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Cardiovascular disease—risk benefits of clean fuel technology and policy: A statistical analysis

Paul Gallagher [a,*], William Lazarus [b], Hosein Shapouri [c], Roger Conway [c], Fantu Bachewe [b], Amelia Fischer [a]

[a] Economics Department, 481 Heady Hall, Iowa State University, Ames Iowa 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, Washington, DC 20250, USA

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ABSTRACT

The hypothesis of this study is that there is a statistical relationship between the cardiovascular disease mortality rate and the intensity of fuel consumption (measured in gallons/square mile) at a particular location. We estimate cross-sectional regressions of the mortality rate due to cardiovascular disease 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 cardiovascular disease rate improvement estimate suggests that up to 60 cardiovascular disease 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 $30.3 billion annually. Across the US, the estimated Value of Statistical Life (VSL) benefit is $202.7 billion annually. There are likely three inseparable reasons that contributed importantly to this welfare improvement. First, the CAA regulations banned leaded gasoline, and mandated reduction in specific chemicals and smog components. Second, technologies such as the Catalytic Converter (CC) for the automobile and the 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 (U.S. 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 (Kittman).

Our estimate of the relation between an important health indicator, the mortality rate for heart disease and stroke (HDS), 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 HDS 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 HDS risk. Second, statistical estimates of the cross section relationship between the HDS mortality rate and the intensity of fuel consumption are presented. Third, policy-related reductions in HDS 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

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

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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 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 (U.S. EPA (2007), p. D-6).

Generally speaking, the long run health risks from air pollution are difficult to measure, because the many health effects are present only decades after exposure and air pollution is difficult to isolate as the sole cause (Cohen, 2003, p. 1011). One important determinant of long run HDS 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 study, not incorporated in policy analyses, suggests long-run relations between HDS risk and high emissions of nitrogen oxide and sulphate—apparently accumulated lead exposure (measured by bone lead) aggravates susceptibility in exposure to ozone and sulfates (Park et al., 2008, p. 6). Further, current research investigations focus on the HDS risks associated with other elements of urban air that come from the gasoline engine, especially Polycyclic Aromatic Hydrocarbons (PAH) (Lewtas, 2007, p. 95). Urban air chemical–HDS risk relationships are partially known, partially unknown.

The long run (HDS death rate) effect of policy and 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 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. Leaded gasoline was gradually banned at the same time because it damaged new cars equipped with the CC . There was an immediate decline in the urban population's lead blood level as the lead ban progressed (Kitman, 2000, p. 37). Further, a safe minimum threshold for exposure to lead apparently does not exist (Navas-Acien et al., 2007). Hence, a reduction in the long-run HDS rate due to the lead ban is plausible.

Production of high-octane lead-substitute additives increased steadily with the introduction of the CC. The lead ban was complete in 1995 (U.S. Department of Energy, 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, benzene restrictions were included to address cancer mortality risks; benzene in reformulated fuel was limited to 2.0% (U.S. Department of Energy, pp. 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 extensive HDS rate reductions over the last 20 years.

Particulate regulations for diesel were introduced after the 1990 CAA. New 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 h for 1994 models (U.S. 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% esther-blended diesel fuel only emits 85% of the particulates of #2 fuel oil (Manicom et al.). Some esther-blend tests have shown an increase in nitrous oxide emissions. However, adjusted engines reduce all categories of pollutants in some tests (Goetz). Overall, improved diesel engines and esther 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 (Ragsdale, 1994). Reduced criteria pollutant emissions include chemicals with known HDS-provoking characteristics: nitrogen oxides, ozone (Gryparis et al., 2004), and sulphur oxides (Sunyer et al., 2003). Further, there are potential long term effects due to an interaction between previous lead exposure and current susceptibility to ozone exposure (Park et al., 2008). Finally, emerging research on the HDS risks associated with PAH's, and the appearance of these chemicals with particulates, suggest a possible understatement of the importance of gasoline emissions and regulations.

3. Estimation procedures

A disease 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 harmful substances, or modern clean-burning engines dominate the vehicle fleet. In contrast, f_i is

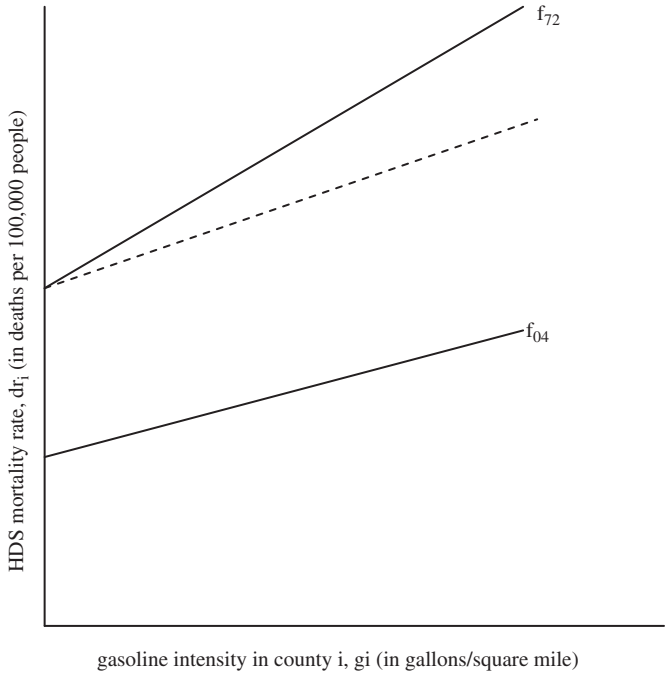

Fig. 1. HDS mortality rate—fuel intensity relationship.

