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Paul Gallagher
Iowa State University, paulg@iastate.edu

William Lazarus
University of Minnesota - Twin Cities

Hosein Shapouri
United States Department of Agriculture

Roger Conway
United States Department of Agriculture

James Duffield
United States Department of Agriculture

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Paul Gallagher [a,*], William Lazarus [b], Hosein Shapouri [c], Roger Conway [c], James Duffield [c]

[a] Economics Department, 481 Heady Hall, Iowa State University, Ames, IA 50011, USA
[b] Applied Economics Department, 253 COB, University of Minnesota, St. Paul, MN 55455, USA
[c] Office of Energy Policy &New Uses, 400 Independence Avenue, SW (Rm. 4059 So. Bldg), United States Department of Agriculture, WA, DC 20250, USA

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ABSTRACT

The hypothesis of this study is that there is a statistical relationship between the lung cancer mortality rate and the intensity of fuel consumption (measured in gallons/square mile) at a particular location. We estimate cross-section regressions of the mortality rate due to lung cancer against the intensity of fuel consumption using local data for the entire US, before the US Clean Air Act (CAA) in 1974 and after the most recent policy revisions in 2004. The cancer rate improvement estimate suggests that up to 10 lung cancer deaths per 100,000 residents are avoided in the largest urban areas with highest fuel consumption per square mile. In New York City, for instance, the mortality reduction may be worth about $5.7 billion annually. Across the US, the estimated value of statistical life (VSL) benefit is $27.2 billion annually. There are likely three inseparable reasons that contributed importantly to this welfare improvement. First, the CAA regulations mandated reduction in specific carcinogenic chemicals or smog components. Second, technologies such as the catalytic converter (CC) and low-particulate diesel engine were adopted. Third, biofuels have had important roles, making the adoption of clean air technology possible and substituting for high emission fuels.

1. Introduction

Measurements of the health consequences of urban fuel consumption are central to evaluation of regulations, technologies and clean fuels that improve urban air quality. Presently, measurements combine known health effects with simulations of emissions, ambient air quality, and mortality risk estimates (US Environmental Protection Agency, 2007; European Commission). However, estimated health effects emphasize short-run response to specific atmospheric chemicals. Further, the incorporation of long-term effects of chronic and low-level exposure to air pollution is incomplete. Long-term effects of pollution on health are difficult to measure because the low level and chronic exposure must take place for several years before effects will occur. Further, potential long-term effects are easy for critics to discredit (Kitman, 2000).

Our estimate of the relation between an important health indicator, the lung cancer mortality rate, and a pollution variable, the intensity of fuel consumption at a particular location, provides a glimpse of the overall long-term effects of chronic exposure to air pollution. Optimistically, scientists will eventually understand the complex chemistry of pollutant emission and transformation in the environment, and the medical risks of chronic exposure to an array of urban air components. Until then, reduced form equations can estimate the composite relation between the final (endogenous) effects and initial (exogenous) causes (Greene, 2003, p. 379). Reduced form estimates can supplement an exhaustive understanding of individual cause- and effect relationships. Specifically, we estimate the total physical and social response to the technology improvements, product bans/substitutions, and economic policies associated with the US Clean Air Act (CAA) on lung cancer death risk—it is shown that the package of public actions had a substantial economic benefit.

Regarding organization, we first review the state of scientific understanding and uncertainty about air quality related determinants of health and cancer risk. Second, statistical estimates of the cross-section relationship between the lung cancer mortality rate and the intensity of fuel consumption are presented. Third, policy-related reductions in cancer mortality are calculated by comparing slopes of the fuel intensity regression, before the US Clean Air Act (1974) and after the most recent policy revisions in 2004. Next, the cancer rate improvement estimate is combined with value of statistical life estimates (VSL) from the literature for a direct statistical estimate of overall program gains. Lastly, allocation of the overall welfare gain to components is discussed.

2. Fuel consumption–health relationships for policy analysis: state of knowledge and uncertainty

Exceptional complexity arises because the fuel consumption–human health relationship has at least three dimensions. First, the

* Corresponding author. Tel.: +1 515 294 6181; fax: +1 515 294 0221.
E-mail address: paulg@iastate.edu (P. Gallagher).

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auto technology for burning fuel influences the composition and extent of chemical emissions into the atmosphere. And the nature of emissions changes over time with changing auto technology and regulation. Second, the reactive chemicals emitted from vehicles are transformed in the atmosphere, and sometimes the atmosphere itself is changed. Indeed, a separate branch of chemistry, atmospheric chemistry, has arisen in an attempt to understand the interactions between fuel-based emissions and the air we breathe. Third, science understands that air pollution adversely influences human health, but agreement on the mechanisms and effects is incomplete. The following statements illustrate that each of the components also have multiple dimensions:

"Combustion emissions and their contribution to ambient particulate, semivolitile, and gaseous air pollutants all contain organic compounds that induce toxicity, mutagenicity, genetic damage, oxidative damage, and inflammation (Lewtas, 2007, p. 27)".

Most of the medical literature on health risks from urban air pollution used in policy analysis focuses on short-run effects caused by specific chemicals uniquely present in urban areas. For instance, ozone's role in death from asthma, bronchitis, and emphysema has been verified and suggested for incorporation in future policy analysis (Bailar et al., 2008). Less extreme health problems from the same diseases are emphasized in existing benefit cost studies, but such studies frequently include a longer list of health reducing chemicals (sulfates, carbon monoxide, nitrogen oxides, sulfur dioxide, and lead). For example, (see US Environmental Protection Agency, 2007, p. D-6).

The long-run (cancer) risks from air pollution are difficult to measure, because the cancer effect is present only decades after exposure (Cohen, 2003, p. 1011), but also because air pollution is difficult to isolate as the sole cause. One important determinant of cancer risk, particulate air pollution, has been included as a criterion for designing appropriate policies for mitigating air pollution for policy analysis (Pope et al., 2002). Another long-term cohort study, not incorporated in policy analysis, suggests a relation between lung cancer and high concentrations of nitrogen oxide (Nafstad et al., 2003). However, the cancer risks of several other toxic substances in urban air are still undocumented (Nafstad et al., 2003). Urban air chemical–cancer-risk relationships are partially known, partially unknown.

The long-run (cancer rate) effect of policy and the induced technology changes should also be taken into account, because government policies are part of the fuel–health matrix. CAAs aimed at cleaner emissions have directly regulated the engine technology and fuel recipes for both gasoline and diesel engines. Indirectly, these policies have caused a substitution of polluting substances in favour of relatively clean additives. And fuel recipe regulations of the last 15 years have restricted several other toxic chemicals.

To curb the gasoline engine's pollution, the catalytic converter (CC) was introduced in 1973 to remove olefins (highly reactive compounds that promote smog formation) from auto exhaust (US Department of Energy, 2008). Switching to the CC generally reduces benzo(a)pyrene (BaP) emissions from 6.6 to 0.3 ug/km (Lewtas, 2007, p. 8; Ragsdale, 1994).[1] Hence, a reduction in the cancer rate is plausible because BaP is considered as a known animal carcinogen and probable human carcinogen by the International Agency for Research on Cancer (IARC) IARC (1983, p. 211), IARC (2009).

[1] However, exhaust benzene could increase if fuel aromatic content also increases.

Production of high-octane lead-substitute additives increased steadily with the introduction of the CC. Leaded gasoline was gradually banned because it damaged new cars equipped with the CC (Kitman, 2000). The lead ban was complete in 1995 (US Department of Energy, 2008, pp. 9 and 22). Initially, MTBE, benzene-rich reformate, and ethanol shared the new additive market, because they all had octane-boosting properties that were similar to lead. When the 1990 CAA took effect, though, the benzene in reformulated fuel was limited to 2.0% (US Dept of Energy, p.9). Recently, the benzene content of gasoline was limited to 0.62% in all gasoline (Octane Week, 2007a, b, p. 1). Also, MTBE was banned in several states and mostly removed from the national market in 2005 amidst concerns for ground water pollution. Gradually, ethanol substitutes have been removed from the lead-substitute market. In effect, the CC and ethanol are complementary inputs, used in fixed proportions, and jointly responsible for any potential cancer rate reductions over the last 20 years.

