VIII. Concluding Remarks

In April of 2002, Senator Harry Reid (D-NV) announced that he had succeeded in securing nearly \$28 million in federal funds for public health projects in Churchill County. Together with Senator Hillary Clinton (D-NY), he has introduced the Health Tracking Act, which would create a national network for tracking chronic diseases with possible environmental causes. The estimates from this project provide part of the information necessary to assess the cost-effectiveness of such programs. Much work remains to be done on the marginal cost side of the equation. In particular, will this new spending allow epidemiologists to identify the source of the leukemia increase? What will have to be done to lower leukemia risk? How much will it cost per leukemia case avoided? Together with the answers to these difficult questions, the estimates from this project could be used to evaluate programs like the Health Tracking Act on the basis of efficiency. More generally, the estimates from the paper provide a benchmark for assessing the cost-effectiveness of a broad range of public policies that affect human health. In 2002 the budget for the Environmental Protection Agency included \$3.2 billion for safe drinking water, \$1.7 billion for waste management, and \$598 million for clean air. A primary motivation for this spending is to protect households from cancer-causing substances and other environmental health risks. Reliable estimates of household valuations of these risks are imperative if programs are to be funded at cost-effective levels.

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