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 obesity, drinking, and 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.

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:

$$\mathrm{dr}_{it} = \Sigma_t a_t \mathrm{Dt}_{it} + \Sigma_i a_i \mathrm{Ds}_{it} + \beta_t \mathrm{gi}_{it} + \varepsilon_{it} \quad (1)$$

dr_{it} is the 'age-adjusted' mortality rate due to cardiovascular disease, in deaths/100,000 people; gi_{it} the fuel (gasoline and diesel) use intensity, in gallons/mi^2; Dt_{it} is 1 for year t (1972, 2004), and 0 otherwise; Dsit is 1 for state s (s=al, ar, etc.), and 0 otherwise; ε_{it} is a random variable; α_t, α_i, β_i are parameters for estimation

Eq. (1) defines 2 cross-section regressions, defined by t=72 and t=04. Also, the index, i, refers to sub-state observations, mainly metropolitan counties of the US.

Initially, we expected to include explicit other health-determining factors as explanatory variables. Some state-level data on cigarette consumption, weight status, and health expenditures was 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 proxied the state of health habits, and health care delivery at each time and location using the 'state' and 'time' variables. The death rate – fuel intensity – binary variable approach to estimation is likely viable under most circumstances. First, bias in regression coefficients due to an omitted explanatory variable, such as health habits, does not occur when the independent variables are uncorrelated (Judge et al., 1982, p. 597). There is no reason to expect typical health habits to vary systematically across low density and high density urban areas. Thus, bias in β due to exclusion of cigarette consumption, using 2001 data, is not extensive—the 2004 correlation between fuel intensity and cigarette consumption was −.01. Similarly, the correlation between fuel intensity and the fraction overweight population was moderate, at −.13. Second, policy inferences based on changes in the slope of the fuel consumption-health relationship are likely valid even in the presence of higher correlations between fuel intensity and other (omitted) health variables, provided that the correlation pattern among independent variables is similar before and after the policy change.[1]

The dependent variable in Eq. (1) removes the effect of changing age distribution. We used the 'age adjusted' death rate due to cardiovascular disease. The age adjusted death rate for n age groups is:

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

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, 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.[2] Further, a particular state coefficient is constrained to be the same across both cross sectional equations.

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 by the National Center for Health Statistics (National Bureau of Economic Research). Individual records were available for 215 counties that were classified as metropolitan in 1972, which were all included in the analysis.

[1] See Appendix A for further discussion.

[2] 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.

Table 1
SUR estimate of heart disease and stroke mortality function.

$$d_{it} = \underset{(56.13)}{272.023}\mathrm{Dt}_{72} + \underset{(41.02)}{220.229}\mathrm{Dt}_{04}$$

$$+\underset{(5.64)}{78.753}\,\mathrm{Dal}_{it} + \underset{(1.58)}{36.809}\,\mathrm{Dar}_{it} + \underset{(2.43)}{15.986}\,\mathrm{Dca}_{t} - \underset{(-1.16)}{14.296}\,\mathrm{Dco}_{it} + \underset{(3.02)}{70.350}\,\mathrm{Dde}_{it} + \underset{(1.84)}{13.291}\,\mathrm{Dfl}_{it} + \underset{(2.26)}{28.169}\,\mathrm{Dga}_{it}$$

$$+\underset{(1.34)}{31.369}\,\mathrm{Dia}_{it} + \underset{(5.69)}{50.164}\,\mathrm{Dil}_{it} + \underset{(4.12)}{50.594}\,\mathrm{Din}_{it} + \underset{(3.00)}{50.473}\,\mathrm{Dky}_{it} + \underset{(4.85)}{59.550}\,\mathrm{Dla}_{it} + \underset{(2.09)}{19.317}\,\mathrm{Dma} + \underset{(3.10)}{34.860}\,\mathrm{Dmd}_{it}$$

$$+\underset{(4.70)}{45.765}\,\mathrm{Dmi}_{it} + \underset{(2.96)}{41.299}\,\mathrm{Dmo}_{it} + \underset{(1.37)}{32.001}\,\mathrm{Dms}_{it} + \underset{(3.68)}{41.096}\,\mathrm{Dnc}_{it} + \underset{(0.76)}{12.721}\,\mathrm{Dnh}_{it} + \underset{(5.82)}{44.325}\,\mathrm{Dnj}_{it} - \underset{(-1.25)}{29.269}\,\mathrm{Dnm}_{it}$$