Particulate regulations for diesel were introduced after the 1990 CAA. Early estimates of yearly cancer deaths associated with particulate emissions extrapolated laboratory animal results—calculations ranged widely from about 70 to 4873 terminal cancer cases annually (Brodowicz et al., 1993, pp. 4–13). Later, standards specified cleaner diesel engines—a new heavy truck emitted 0.751 g/hp h of particulates before regulation, and gradually reduced to 0.1 g/hp hr for 1994 models (US Environmental Protection Agency, 1985, p. 10630). It takes a long time for actual particulate reductions, however, owing to the long useful life of a diesel truck.

Esther fuels from soybean or rapeseed oil also reduce particulate emissions. Experimental data suggest that 20% ester-blended diesel fuel only emits 85% of the particulates of #2 fuel oil (Manicom et al., 1993). Some esther-blend tests have shown an increase in nitrous oxide emissions. However, adjusted engines reduce all categories of pollutants in some tests (Goetz, 1993). Overall, improved diesel engines and ester fuel blends are substitute inputs for reducing particulate emissions.

Separately, the CAA regulations of 1990 and 2000 both specified reduction in smog-causing gasoline engine emissions that were achieved by regulating fuel composition. Toxic emission reductions included several known or potentially carcinogenic chemicals: benzene, 1,3 butadiene, aldehydes, napthalene, and polycyclic organic matter (Lewtas, 2007, pp. 4 and 8).

3. Estimation procedures

A cancer rate–fuel intensity relationship underlies our empirical analysis. In Fig. 1, the function f_i has a positive slope because residents of highly populated areas are exposed to higher concentrations of pollutants from fuel consumption than residents of small towns or rural areas. Further, f_i is hypothesized to be relatively flat (has a smaller slope) when strict fuel blending regulations, clean fuels that exclude carcinogenic substances, or modern clean-burning engines dominate the vehicle fleet. In contrast, f_i is hypothesized to be steeper before regulation, because older cars emitted more harmful exhaust pollutants, and fuel blending was not regulated for health benefits. Other factors may shift the position of f_i over time; examples of time-shifting variables include improving health care and deteriorating health habits such as smoking. Our estimation of health benefits consists of estimating f_i before the Clean Air Act in 1972, and after the CAA in 2004. Then the 'other health-determining factors' are adjusted to their 2004 values, and a before and after comparison of mortality rates is calculated.

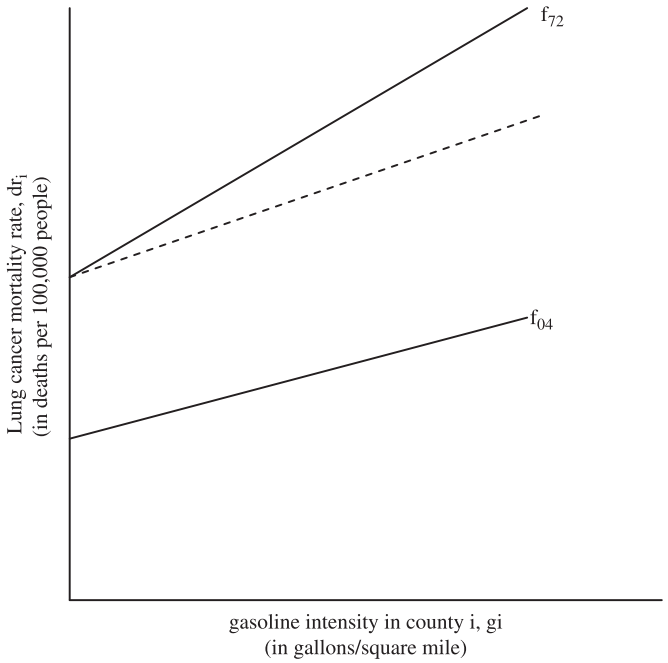

Fig. 1. Cancer rate–fuel intensity relationship.

We used the 'fixed time and group effects' model for cross-section-time series estimation (Greene, 2003, p. 291). Accordingly, the mortality rate is the dependent variable, and the intensity of fuel use is one explanatory variable. Additionally, a dummy variable for the observation's state and year are also included to capture the effects of other health-determining variables. The regression specification is:

$$dr_{it} = \sum\nolimits_t \alpha_t Dt_{it} + \sum\nolimits_i \alpha_i Ds_{it} + \beta_t gi_{it} + \varepsilon_{it}, \quad (1)$$

dr_{it} is the 'age-adjusted' mortality rate due to lung cancer, in deaths per 100,000 people; gi_{it} is the fuel (gasoline and diesel) use intensity, in gallons/square mile; Dt_{it} is 1 for year t (1972, 2004), and 0 otherwise; Ds_{it} is 1 for state s ($s =$ al, ar, etc.), and 0 otherwise; ε_{it} is the random variable; α_t, α_i, β_i are parameters for estimation

Eq. (1) defines 2 cross-section regressions, defined by $t = 72$ and 04. Also, there are $i = 1, \ldots, n = 263$ sub-state observations.

Initially, we expected to include explicit other health-determining factors as explanatory variables. Some state-level data on cigarette consumption and health expenditures were available for recent years, but not for the pre-CAA period of 1972. Further, local data were unavailable for both health variables in all time periods. Hence, we chose to proxy the state of health habits, and health care delivery at each time and location using the 'state' and 'time' variables. Bias in regression coefficients due an omitted explanatory variable, such as health habits, does not occur when the independent variables are uncorrelated (Judge et al., 1982, p. 597). Apparently, bias in β_t due to exclusion of cigarette consumption is unlikely—the correlation between fuel intensity and cigarette consumption was 0.0548 in 2004.[2]

The dependent variable in Eq. (1) removes the effect of changing age distribution. We used the 'age-adjusted' death rate due to lung cancer (malignant neoplasams of the bronchus and lung). The age-adjusted death rate for n age groups is:

$$\frac{d_t}{N_t^T} = \sum_{i=1}^{n} \frac{d_t^i}{N_t^i}\frac{N_0^i}{N_0^T}, \text{ where}$$

d_t^i deaths in age group i and year t

N_t^i population in age group $i$in year t

$d_t = \sum_{i=1}^{n} d_t^i$ total deaths across age groups in year t

$N_t^T = \sum_{i=1}^{n} N_t^i$ total population across age groups in year t

N_0^i population in age group i in base year 0 (2000)

N_0^T total population in base year 0 (2000).

Thus, the actual mortality rate within each age group in each county is weighted by a fixed age distribution proportion for a base year period. The 2000 age distribution of US population defines the fixed age distribution weights (National Center for Health Statistics, 2006, p. 479).

For national policy analysis, it is convenient that the standardized national death rate becomes the actual death rate in the base year. That is, $d_0/N_0^T = \sum_{i=1}^{n}(d_0^i/N_0^T)$ because $N_t{}^i = N_0^i$. Similarly for local data, the actual death rate is approximately equal to the standardized death rate when the area's age distribution is approximately equal to the national age distribution in the base year. Then the number of deaths is approximately equal to the current population times the age-adjusted death rate for the base year.

Estimation was executed on two cross-sectional regressions using the seemingly unrelated regression (SUR) procedure from The Statistical Analysis System (SAS) software package. Each equation had its own intercept term, which defined two α_t's. An explicit dummy variable takes a unit value, ($Ds_i = 1$) for each state that has one or more metropolitan counties and a rural 'rest-of-state' region.[3] Further, a particular state coefficient is constrained to be the same across both cross-sectional equations. The $Ds_i = 0$ situation refers to six rural states that did not have a metropolitan county in the 1972 reference data.[4]

4. Data

Individual death records data were compiled for our statistical analysis. The adjusted mortality rate data were constructed from individual records kept by the Center for Disease Control and made available (National Bureau of Economic Research, 2008). Individual records were available for 215 counties that were classified as metropolitan in 1972, which were all included in the analysis. Also, state-level data were available—so 48 'other state' observations were constructed by subtracting the appropriate metropolitan counties from state-level data.[5] In this fashion, the

[2] But more generally, policy inferences based on changes in the slope of the fuel consumption–health risk relationship are likely valid even in the presence of higher correlation between fuel intensity and other (omitted) health variables, provided that the correlation pattern among independent variables is similar before and after the policy change. See Appendix B for further discussion.