$$+\underset{(2.53)}{42.656}\,\mathrm{Dnv} + \underset{(4.04)}{33.175}\,\mathrm{Dny}_{t} + \underset{(6.00)}{50.868}\,\mathrm{Doh}_{it} + \underset{(3.65)}{61.357}\,\mathrm{Dok}_{it} + \underset{(6.77)}{49.975}\,\mathrm{Dpa}_{it} + \underset{(1.56)}{36.342}\,\mathrm{Dri}_{it} + \underset{(4.05)}{49.837}\,\mathrm{Dsc}_{it} + \underset{(4.85)}{59.516}\,\mathrm{Dtn}_{it}$$

$$+\underset{(2.93)}{22.152}\,\mathrm{Dtx}_{it} + \underset{(3.96)}{55.157}\,\mathrm{Dva}_{it} + \underset{(1.72)}{19.276}\,\mathrm{Dwa}_{it} + \underset{(3.42)}{0.007483}\,g_{i72} - \underset{(-1.39)}{0.00418}\,g_{i04}$$

(Rural states of reference)
(MT, ND, SD, VT, WV, WY)
States without statistically significant Ds_i;
i=AZ, CT, DC, ID, KS, ME, NE, OR, UT, WI
States with statistically significant and positive Ds_i;
i=AL, AR, CA, DE, FL, GA, IA, IL, IN, KY, LA, MA, MD, MI, MO, MS, NC, NH, NJ, NM, NY, OH, OK, PA, RI, SC, TN, TX, VA, WA
States with statistically significant and negative Ds_i;
i=CO, NM
adj.R^2_{72}=.3575
adj.R^2_{04}=.1742

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 U.S. Environmental Protection Agency (Driver et al., 2007). The VMT data was 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 cardiovascular disease 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.

Also, 48 'other state' or 'rural' observations were used for preliminary estimations. These observations were constructed by subtracting the appropriate metropolitan counties from state level data.[3,4] This extended the range of exposure on the low side, and increased the sample size to 263 observations in each year.

5. Estimates

Estimates for the mortality rate function were based on Eq. (1). But several specifications were estimated to evaluate inclusion of specific dummy variables. Preliminary estimates suggested that both time dummies were significant and should be included. Initially, the Ds_i=0 situation refers to six rural states that did not have a metropolitan county in the 1972 reference data (mt, nd, sd, vt wv, and wy). Other state dummies were also excluded when the effect was not significantly different from zero. Furthermore, including rural observations did not affect the estimates, and precluded calculation of 2004 fuel-health correlations because appropriate state-level health data were unavailable. Hence, rural data is excluded from the reported results (Table 1).[5]

The estimated mortality response function is given in Table 1, and t-values for individual variables indicate statistically significant effects. The reported set of explanatory variables explain 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 Alabama, and the state with the lowest negative effect is New Mexico. Time and spatial variation in these effects can be attributed to changing health care technology, health care delivery, and health habits in particular locations. Indeed, the pattern of state dummies with large negative effects in several southern states conforms to preliminary estimates for the 2004 regression with available cigarette consumption data for 2004—cardiovascular disease rates tended to be high in the south (Alabama, Louisiana, Oklahoma, and Virginia).

[3] For the dependent variable, the raw data, the number of cardiovascular disease 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-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.

[4] 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.

[5] Similar estimates that include these rural observations are given in Appendix B.

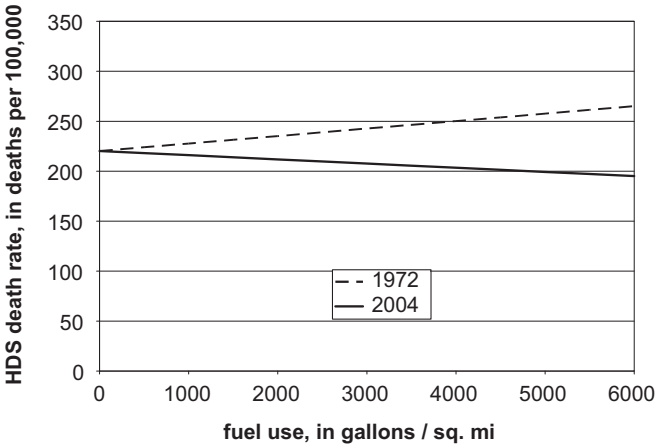

Fig. 2. Estimated death rate vs. fuel intensity: 2004 basis.

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.[6]

One would not expect a negative estimate $\hat{\beta}_{04}$ in Table 1 when all health shifting variables are uncorrelated with gi. In the strictly urban sample defining the results of Table 1 and Appendix D, however, there is a moderate negative correlation between overweight and fuel intensity in 2004. A priori bias analysis suggests that the estimate $\hat{\beta}_{04}$ would be low without the overweight variable. The 2004 regression of an all urban sample with the overweight variable included (in Appendix Table D1 bears this out—here, $\hat{\beta}_{04}$ is positive but not strictly significant.