[3] These states are $i =$ al,ar,az,ca,co,ct,dc,de,fl,ga,ia,id,il,in,ks,la,ma,md,me,mi, mn,mo,ms,nc,ne,nh,nj,nm,nv,ny,oh,ok,or,pa,ri,sc,tn,tx,ut,va,wa,wi.

[4] These rural states are mt, nd, sd, vt, wv, and wy.

[5] For the dependent variable, the raw data, the number of cancer deaths by age group, was given at the state level and for each metropolitan county. The total number of deaths (by age group) for the rural "rest-of-state" region is the residual difference between the number of deaths in the state less the sum of deaths in the metro-counties. The population data by age group is also given at the state and metro-county level by age groups. So the residual population by age group in the rural rest-of-state region is the state population less the sum of population in the metro-counties. Next, the death rate by age group for the rural was calculated as the ratio of the number of deaths divided by the population for each age group. Finally, the "age-adjusted death rate" was calculated as a weighted average using weights from the national average age distribution. For the fuel intensity variable, we started with fuel consumption data at the state level and for the metro-

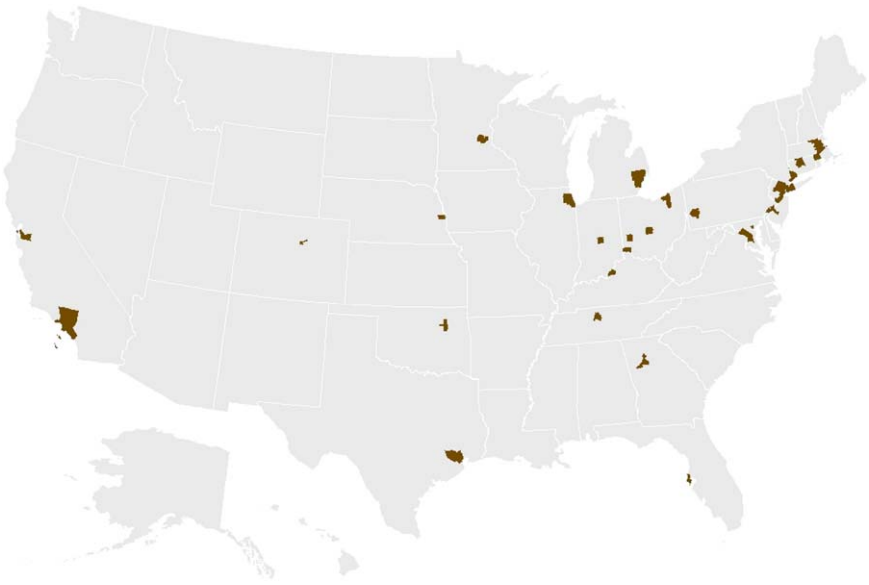

Fig. 2. Top 50 countries for fuel consumption intensity in 1972.

data set included rural areas with low levels of exposure and urban areas with high levels of exposure.[6] Together, there were 263 observations.

The gasoline intensity variable was also constructed. We used county-level data on vehicle miles travelled (VMT), which is collected jointly by the US Department of Transportation and the US Environmental Protection Agency (Driver et al., 2007). The VMT data were combined with fuel economy estimates for the appropriate year from the EPA's MOBILE6 model (e.g., Landman). Fuel consumption for each county was approximated by multiplying miles by fuel economy, and aggregating across vehicle classes. We matched 1978 VMT data with the pre-regulation cancer rate observation, because it was the earliest data available. Lastly, fuel consumption for each county or 'other state' observation was divided by the geographical area of the appropriate unit.

In 11 instances, the high mortality and high fuel counties are the same place. In 23 instances, a high fuel intensity county and a high cancer county are adjacent. Overall, spatial data for counties with emission problems and cancer problems are suggestive of a causal relationship.

5. Accounting for nearby counties and prevailing winds

We examined the hypothesis that high-level emission levels from one county could influence the mortality rate in a nearby county. To measure a nearby county effect, the 'nearby emission center (NEC)' for each county was selected from the 50 highest fuel consumption intensity counties. The top 50 counties are shown in Fig. 2. Generally, the NEC is the closest large (top 50) and 'upwind' county when both criteria could be met. Otherwise, the closest county was chosen. The gasoline intensity for the NEC was added to the gasoline intensity observed for a particular county, if the prevailing winds place the county of interest downwind of the NEC county. That is, the gasoline intensity for a particular county is given by

$$g = gi + d_d * D_p * gin.$$

The adjusted fuel intensity (g) becomes the independent variable in Eq. (1) regression. It includes the measured fuel intensity for the county (gi) plus a term for emissions from a nearby emission center (gin). Also, d_d is a decay function that accounts for the county's distance from the emission center. Also, D_p accounts for the direction of the prevailing wind.

The distance decay function is a geometric function that declines with distance. The function,

$$d_d = w^{(d_i/10)},$$

is defined by the distance between the county and the closest downwind emission center (d_i), in miles. The value, $w = 0.9$, was chosen initially because it has plausible values: $d_d = 1.0$ when $d_i = 0.0$, $d_d = 0.56$ when $d_i = 50.0$ miles, and $d_d = 0.2$ when $d_i = 150.0$ miles. The distance decay functions with alternative choices of the exponent are shown in Fig. 3.[7]

The critical summer prevailing winds come from the southwest in most of the continental US. But in the Atlantic Gulf region the prevailing winds originate in the northeast (Wallace and Hobbs, p. 17 and Encyclopedia of the Atmospheric Environment). For counties located in most areas then, the prevailing wind (pw) originated in the southwest (pw = sw). So when a county has a position (pos) northeast (ne), north (n), or east (e) of the nearest emission center, a unit value was assigned to D_p. Otherwise, D_p was assigned a value of 0. For counties in the gulf coast area of the US, the prevailing wind comes from the northeast (pw = ne). Then counties positioned southwest (sw), west (w), or south (s) of the

(*footnote continued*)
counties. So the "rural fuel consumption" was calculated as the difference between state consumption and the sum of metro-county consumption. Next, we obtained data on the physical area of each state and metro-counties. Then we calculated the area of the rural area as the difference between the state total and the sum of urban counties-area. Finally, fuel intensity for the rural area is rural fuel consumption divided by rural population.

[6] The 'other state' observations do extend the physical area of some observations, but not abruptly. Specifically, one-half of the 'other state' areas are smaller than the largest county in the sample. Further, one-fourth of the other state areas are no more than twice the size of the second largest county.

[7] An estimated value for this nonlinear parameter was obtained using a grid search for the value that minimized the sum of squares. The estimated value is $w = 0.765$.

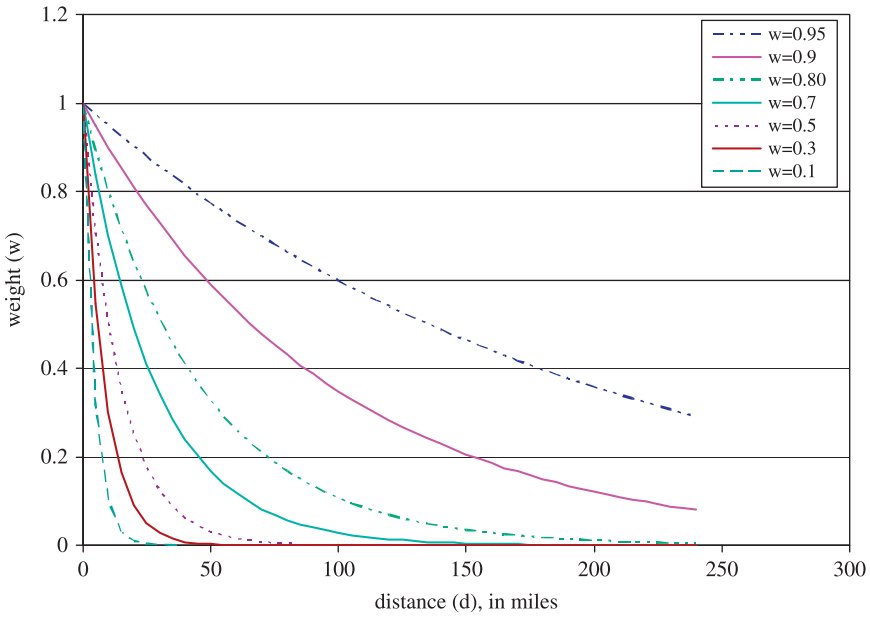

Fig. 3. Weight on nearest county's gasoline intensity: $W = w^{d/10}$.

Table 1
SUR estimate of lung cancer mortality function.

$$d_{it} = \underset{(23.71)}{30.718}Dt_{72} + \underset{(32.78)}{43.225}Dt_{04}$$
$$+\underset{(3.88)}{11.675}Dal_{it} + \underset{(3.47)}{14.140}Dar_{it} + \underset{(2.86)}{4.812}Dca_t - \underset{(-2.43)}{6.669}Dco_{it} + \underset{(1.85)}{5.085}Dct_t + \underset{(1.40)}{8.659}Ddc_t + \underset{(4.39)}{17.939}Dde_{it} + \underset{(8.12)}{14.722}Dfl_{it} + \underset{(4.23)}{11.660}Dga_{it} + \underset{(1.78)}{7.239}Dia_{it} + \underset{(4.06)}{8.655}Dil_{it} + \underset{(4.45)}{12.259}Din_{it}$$
$$+\underset{(1.76)}{5.994}Dks + \underset{(6.09)}{20.751}Dky_{it} + \underset{(6.84)}{18.802}Dla_{it} + \underset{(3.45)}{7.674}Dma + \underset{(4.14)}{10.697}Dmd_{it} + \underset{(2.85)}{11.617}Dme + \underset{(4.45)}{10.301}Dmi_{it} + \underset{(5.78)}{17.426}Dmo_{it} + \underset{(3.31)}{13.480}Dms_{it} + \underset{(3.16)}{8.089}Dnc_{it} + \underset{(1.43)}{4.875}Dne_{it} + \underset{(3.87)}{13.166}Dnh_t$$
$$+\underset{(3.73)}{7.285}Dnj_t - \underset{(-1.19)}{4.859}Dnm_t + \underset{(4.38)}{14.932}Dnv_t + \underset{(3.57)}{7.351}Dny_t + \underset{(6.71)}{13.911}Doh_{it} + \underset{(3.40)}{11.590}Dok_{itt} + \underset{(3.09)}{7.918}Dor_{it} + \underset{(3.83)}{7.204}Dpa_{it} + \underset{(2.13)}{8.696}Dri_{it} + \underset{(3.48)}{9.577}Dsc_{it} + \underset{(5.51)}{15.144}Dtn_{it} + \underset{(3.62)}{6.797}Dtx_{it}$$
$$-\underset{(-4.67)}{15.893}Dut_{it} + \underset{(4.94)}{14.862}Dva_{it} + \underset{(2.39)}{6.105}Dwa_{it} + \underset{(3.48)}{.00182}g_{i72} - \underset{(-.06)}{.00003}g_{i04}$$

(Rural states of reference)
(mt, nd, sd, vt, wv, wy)

States without statistically significant Ds_i;
i = az, id, mn, wi

States with statistically significant and positive Ds_i;
i = al, ar, de, fl, ga, il, in, ky, la, md, mi, mo, nc, nh, nj, nv, oh, ok, or, sc, tn, tx

States with statistically significant and negative Ds_i;
i = co, nm, ut

$S_{72} = 7.45$ adj.$R^2_{72} = .2914$
$S_{04} = 8.13$ adj.$R^2_{04} = .3605$

emission center received a value of unity. To summarize,

$$D_p = \begin{cases} 1.0, & \text{if pw} = \text{sw and pos} = \text{ne, n, or e, or if pw} = \text{ne and pos} = \text{sw, w or s.} \\ 0.0, & \text{otherwise.} \end{cases}$$

6. Estimates

Estimates for the mortality rate function were based on Eq. (1). But preliminary specifications were estimated for an evaluation of inclusion for specific state dummy variables. Initial estimates also suggested that both time dummies were significant and should be included. But there is a set of state effects that was not statistically significant—these variables were removed.

The estimated mortality response function is given in Table 1. t-Values for individual variables indicate statistically significant effects. Further, the reported set of explanatory variables explains about one-third of sample variation in the two sample years, which is typical for cross-sectional regressions.

Regarding the magnitude of estimated coefficients, the two time dummies suggest an increase in the mortality rate over time. Also, the state effects that are positive, zero, and negative define three groups of states (which are summarized in Table 1). The state with the largest positive effect is Kentucky, and the state with the lowest negative effect is Utah. Time and spatial variations in these effects can be attributed to changing the health care technology, health care delivery, and health habits in particular locations. Indeed, the pattern of state dummies with

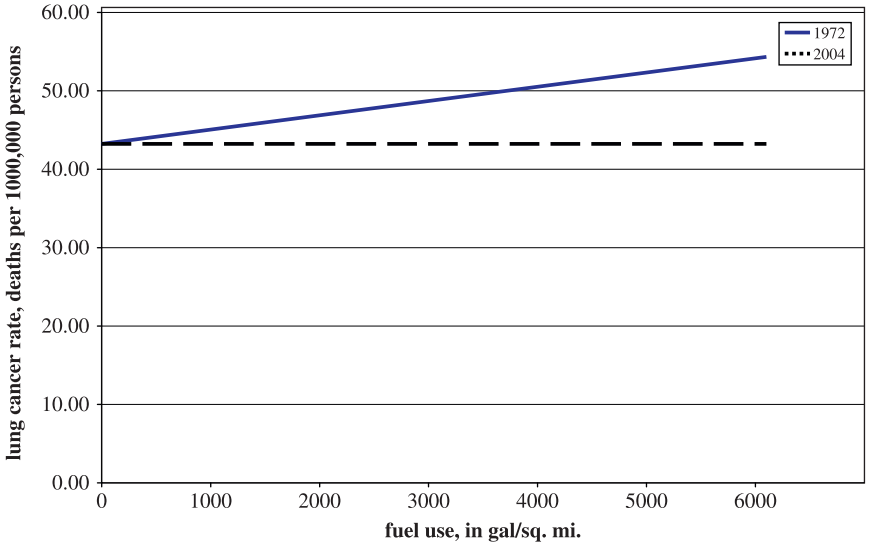

Fig. 4. Estimated cancer rate–fuel intensity relationship: 2004 basis.

large negative effects in several southern states conforms to preliminary estimates for the 2004 regression with available cigarette consumption data for 2004—cancer rates tended to be high in the tobacco producing states of the south (Kentucky, Tennessee, Alabama) and low in Utah.

The estimated response to the fuel intensity variable is important for policy analysis. As anticipated, the slope effect for the initial period is significant. Further, the fuel intensity for 2004 effect is smaller and not statistically significant.

7. Calculations of policy change effects

For an estimate of today's cancer risk without the CAA policies, use today's values for 'other health variables' with the 1972 estimate of the mortality response to fuel intensity. Thus, the level and position of today's response functions with the CAA and without the CAA are identified. These two response curves are shown in Fig. 4. The lower response curve is calculated using 2004 values of binary variables and the 2004 coefficient for fuel intensity. The upper response curve differs only in the use of the 1972 coefficient for fuel intensity response. The mortality gain from the CAA policies for a county with a given g is defined by the difference between the two response curves.