Still, the estimate of the difference, $\hat{\beta}_{04} - \hat{\beta}_{72}$, drives the policy analysis. The analysis of Appendix A suggests that unbiased difference estimates will likely occur. Unbiased difference estimates should hold as long as the overweight/fuel intensity intercorrelation across counties holds in both time periods. Overall, the results of Table 1 (and Table B1) confirm that the impact of increasing fuel intensity on HDS has been reduced since CAAs, CC, and ethanol. However, one should not necessarily conclude that the effect is eliminated.

6. Calculations of policy change effects

We now present the mortality rate reduction and associated economic value associated with the policy change, including individual metropolitan areas and for the entire US. First, the calculation of mortality rate change is explained. Second, the valuation procedure is discussed. Third, the calculations are presented.

6.1. Mortality change

For an estimate of today's cardiovascular disease 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. 2. 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, $t = \Delta/\mathrm{sd}(\Delta)$, has a t distribution with N-K degrees of freedom under the null hypothesis that the mortality function does not change with CAA policy.[7] For the estimates of Table 1, $\Delta = -0.011663$, $\mathrm{sd}(\Delta) = 0.00357$, and $t = -3.25$. 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

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

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

Then the mortality change accross all counties is

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

6.2. Valuation

We use a Value of Statistical Life (VSL) estimate to value the mortality reduction associated with the CAAs. Now we summarize the important concepts, estimates and limitations associated with this method.

Measurements of individuals' willingness to trade money for a slightly higher death risk often rely on a statistical relationship between wages and the risk of accidental death in various occupations (Moore and Viscusi, 1988, p. 484). The essential estimates from a well-known cross sectional study are

$$\ln(w_i) = k + 0.0075\, p_i - 0.0081\, p_i c_i,$$

[6] 2004 regression that includes other health variables is given in Appendix Table D1. The estimated fuel intensity effect for 2004 is not significant, but may suggest that a positive effect remains.

[7] For demonstration, the mortality function estimate in the terminal period n is:

$$\hat{d}_{in} = \hat{\alpha}_n \mathrm{Dt}_n + \hat{\alpha}_i \mathrm{Ds}_{in} + \hat{\beta}_n g_{in}.$$

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, $g_{in} = g_{io} = g_i$, gives:

$$\Delta = (\hat{\beta}_n - \hat{\beta}_0) g_i.$$

The statistic, 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.

$$t = \Delta/sd(\Delta) = \frac{\hat{\beta}_n - \hat{\beta}_o}{\sqrt{\mathrm{Var}(\hat{\beta}_n) + \mathrm{Var}(\hat{\beta}_o) - 2\mathrm{Cov}^1(\hat{\beta}_n, \hat{\beta}_o)}}$$

has a t distribution with N-K degrees of freedom (Kmenta, p. 372).

Table 2
Mortality (heart disease and stroke) reductions and lives saved by clean air act: counties with largest effects, and total US.

State	County seat	2004 Fuel use (gal/mi^2)	2004 Population	Mortality change (deaths/100,000)	Deaths avoided:	
					Number	Value-VSL (mil. $)
Most improvement in mortality—top 10 counties						
DC	DC	5148.1	554,239	−60.431	−335	2344.5
NY	White Plains	3996.4	941,380	−55.617	−524	3664.9
PA	Philadelphia	3875.4	1,471,255	−55.111	−811	5675.7
NY	New York	3393.2	8,164,706	−53.095	−4335	30,345.5
NJ	Jersey City	3342.1	605,359	−52.882	−320	2240.9
NJ	Newark	3400.5	795,015	−51.165	−407	2847.4
PA	Doylestown	3663.3	617,214	−50.471	−312	2180.6
MD	Baltimore	3550.2	641,943	−45.575	−293	2047.9
NJ	Elizabeth	3552.4	530,846	−41.736	−222	1550.9
MA	Boston	2915.5	664,263	−39.919	−265	1856.2
				Subtotal	−7822	54,754.5
Most improvement in mortality—top 20 counties						
MI	Mount Clemens	3213.0	822,965	−38.514	−317	2218.7
NY	Goshen	2277.0	369,511	−38.258	−141	989.6
PA	Easton	2676.1	283,333	−37.745	−107	748.6
IL	Decatur	5200.0	674,335	−36.421	−246	1719.2
CA	Oakland	2800.2	1,452,096	−35.203	−511	3578.2
MI	Detroit	2680.7	2,013,771	−32.812	−661	4625.4
NJ	Boston	2915.5	664,263	−31.432	−283	1856.2
CO	Denver	3173.7	555,991	−28.960	−161	1127.1
NY	New City	3029.4	293,049	−28.819	−84	591.2
DE	Wilmington	2579.4	518,728	−28.833	−150	1046.9
				Subtotal	−2661	18,501.1
			Total, United States		−28,915	202,999

where w_i (7.01(7.0)) is the wage for individual i, in $/hr, p_i (7.98(9.7)) is i's chance of accidental death, in deaths /100,000 workers, c_i (0.54(0.2)) is i's salary replacement rate under workers' compensation, 0–1.