Further, there is a statistically significant difference between the post-policy and the pre-policy mortality rate function. When the 'pre-function' and the 'post-function' are compared on a 2004 basis with a given fuel intensity level, the statistic, $t = \Delta/sd(\Delta)$, has a t distribution with N–K degrees of freedom under the null hypothesis that the mortality function does not change with the CAA policy.[8] For the estimates of Table 1, $\Delta = -0.001822$, $sd(\Delta) = 0.000547$, and $t = -3.30$. Also, a normal approximation holds with our large sample. Hence, the null hypothesis, no change in the mortality function associated with the policy, is rejected at any reasonable significance level.

However, an ex-post-estimate of the mortality change from policy inception should probably take into account the change in fuel intensity over the period, as well as the shift in the mortality function. The estimate of mortality rate change in county i since the policy change is

$$\Delta_i = \hat{\beta}_{04} g_{i04} - \hat{\beta}_{72} g_{i72}$$

$$\text{Or } \Delta_i = (\hat{\beta}_{04} - \hat{\beta}_{72}) g_{i04} + \hat{\beta}_{72} \Delta g_i, \text{where } \Delta g_i = g_{i04} - g_{i72}.$$

Then the mortality change across all counties is

$$\Delta = \sum \Delta_i.$$

The estimates of mortality reduction in Table 2 do account for the change in the mortality function and the fuel intensity change. Then the death reduction is calculated as the mortality rate reduction times the 2004 population. In turn, the mortality rate reduction includes slope and fuel use changes. We estimate an annual death reduction of 1842 people annually for the largest 10 cities, a cumulative total 2305 people annually for the top 20 cities, and 3887 people for the entire US.[9]

For valuation, a value of statistical life (VSL) estimate of $7 million/person is used. This estimate is the median value for 30 US studies (Viscusi et al., 2005). In this fashion, we estimate that the combined reductions, technology advances, and subsidies provide an annual value of $27.2 billion for the entire US. Further, the 10 large cities reduce their annual loss by nearly $14 billion and the top 20 cities reduce their death loss by $18.2 billion, annually. That is, the value of reduced loss of human life declines by $27.2 billion throughout the US.

For comparison, the changing fuel intensity effect reduced the US VSL estimate by about 15%, from $30.5 billion to the $27.2 billion shown in Table 2. There were a few large cities in California with reduced fuel intensity between 1972 and 2004, possibly due to the development of mass transit. Otherwise, fuel intensity increased between 1972 and 2004 partially offsetting the mortality function decline.

[8] For demonstration, the mortality function estimate in the terminal period n is: $\hat{d}_{in} = \hat{\alpha}_n Dt_n + \hat{\alpha}_i Ds_{in} + \hat{\beta}_n g_{in}$.The mortality function estimate in the initial period 0 is: $\hat{d}_{i0} = \hat{\alpha}_0 Dt_0 + \hat{\alpha}_i Ds_{i0} + \beta_0 g_{i0}$. So the pre-policy mortality function in today's health situation is: $d^*_{i0} = \hat{\alpha}_n Dt_n + \hat{\alpha}_i Ds_{in} + \hat{\beta}_0 g_{i0}$. Taking the difference gives: $\Delta = d_{in} - d^*_{in} = \hat{\beta}_n g_{in} - \hat{\beta}_0 g_{i0}$. Assuming that fuel intensity does not change, gives: $\Delta = (\hat{\beta}_n - \hat{\beta}_0) g_i$. The statistic, $t = \Delta/sd(\Delta) = \hat{\beta}_n - \hat{\beta}_0 / \sqrt{\text{Var}(\hat{\beta}_n) + \text{Var}(\hat{\beta}_0) - 2\,\text{Cov}^1(\hat{\beta}_n, \hat{\beta}_0)}$ has a t distribution with N–K degrees of freedom (Kmenta, 1971, p. 372).

[9] Estimates of mortality reduction from CAA policies for all counties, sub-state rural areas, and the US are given in Appendix Table A.

Table 2
Mortality reductions, and lives saved by clean air act: total US, and countries with largest effects.

State	County seat	Fuel use	Population	Mortality rate reduction	Deaths avoided	
		Gallons/square mile	Number	Deaths/100,000	Number	million $
Top 10:						
CA	Oakland	2531	1452,096	−5.1	−75	−523
CA	Los Angeles	1255	9917,331	−2.0	−199	−1391
CA	Santa Ana	2137	2982,094	−2.6	−76	−535
IL	Chicago	1953	5327,165	−3.7	−195	−1370
MI	Detroit	2681	2013,771	−5.3	−108	−752
NJ	Jersey City	3342	605,359	−12.3	−75	−523
NY	New York	3393	8164,706	−9.9	−810	−5673
PA	Philadelphia	3875	1471,255	−10.1	−148	−1040
TX	Houston	1571	3641,114	−2.3	−84	−591
NJ	Newark	3401	795,015	−9.1	−72	−505
				Subtotal	−1842	−12,903
Top 20:						
DC	Washington	5148	554,239	−11.7	−65	−453
MD	Baltimore	3550	641,943	−7.6	−49	−341
PA	Doyles Town	2639	617,214	−6.0	−37	−259
Nj39	Elizabeth	3552	530,846	−6.6	−35	−247
NY	White Plains	3067	941,380	−6.9	−65	−457
NJ	Hackensack	2892	901,745	−4.8	−43	−302
Ma17	Cambridge	1376	1462,882	−2.2	33	−230
MA	Boston	2915	6664,263	−6.8	−45	−318
MI	Mt Clemens	2597	822,965	−6.7	−41	−286
NC	Rural	1195	6062,415	−0.8	−50	−346
				Subtotal	−463	−3239
Total, US					−3887	−27,207

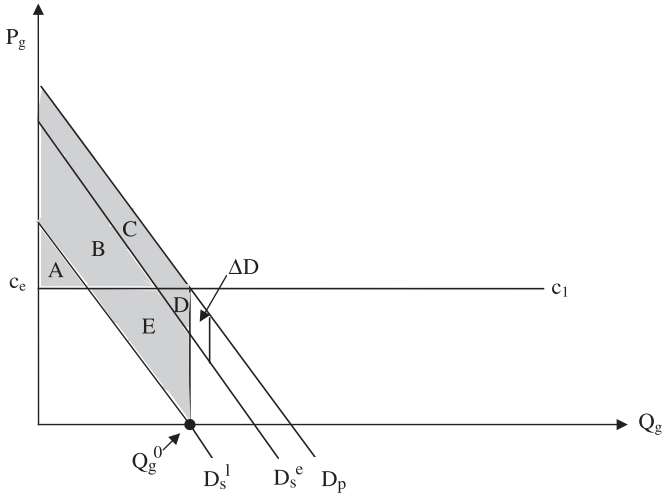

Fig. 5. Welfare analysis: ethanol gas or gas blended with lead.

8. Welfare interpretation of estimates

To welfare economics, the $27 billion estimate of annual cancer reduction benefit represents a market externality. Next we show how policy, technology, and product substitution reduced the size of the cancer risk and internalized the externality. To illustrate, consider the market for gasoline that is blended with high-octane gasoline additives.

In Fig. 5, the private demand for mixed gasoline is D_p. Initially, suppose the supply is defined by constant-cost production of leaded-fuel, C_l. In the initial baseline, the actual social demand curve, D_s^l, is below D_p due to the adverse cancer and other health effects of smog formed by using the lead additive to produce blended gasoline. Next, a policy change jointly requires the catalytic converter (CC), bans lead, and introduces ethanol. Then ethanol-blended gasoline's social demand curve, D_s^e, is slightly below private demand, D_p. For illustration, suppose that the ethanol blend has the same production cost as lead: $C_l = C_e$. Then gasoline (blended-fuel) output is the same, at Q_g^0, initially, and after joint CC introduction, lead ban, and ethanol development.

Now consider the welfare change. Before the policy change, consumer surplus is A+B+C, external cost is B+C+D+E, and net surplus level is A−(D+E). After the policy change, consumer surplus is still A+B+C. But external cost, C+D, is smaller. Consequently, net benefit level, A+B−D, is larger. Taking the difference between the final and initial welfare areas gives the overall net benefit change from the joint technology change, lead ban, and ethanol substitution: area B+E in Fig. 5.