Above, sample means (m) and standard deviations (s) are in parentheses: (m(s)).

The wage change that compensates the typical worker for an increase in the accidental death probability of 1 more worker out of 100,000 is:

$$\Delta \ln(w_i) = 0.0075 - 0.0081\, c_i$$

Evaluating at sample means (with 8 h/day, 5 day/week, and 52 wk/yr) implies that the wage-accidental death chance tradeoff is

$$\frac{\Delta wi}{\Delta pi} = \frac{\$44.72/\text{yr}}{\text{one fatality}}$$

An extrapolation of the incremental tradeoff to the certainty case (Δpi=100,000) is computationally useful. That is, the value of statistical life (VSL) implied by the wage-fatality risk tradeoff is

$$\text{VSL} = \frac{\Delta wi}{\Delta pi} \times 100{,}000 = \$4.472 \text{ million/yr}$$

The VSL is merely an arithmetically convenient way of summarizing the wage-accident tradeoff. To illustrate, the cost to a typical worker of a policy that increases the accidental death probability by 0.0001 (10 per 100,000 workers) is

$$0.0001 \times VSL = \$447.2/\text{yr}.$$

Now consumers' willingness to accept a higher mortality rate environment can be calculated without knowing all the details of the data used in a particular study (akin to the elasticity).

The VSL estimate of $7 million/person used in this study is the median value of VSL estimates from 30 US studies (Viscusi and Aldy (2003), p. 63). Similarly, the US government recommends an estimate of $7.4 million, based another meta-analysis of several studies (US EPA).

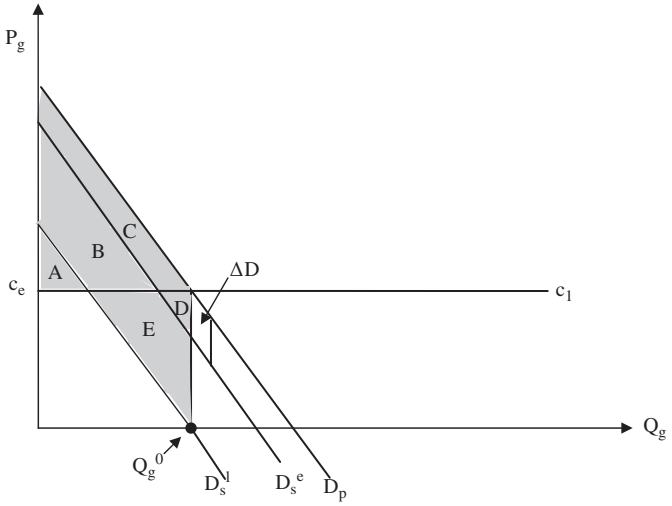

Fig. 3. Welfare analysis: ethanol gas or gas blended with lead.

Occasionally, it makes sense to place a value on human life. For instance, a calculation of the discounted value of lost earnings associated with an accidental death is routinely used as the basis for a damage estimate where someone is legally responsible for the death. But most reject the notion of placing a value on human life in a policy analysis setting. Importantly, the VSL literature does something else: it measures individuals' willingness to trade money for an environment with an incrementally higher mortality risk, the type of decision that occurs routinely in labor markets. It also extrapolates the estimated willingness to pay for small changes in the mortality risk that are associated with policy changes. Still the VSL terminology is unfortunate, because it invites comparison to the related concept, the value of a human

Table B1
SUR estimate of heart disease and stroke mortality function: rural counties are included.

$$d_{it} = 274.623\ \mathrm{Dt}_{72} + 210.473\ \mathrm{Dt}_{04}$$
$$(70.85) \quad (48.78)$$

$$+76.695\ \mathrm{Dal}_{it} + 42.185\ \mathrm{Dar}_{it} + 17.515\ \mathrm{Dca}_{t} - 21.969\ \mathrm{Dco}_{it} + 57.068\ \mathrm{Dde}_{it} + 12.053\ \mathrm{Dfl}_{it} + 32.818\ \mathrm{Dga}_{it}$$
$$(6.44) \quad (2.56) \quad (2.96) \quad (-2.04) \quad (3.47) \quad (1.84) \quad (3.02)$$

$$+21.068\ \mathrm{Dia}_{it} + 50.347\ \mathrm{Dil}_{it} + 49.657\ \mathrm{Din}_{it} + 55.924\ \mathrm{Dky}_{it} + 60.891\ \mathrm{Dla}_{it} + 16.784\ \mathrm{Dma} + 34.535\ \mathrm{Dmd}_{it} + 21.561\ \mathrm{Dme}$$
$$(1.28) \quad (6.28) \quad (4.61) \quad (4.11) \quad (5.66) \quad (2.00) \quad (3.44) \quad (1.31)$$

$$+45.419\ \mathrm{Dmi}_{it} + 41.298\ \mathrm{Dmo}_{it} + 51.006\ \mathrm{Dms}_{it} + 43.354\ \mathrm{Dnc}_{it} + 15.215\ \mathrm{Dnh}_{it} + 44.852\ \mathrm{Dnj}_{it} - 27.655\ \mathrm{Dnm}_{it}$$
$$(5.16) \quad (3.47) \quad (3.10) \quad (4.37) \quad (1.12) \quad (6.41) \quad (-1.68)$$

$$+28.358\ \mathrm{Dnv} + 34.793\ \mathrm{Dny}_{t} + 51.743\ \mathrm{Doh}_{it} + 58.854\ \mathrm{Dok}_{it} + 51.789\ \mathrm{Dpa}_{it} + 29.438\ \mathrm{Dri}_{it} + 53.508\ \mathrm{Dsc}_{it} + 61.510\ \mathit{Dtn}_{it}$$
$$(2.08) \quad (4.64) \quad (6.70) \quad (4.33) \quad (7.70) \quad (1.79) \quad (4.97) \quad (5.71)$$

$$+24.209\ \mathrm{Dtx}_{it} + 46.474\ \mathrm{Dva}_{it} + 16.352\ \mathrm{Dwa}_{it} + .00627\ g_{i72} + .001142\ g_{i04}$$
$$(3.53) \quad (3.91) \quad (1.65) \quad (3.05) \quad (0.41)$$

(Rural states of reference)
(MT, ND, SD, VT, WV, WY)
States without statistically significant Ds_i;
i=AZ, CT, DC, ID, KS, NE, OR, UT, WI
States with statistically significant and positive Ds_i;
i=AL, AR, CA, DE, FL, GA, IA, IL, IN, KY, LA, MA, MD, ME, MI, MO, MS, NC, NH, NJ, NM, NY, OH, OK, PA, RI, SC, TN, TX, VA, WA
States with statistically significant and negative Ds_i;
i=CO, NM
adj.R^2_{72}=.3599
adj.R^2_{04}=.2389

life, which is objectionable. Ironically, more obscure economic language would probably enhance widespread understanding of the VSL concept.

VSL estimates have some characteristics that are consistent with willingness to pay or accept concepts. First, VSL estimates are higher in wealthy countries than in poor countries. This may explain why wealthier societies are more interested in environmental regulations that improve health. On the other hand, it would likely preclude across country comparisons. Second, VSL is will likely increase with an individual's age due to the accumulation of wealth. In contrast, a discounted value of lost earnings estimate would decrease with age. In short, VSL will not discriminate against wealthy old people. But it might not assign an appropriate value for poor young people.

6.3. Results

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 7823 people annually for the highest 10 cities, a cumulative total 10,589 people annually for the top 20 cities, and 29,000 people for the entire US.[8]

For valuation, we estimate that the combined reductions, technology advances, and subsidies provide an annual value of $203 billion for the entire US. Further, the 10 high cities reduce their annual loss by nearly $55 billion and the top 20 cities reduce their death loss by $74.1 billion, annually. That is, the value of reduced loss of human life is $203 billion throughout the US.

For comparison, the changing fuel intensity effect reduced the US VSL estimate by about 8%, from $221 billion to the $203 billion shown in Table 2. There were a few large coastal cities 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] Estimates of mortality reduction from CAA policies for all counties, sub-state rural areas, and the US are given in Appendix Table C.

7. Welfare interpretation of estimates

To welfare economics, the $203 billion estimate of annual cardiovascular disease reduction benefit represents a market externality. Next we show how policy, technology, and product substitution reduced the size of the cardiovascular disease risk and internalized the externality. We also discuss effects of today's incremental fuel changes with current CAA policy, technology, and substitution possibilities in place

To illustrate, consider the market for gasoline that is blended with high-octane gasoline additives. In Fig. 3, the private demand for mixed gasoline is D_p. Initially, suppose the supply is defined by constant-cost production of leaded-fuel, C_1. In the initial baseline, the actual social demand curve, D^1_s, is below D_p due to the adverse cardiovascular disease 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^e_s, 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^0_g, 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

Table C1
Mortality reductions and lives saved by clean air act, county by county.