The reduction in external cost that is jointly attributable to the CC, lead ban, and ethanol substitution (area B+E) is included in our previous estimate of the economic value of the cancer rate reduction. A parallel set of external cost reductions for introducing the clean diesel engine/biodiesel also exists.[10] They are also included in the $27.2 billion estimate of net benefits. Besides CC and diesel engine components of the externality reductions, conceptualization of the cancer benefit should include regulations that reduced benzene and other toxic chemicals in fuel, understanding that the position of D_s^e includes appropriate restrictions. However, the estimate here is limited to long-run cancer rate reductions. It excludes short-run health effects.[11]

[10] Fig. 5 also defines a parallel analysis of clean diesel engines when Q_g refers to diesel fuel consumption. Also, D_s^l, and D_s^e refer to the social benefit curve with old and new engines, respectively. The position of the demand curves is unique to the diesel analysis, but the net welfare gain is still A+B−D. Generally, an analysis of new engine, biodiesel or joint biodiesel-engine only requires a specific definition of D_s^e to represent the social demand with the appropriate new technology/fuel blending situation.

[11] For comparison, the welfare area, ΔD, measures the incremental environmental cost of another unit of blended-fuel with the today's technology and laws. Measurements based on this area typically calculate incremental increases in fuel consumption, emissions, and health.

9. Conclusions

Our analysis suggests that there is a statistical relation between lung cancer mortality rate and the intensity of fuel consumption in metropolitan and rural areas. Also, US Clean Air policies have reduced this dependence, by as much as 10 lung cancer deaths per 100,000 residents in the largest urban areas with high fuel consumption per square mile. The initial period estimated the cancer rate increase in intense fuel-using areas during 1972, the end of a long period without regulation–it is plausible that the early death rate increase reflects equilibrium differences in exposure to chronic and low-level air pollution. The final period estimate found little cancer rate increase with fuel intensity during 2004–it is plausible that pollution exposures are not substantially higher in high fuel use areas, given the new equilibrium exposures 33 years later.

The welfare value of the cancer mortality difference before and after clean air regulations is substantial. In New York City, for instance, the mortality reduction may be worth about $5.7 billion annually. Across the US, the benefit is $27 billion annually, when valuing the mortality reduction estimates with a typical VSL estimate. The mortality reduction benefit is somewhat smaller than a typical technology: regulation: health estimate would suggest; because increasing fuel consumption has offset some of the benefit. It is also plausible that EPA Pollution monitoring and advisory programs have helped mitigate the long-term health risk, as good health risk information enables people to avoid the outdoors when urban air pollution is at its worst.

The welfare change estimates are relevant to ex-post present value analysis that balances the stream of health benefits against public investment in the package of clean fuel technologies (the CC and low-particulate diesel engines), biofuel industry subsidies (ethanol and biodiesel), and regulatory bureaucracy. For ex ante analysis, the welfare estimates may be relevant to public investment for new clean fuel industries and clean car technologies.

Appendix

See (Table A1).

Table A1

State	County seat	Fuel use	Population number	Mortality change	Deaths avoided	
		gallons/square mile		Deaths per 100,000	Number	Value (million $)
AL	Birmingham	640	658,468	−1.0	−7	−47
AL	Huntsville	314	293,598	−0.5	−1	−10
AL	Mobile	198	400,107	−0.4	−2	−11
AR	Little Rock	579	365,228	−0.7	−2	−16
AZ	Phoenix	311	3,498,587	−0.3	−8	−55
AZ	Tucson	77	906,540	−0.1	−1	−6
CA	Oakland	2531	1,452,096	−5.1	−75	−523
CA	Visalia	67	400,952	−0.1	−1	−4
CA	Ventura	217	796,165	−0.3	−3	−18
CA	Martinez	937	1,007,606	−1.3	−13	−87
CA	Fresno	109	865,620	−0.2	−2	−10
CA	Bakersfield	148	734,077	−0.8	−6	−10
CA	Los Angeles	1255	9,917,331	−2.0	−199	−1391
CA	Salinas	98	414,551	−0.2	−1	−5
CA	Santa Ana	2137	2,982,094	−2.6	−76	−535
CA	Riverside	972	1,869,465	−1.1	−20	−143
CA	Sacramento	965	1,351,428	−1.1	−15	−107
CA	San Bernardino	337	1,916,418	−0.5	−10	−68
CA	San Diego	537	2,935,190	−0.7	−19	−133
CA	San Francisco	1744	743,193	−4.3	−32	−222
CA	Stockton	568	649,241	−0.9	−6	−42
CA	Redwood City	898	698,156	−1.2	−9	−60
CA	Santa Barbara	77	401,708	−0.1	0	−3
CA	San Jose	959	1,681,980	−1.4	−23	−160
CA	Santa Cruz	288	250,837	−0.6	−2	−11
CA	Fairfield	855	411,896	−1.5	−6	−44
CA	Santa Rosa	613	467,932	−1.4	−7	−47
CA	Modesto	446	497,599	−0.8	−4	−27
CO	Denver	3174	555,991	−3.9	−22	−152
CO	Co. Springs	179	557,752	−0.2	−1	−8
CO	Littleton	449	522,346	−0.5	−3	−17
CO	Fort Collins	665	268,960	−0.8	−2	−15
CT	Bridgeport	763	901,819	−1.8	−16	−114
CT	New London	344	266,107	−0.5	−1	−9
CT	Hartford	885	873,879	−1.5	−13	−88
CT	New Haven	1117	844,342	−2.1	−18	−123
DC	DC	5148	554,239	−11.7	−65	−454
DE	Wilmington	1444	518,728	−2.0	−11	−74
FL	Dade City	370	408,046	−0.5	−2	−14
FL	Clearwater	1407	927,498	−1.4	−13	−94
FL	Bartow	266	524,286	−0.3	−2	−11
FL	Fort Lauderdale	1029	1,753,000	−0.9	−17	−114
FL	Sarasota	797	355,722	−0.7	−3	−18
FL	Sanford	1150	391,241	−0.9	−3	−24
FL	De Land	341	478,951	−0.3	−2	−11

Table A1 (*continued*)