State	County seat	Fuel use	Mortality change	Population	Deaths Avoided	
		gal/mi^2	Deaths per 100,000		Number	Value (mil $)
AL	Birmingham	639.5	−6.80	658468	−45	314
AL	Huntsville	313.9	−3.20	293598	−9	66
AL	Mobile	198.2	−2.35	400107	−9	66
AR	Little rock	655.37	−5.29	365228	−19	135
AZ	Phoenix	335.96	−2.45	3498587	−86	600
AZ	Tuscon	76.9	−0.67	906540	−6	43
CA	Oakland	2800.21	−35.20	1452096	−511	3578
CA	Visalia	66.8	−0.78	400952	−3	22
CA	Ventura	217.1	−2.21	796165	−18	123
CA	Martinez	936.7	−8.89	1007606	−90	627
CA	Fresno	109.4	−1.13	865620	−10	69
CA	Bakersfield	471.63	−4.82	734077	−35	248
CA	Los Angeles	1255	−13.33	9917331	−1322	9254
CA	Salinas	98.2	−1.05	414551	−4	30
CA	Santa ana	2136.9	−19.21	2982094	−573	4010
CA	Riverside	1631.97	−14.37	1869465	−269	1880
CA	Sacremento	965.2	−8.58	1351428	−116	812
CA	San bernardino	752.56	−7.87	1916418	−151	1056
CA	San diego	536.9	−4.84	2935190	−142	995
CA	San fransisco	1744	−24.62	743193	−183	1281
CA	Stockton	1108.32	−13.70	649241	−89	623
CA	Redwood city	897.5	−8.65	698156	−60	423
CA	Santa barbara	76.5	−0.77	401708	−3	22
CA	San jose	958.9	−9.48	1681980	−159	1116
CA	Santa cruz	288.4	−3.64	250837	−9	64
CA	Fairfield	1421.7	−17.80	411896	−73	513
CA	Santa rosa	1190.14	−8.16	467932	−38	267
CA	Modesto	988.34	−12.68	497599	−63	442
CO	Denver	3173.7	−28.96	555991	−161	1127
CO	Col springs	178.7	−1.54	557752	−9	60
CO	Littleton	448.6	−3.77	522346	−20	138
CO	Fort collins	1710.53	−15.40	268960	−41	290
CT	Bridgeport	762.7	−10.53	901819	−95	665
CT	New london	344.3	−3.30	266107	−9	62
CT	Hartford	884.8	−9.50	873879	−83	581
CT	New haven	2197.14	−30.46	844342	−257	1800
DC	Dc	5148.1	−60.43	554239	−335	2345
DE	Wilmington	2579.41	−28.83	518728	−150	1047
FL	Dade city	369.7	−3.47	408046	−14	99
FL	Clearwater	1406.6	−11.65	927498	−108	756
FL	Bartow	266.3	−2.31	524286	−12	85
FL	Fort lauderdale	1028.8	−7.99	1753000	−140	980
FL	Sarasota	1262.6	−10.13	355722	−36	252
FL	Sanford	1150.2	−8.26	391241	−32	226
FL	De land	340.7	−2.75	478951	−13	92
FL	Jacksonville	1023.1	−8.60	819623	−70	493
FL	Pensacola	401.3	−3.34	296739	−10	69
FL	Tampa	774.4	−7.27	1100333	−80	560
FL	Fort meyers	456.8	−3.39	514923	−17	122
FL	Bradenton	285	−2.22	295974	−7	46
FL	Ocala	221	−1.59	291768	−5	32
FL	Titusville	351.8	−2.72	518812	−14	99
FL	Orlando	901.4	−6.85	989873	−68	474
FL	W. Palm beach	402.9	−3.04	1244189	−38	265
GA	Atlanta	2432.7	−16.02	905802	−145	1016
GA	Lawrenceville	1593.4	−9.43	700577	−66	463
GA	Marietta	4146.63	−26.93	654649	−176	1234
GA	Decatur	5200	−36.42	674335	−246	1719
IA	Des moines	677.6	−5.67	394031	−22	157
ID	Boise	240.84	−2.12	332545	−7	49
IL	Woodstock	318.1	−2.84	296260	−8	59
IL	Edwardsville	2267.71	−12.00	263443	−32	221
IL	Belleville	2539.44	−13.55	259123	−35	246
IL	Joliet	491.9	−4.36	617494	−27	189
IL	Rockford	445.8	−4.50	286283	−13	90
IL	Chicago	1953.1	−23.03	5327165	−1227	8588
IL	Wheaton	2364	−19.10	928126	−177	1241
IL	Geneva	610.5	−3.41	472761	−16	113
IL	Waukegan	1706.62	−18.61	692869	−129	903
IN	South bend	1169.07	−14.15	265718	−38	263
IN	Fort wayne	889.98	−10.10	341816	−35	242
IN	Crown point	634.3	−8.21	490089	−40	282
IN	Indianapolis	1656.5	−19.13	861847	−165	1154

Table C1 (*continued*)