State	County seat	Fuel use	Population number	Mortality change	Deaths avoided	
		gallons/square mile		Deaths per 100,000	Number	Value (million $)
FL	Jacksonville	1023	819,623	−1.1	−9	−62
FL	Pensacola	401	296,739	−0.4	−1	−9
FL	Tampa	774	1,100,333	−1.0	−11	−77
FL	Fort Meyers	457	514,923	−0.4	−2	−14
FL	Bradenton	285	295,974	−0.3	−1	−5
FL	Ocala	221	291,768	−0.2	−1	−3
FL	Titusville	352	518,812	−0.3	−2	−11
FL	Orlando	901	989,873	−0.8	−8	−54
FL	W. Palm Beach	403	1,244,189	−0.3	−4	−30
GA	Atlanta	2433	905,802	−1.5	−14	−95
GA	Lawrenceville	1593	700,577	−0.7	−5	−35
GA	Marietta	3591	654,649	−2.1	−14	−97
GA	Decatur	5173	674,335	−3.6	−24	−170
IA	Des Moines	678	394,031	−0.7	−3	−20
ID	Boise	240	332,545	−0.3	−1	−6
IL	Woodstock	318	296,260	−0.4	−1	−8
IL	Edwardsville	1662	263,443	−0.6	−2	−12
IL	Belleville	2056	259,123	−0.8	−2	−14
IL	Joliet	492	617,494	−0.6	−4	−25
IL	Rockford	446	286,283	−0.7	−2	−13
IL	Chicago	1953	5,327,165	−3.7	−196	−1370
IL	Wheaton	2364	928,126	−2.3	−22	−150
IL	Geneva	611	472,761	−0.2	−1	−8
IL	Waukegan	1104	692,869	−1.7	−12	−83
IN	South Bend	588	265,718	−1.2	−3	−22
IN	Fort Wayne	511	341,816	−0.9	−3	−21
IN	Crown Point	634	490,089	−1.4	−7	−47
IN	Indianapolis	1657	861,847	−3.0	−26	−182
KS	Wichita	318	463,383	−0.5	−2	−16
KS	Olathe	901	496,892	−1.0	−5	−36
KY	Eddyville	2019	698,903	−2.7	−19	−134
KY	Lexington	699	266,451	−1.0	−3	−18
LA	Shreveport	260	250,893	−0.3	−1	−6
LA	Baton Rouge	680	411,564	−0.8	−4	−24
LA	Gretna	935	453,089	−1.6	−7	−50
LA	New Orleans	710	461,115	−1.2	−6	−40
MA	Hambden	498	461,491	−1.0	−5	−32
MA	Cambridge	1377	1,462,822	−2.3	−33	−230
MA	Dedham	1889	653,621	−1.9	−12	−84
MA	Plymouth	204	489,979	−0.6	−3	−19
MA	Boston	2916	664,263	−6.9	−45	−318
MA	Worcester	452	778,608	−0.6	−5	−34
MA	Taunton	342	547,278	−0.9	−5	−33
MA	Newburyport	984	737,447	−2.0	−15	−104
MD	Annapolis	786	508,356	−0.7	−4	−25
MD	Rockville	1250	921,631	−2.0	−18	−126
MD	Upper Marlboro	1436	841,642	−2.3	−19	−133
MD	Bristol	984	781,171	−1.1	−8	−57
MD	Baltimore	3550	641,943	−7.6	−49	−341
ME	Portland	231	273,622	−0.3	−1	−6
MI	Pontiac	1359	1,212,181	−1.8	−22	−152
MI	Ann Arbor	526	338,782	−0.6	−2	−14
MI	Detroit	2681	2,013,771	−5.3	−107	−752
MI	Flint	760	443,497	−1.2	−5	−38
MI	Mason	436	280,093	−0.7	−2	−14
MI	Grand Rapids	649	592,999	−0.8	−5	−32
MI	Mount Clemens	2597	822,965	−5.0	−41	−286
MN	Saint Paul	2547	499,206	−3.8	−19	−132
MN	Anoka	1841	319,548	−2.6	−8	−57
MN	Hastings	527	378,343	−0.5	−2	−13
MN	Minneapolis	1599	1,119,866	−2.2	−24	−170
MO	Saint Charles	497	320,459	−0.2	−1	−5
MO	Saint Louis	2499	1,007,723	−0.2	−2	−16
MO	Kansas City	727	662,185	−0.4	−3	−19
MS	Jackson	357	249,828	−0.3	−1	−6
NC	Charlotte	1019	771,573	−1.3	−10	−68
NC	Raleigh	771	719,733	−0.6	−4	−31
NC	Fayetteville	401	306,943	−0.6	−2	−12
NC	Winston-Salem	853	320,780	−1.2	−4	−26
NC	Greensboro	851	437,879	−1.0	−4	−29
NE	Lincoln	382	261,742	−0.3	−1	−6
NE	Omaha	900	481,203	−1.5	−8	−50
NH	Nashua	1419	398,355	−3.0	−12	−84
NH	Concord	409	292,346	−0.4	−1	−9

Table A1 (*continued*)

State	County seat	Fuel use	Population number	Mortality change	Deaths avoided	
		gallons/square mile		Deaths per 100,000	Number	Value (million $)
NJ	Mays Landing	364	268,311	−0.6	−2	−10
NJ	Newark	3401	795,015	−9.1	−72	−506
NJ	Woodbury	668	272,784	−1.2	−3	−23
NJ	Jersey City	3342	605,359	−12.3	−75	−523
NJ	Trenton	1013	364,381	−2.4	−9	−62
NJ	New Brunswick	2065	783,665	−2.7	−21	−148
NJ	Freehold	740	635,062	−1.2	−8	−53
NJ	Morristown	898	487,437	−1.4	−7	−49
NJ	Toms River	290	553,093	−0.7	−4	−27
NJ	Hackensack	2892	901,745	−4.8	−43	−303
NJ	Paterson	1333	498,939	−3.3	−17	−116
NJ	Somerville	2214	316,223	−3.2	−10	−70
NJ	Elizabeth	3552	530,846	−6.7	−35	−247
NJ	Mount Holly	514	448,656	−0.8	−4	−26
NJ	Camden	1759	515,620	−3.6	−19	−131
NM	Albuquerque	455	592,538	−0.6	−3	−23
NV	Las Vegas	97	1,648,524	−0.1	−1	−8
NV	Reno	48	380,612	−0.1	−0	−2
NY	Albany	660	297,910	−0.9	−3	−19
NY	Riverhead	1112	1,474,519	−1.7	−25	−171
NY	White Plains	3067	941,380	−6.9	−65	−457
NY	Poughkeepsie	766	293,322	−1.9	−6	−38
NY	Buffalo	652	935,946	−1.0	−10	−67
NY	Rochester	470	735,816	−0.7	−5	−34
NY	Mineola	2176	1,337,693	−2.0	−26	−185
NY	Syracuse	537	458,870	−0.9	−4	−28
NY	Goshen	1170	369,511	−3.0	−11	−77
NY	New City	2117	293,049	−2.5	−7	−52
NY	New York	3393	8,164,706	−9.9	−810	−5673
OH	Dayton	1014	549,553	−1.8	−10	−67
OH	Waverly	486	380,545	−1.1	−4	−30
OH	Akron	1009	546,608	−1.8	−10	−71
OH	Hamilton	1470	346,123	−2.2	−8	−52
OH	Cleveland	751	1,349,047	−1.8	−24	−166
OH	Columbus	1685	1,087,462	−2.1	−23	−163
OH	Cincinnati	1660	813,639	−3.2	−26	−182
OH	Elyria	289	293,532	−0.1	0	−3
OH	Toledo	675	450,304	−1.1	−5	−35
OH	Youngstown	559	255,995	−1.1	−3	−19
OK	Oklahoma City	1013	679,498	−1.4	−10	−68
OK	Tulsa	1086	568,611	−1.5	−9	−60
OR	Eugene	53	331,567	−0.1	0	−2
OR	Salem	214	301,702	−0.3	−1	−6
OR	Oregon City	144	362,681	−0.3	−1	−7
OR	Portland	1184	671,363	−1.4	−10	−67
OR	Hillsboro	405	487,548	−0.5	−3	−18
PA	Philadelphia	3875	1,471,255	−10.1	−149	−1041
PA	Reading	343	391,447	−0.6	−2	−17
PA	Greensburg	312	367,937	−0.6	−2	−16
PA	York	1214	401,063	−2.6	−10	−73
PA	Doylestown	2640	617,214	−6.0	−37	−259
PA	West Chester	498	466,043	−0.7	−3	−22
PA	Pittsburgh	1142	1,247,512	−2.5	−31	−219
PA	Harrisburg	990	253,060	−1.7	−4	−31
PA	Media	1785	554,426	−2.6	−15	−102
PA	Erie	196	280,844	−0.4	−1	−7
PA	Lancaster	1009	486,361	−2.0	−10	−67
PA	Allentown	848	325,570	−1.2	−4	−28
PA	Wilkes-Barre	294	313,088	−0.5	−2	−12
PA	Norristown	1311	773,375	−1.4	−11	−77
PA	Easton	1392	283,333	−3.2	−9	−63
RI	Providence	913	641,874	−1.7	−11	−76
SC	Charleston	237	327,403	−0.3	−1	−6
SC	Greenville	488	401,019	−0.6	−2	−16
SC	Columbia	431	335,597	−0.5	−2	−13
SC	Spartanburg	376	264,106	−0.5	−1	−10
TN	Memphis	1087	906,287	−1.4	−13	−90
TN	Nashville	1825	571,948	−1.5	−9	−61
TN	Chattanooga	582	309,729	−0.8	−3	−18
TN	Knoxville	1035	400,340	−0.8	−3	−18
TX	Dallas	2190	2,291,071	−0.9	−4	−26
TX	Denton	401	530,982	−0.1	−3	−18
TX	El Paso	395	712,617	−0.4	−2	−15
TX	Richmond	1004	442,389	−0.6	−4	−28

Table A1 (*continued*)

State	County seat	Fuel use	Population number	Mortality change	Deaths avoided	
		gallons/square mile		Deaths per 100,000	Number	Value (million $)
TX	Galveston	201	272,024	−1.4	−6	−44
TX	Houston	1571	3,641,114	−2.3	−85	−591
TX	Edinburg	240	657,310	−0.4	−2	−16
TX	Beaumont	215	248,308	−0.4	−1	−6
TX	San Antonio	902	1,492,361	−1.1	−17	−119
TX	Corpus Christi	285	317,317	−0.4	−1	−9
TX	Fort Worth	1364	158,7019	−0.2	−3	−24
TX	Austin	686	868,873	−0.7	−6	−40
TX	Brownsville	168	370,829	−0.2	−1	−6
TX	McKinney	452	628,426	−0.2	−2	−10
UT	Salt Lake City	788	934,838	−1.0	−9	−64
UT	Provo	161	434,114	−0.1	−1	−4
VA	Chesterfield	603	282,470	−0.7	−2	−13
VA	Virginia Beach	473	439,224	−0.7	−3	−23
VA	Richmond	1046	275,962	−1.2	−3	−22
WA	Vancouver	1330	392,364	−1.6	−6	−45
WA	Seattle	684	177,7746	−0.9	−16	−109
WA	Tacoma	345	745,778	−0.4	−3	−23
WA	Everett	225	644,205	−0.3	−2	−13
WA	Spokane	190	435,146	−0.3	−1	−10
WI	Menomonie	979	376,476	−1.6	−6	−41
WI	Madison	322	453,051	−0.4	−2	−13
WI	Milwaukee	636	926,764	−1.3	−12	−83
AL	Rural	78	3,173,202	−0.1	−3	−23
AR	Rural	47	2,384,772	−0.1	−2	−11
AZ	Rural	20	1,334,752	0.0	0	−2
CA	Rural	34	3,396,503	−0.1	−2	−16
CO	Rural	24	2,654,316	0.0	−1	−6
CT	Rural	220	612,819	−0.3	−2	−14
DE	Rural	688	311,341	−1.4	−4	−31
FL	Rural	105	4,115,738	−0.2	−6	−44
GA	Rural	116	5,982,766	−0.2	−9	−61
IA	Rural	48	2,558,873	−0.1	−2	−14
ID	Rural	14	1,062,595	0.0	0	−1
IL	Rural	291	3,568,492	−0.1	−5	−35
IN	Rural	144	4,267,067	−0.2	−9	−61
KS	Rural	23	1,773,422	0.0	−1	−5
KY	Rural	90	3,176,481	−0.1	−4	−30
LA	Rural	60	2,930,024	−0.1	−2	−16
MA	Rural	430	611,873	−0.6	−4	−27
MD	Rural	179	1,866,589	−0.3	−5	−34
ME	Rural	104	1,041,363	−0.2	−2	−16
MI	Rural	50	4,399,918	−0.1	−4	−27
MN	Rural	57	2,779,583	−0.1	−2	−16
MO	Rural	65	3,769,165	−0.1	−4	−30
MS	Rural	64	2,650,940	−0.1	−2	−15
MT	Rural	7	926,920	0.0	0	−1
NC	Rural	1195	6,062,415	−0.8	−50	−347
ND	Rural	10	636,308	0.0	0	−1
NE	Rural	17	1,004,759	0.0	0	−2
NH	Rural	105	608,468	−0.2	−1	−8
NJ	Rural	773	708,030	−2.0	−14	−97
NM	Rural	15	1,310,468	0.0	0	−2
NV	Rural	6	303,762	0.0	0	0
NY	Rural	96	12,142,711	−0.2	−29	−203
OH	Rural	128	5,377,335	−0.2	−12	−84
OK	Rural	94	2,275,437	−0.1	−2	−11
OR	Rural	16	1,436,502	0.0	0	−3
PA	Rural	168	4,161,943	−0.3	−14	−98
RI	Rural	266	438,042	−0.7	−3	−21
SC	Rural	122	2,869,767	−0.2	−4	−31
SD	Rural	11	770,621	0.0	0	−1
TN	Rural	105	3,704,994	−0.1	−5	−35
TX	Rural	39	8,299,183	−0.1	−5	−35
UT	Rural	15	1,051,756	0.0	0	−1
VA	Rural	156	6,483,676	−0.2	−13	−94
VT	Rural	84	621,233	−0.2	−1	−7
WA	Rural	32	2,211,807	0.0	−1	−7
WI	Rural	84	3,747,242	−0.1	−5	−32
WV	Rural	76	1,812,548	−0.1	−2	−15
WY	Rural	11	505,887	0.0	−1	−1
				Total	−3887	−27,207

Appendix B

To illustrate the claim of footnote 2, consider a two-variable regression model. The dependent is variable y_i (cancer risk) and the independent variables are x_{1i} (say fuel intensity) and x_{2i} (say smoking), where variables are expressed in mean deviation form. Variables in the post-policy period are identified by the superscript k. Variables in the pre-policy period are identified by the superscript 0. We look at the case where the response to fuel intensity changes between periods, but response to health variables is the same in both periods.

The regression model in the post-policy period is:

$$y_i^k = \beta_1^k x_{1i}^k + \beta_2 x_{2i}^k + \varepsilon_i,$$

where ε is a random variable with zero population mean $E(\varepsilon_i) = 0$. All variables are expressed in mean deviation form.

If x_2 is excluded from the period k regression, the least squares estimator for fuel intensity response is:

$$\hat{\beta}_1^k = \frac{\Sigma x_{1i}^k y_i^k}{\Sigma (x_{1i}^k)^2} = \beta_1^k + \hat{\gamma}_{21}^k \beta_2 + \frac{\Sigma x_{1i}^k \varepsilon_i}{\Sigma (x_{1i}^k)^2},$$

where $\hat{\gamma}_{21}^k = \Sigma x_{1i}^k x_{2i}^k / \Sigma (x_{1i}^k)^2$ is the least squares estimator from a regression between the two independent variables in period k: ${x_{2i}}^k = \gamma_{21}^k {x_{1i}}^k + \eta_i$

Similarly, the least squares estimator for the initial, pre-policy, period 0 is:

$$\hat{\beta}_1^0 = \beta_1^0 + \hat{\gamma}_{21}^0 \beta_2 + \frac{\sum_i x_{1i}^0 \varepsilon_i}{\Sigma (x_{1i}^0)^2}$$

The difference in estimated fuel intensity response after the policy change (in period k) and before the policy change (in period 0) is:

$$\hat{\beta}_1^k - \hat{\beta}_1^0 = \beta_1^k - \beta_1^0 + (\hat{\gamma}_{21}^k - \hat{\gamma}_{21}^0)\beta_2 + \frac{\Sigma x_{1i}^k \varepsilon_i}{\Sigma (x_{1i}^k)^2} - \frac{\Sigma x_{1i}^0 \varepsilon_i}{\Sigma (x_{1i}^k)^2}$$

The bias in the estimate of the change in slope is defined by:

$$E(\hat{\beta}_1^k - \hat{\beta}_1^0) = \beta_1^k - \beta_1^0 + (\hat{\gamma}_{21}^k - \hat{\gamma}_{21}^0)\beta_2$$

Thus, bias in the slope difference arises only when the data pattern among independent variables changes between the period k and period 0. In fact,

$$E(\hat{\beta}_1^k - \hat{\beta}_1^0) = \beta_1^k - \beta_1^0 \text{ when } \hat{\gamma}_{21}^k - \hat{\gamma}_{21}^0 = 0$$

That is, there is no bias when the coefficient for a regression between x_1 and x_2 is the same in the initial and the final periods.

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