State	County seat	Fuel use	Mortality change	Population	Deaths Avoided	
		gal/mi^2	Deaths per 100,000		Number	Value (mil $)
KS	Wichita	317.8	−3.31	463383	−15	107
KS	Olathe	901.4	−7.92	496892	−39	276
KY	Eddyville	2018.5	−19.43	698903	−136	951
KY	Lexington	890.97	−8.33	266451	−22	155
LA	Shreveport	379.03	−3.65	250893	−9	64
LA	Baton rouge	680.4	−6.23	411564	−26	179
LA	Gretna	1006.35	−11.06	453089	−50	351
LA	New orleans	709.6	−7.90	461115	−36	255
MA	Hambden	497.5	−6.05	461491	−28	195
MA	Cambridge	1376.8	−14.83	1462822	−217	1519
MA	Dedham	1889.4	−15.21	653621	−99	696
MA	Plymouth	203.6	−3.07	489979	−15	105
MA	Boston	2915.5	−39.92	664263	−265	1856
MA	Worcsester	452.2	−4.42	778608	−34	241
MA	Taunton	341.8	−4.89	547278	−27	187
MA	Newburyport	1394.51	−16.68	737447	−123	861
MD	Annapolis	786.1	−6.03	508356	−31	215
MD	Rockville	1249.7	−13.09	921631	−121	844
MD	Upper marlboro	1436.1	−15.10	841642	−127	889
MD	Bristol	984.4	−8.30	781171	−65	454
MD	Baltimore	3550.2	−45.57	641943	−293	2048
ME	Portland	247.26	−2.52	273622	−7	48
MI	Pontiac	1359.4	−12.89	1212181	−156	1094
MI	Ann arbor	526.3	−4.55	338782	−15	108
MI	Detroit	2680.7	−32.81	2013771	−661	4625
MI	Flint	760.1	−8.09	443497	−36	251
MI	Mason	435.9	−4.67	280093	−13	92
MI	Grand rapids	649	−5.82	592999	−35	242
MI	Mount clemens	3213.01	−38.51	822965	−317	2219
MN	Saint paul	2546.8	−25.82	499206	−129	902
MN	Anoka	2501.06	−24.65	319548	−79	551
MN	Hastings	527.1	−4.10	378343	−16	109
MN	Minneapolis	1599.4	−15.42	1119866	−173	1209
MO	Saint charles	497.1	−2.89	320459	−9	65
MO	Saint louis	2498.9	−11.08	1007723	−112	782
MO	Kansas city	726.6	−4.60	662185	−30	213
MS	Jackson	388.13	−2.86	249828	−7	50
NC	Charlotte	1204.2	−10.51	771573	−81	568
NC	Raleigh	815.21	−5.93	719733	−43	299
NC	Fayetteville	402.51	−3.95	306943	−12	85
NC	Winston−salem	901.85	−8.49	320780	−27	191
NC	Greensboro	926.25	−7.80	437879	−34	239
NE	Lincoln	478.83	−3.74	261742	−10	68
NE	Omaha	1006.92	−10.74	481203	−52	362
NH	Nashua	2312.51	−30.42	398355	−121	848
NH	Concord	408.7	−3.46	292346	−10	71
NJ	Mays landing	364	−3.75	268311	−10	71
NJ	Newark	3400.5	−51.17	795015	−407	2847
NJ	Woodbury	667.6	−7.54	272784	−21	144
NJ	Jersy city	3342.1	−52.88	605359	−320	2241
NJ	Trenton	1013.1	−14.02	364381	−51	358
NJ	New brunswick	2064.9	−19.45	783665	−152	1067
NJ	Freehold	740.1	−7.90	635062	−50	351
NJ	Morristown	898	−9.54	487437	−46	325
NJ	Toms river	289.5	−4.02	553093	−22	156
NJ	Hackensack	2891.9	−31.43	901745	−283	1984
NJ	Paterson	1332.5	−19.02	498939	−95	664
NJ	Somerville	2534.08	−25.07	316223	−79	555
NJ	Elizabeth	3552.4	−41.74	530846	−222	1551
NJ	Mount holly	513.5	−5.44	448656	−24	171
NJ	Camden	1758.5	−22.07	515620	−114	797
NM	Albuqerque	454.9	−4.09	592538	−24	170
NV	Las vegas	170.56	−1.46	1648524	−24	168
NV	Reno	218.23	−2.84	380612	−11	76
NY	Albany	1311.58	−16.95	297910	−50	353
NY	Riverhead	2186.91	−28.62	1474519	−422	2954
NY	White plains	3996.43	−55.62	941380	−524	3665
NY	Poughkeepsie	1849.2	−28.17	293322	−83	578
NY	Buffalo	651.9	−6.81	935946	−64	446
NY	Rochester	469.8	−4.65	735816	−34	240
NY	Moneola	2175.7	−16.95	1337693	−227	1587
NY	Syracuse	799.31	−9.96	458870	−46	320
NY	Goshen	2277.02	−38.26	369511	−141	990
NY	New city	3029.35	−28.82	293049	−84	591

Table C1 (*continued*)

State	County seat	Fuel use	Mortality change	Population	Deaths Avoided	
		gal/mi^2	Deaths per 100,000		Number	Value (mil $)
NY	New york	3393.2	−53.10	8164706	−4335	30345
OH	Dayton	1013.5	−11.32	549553	−62	435
OH	Waverly	616.65	−8.31	380545	−32	221
OH	Akron	1008.7	−11.67	546608	−64	447
OH	Hamilton	1867.91	−19.60	346123	−68	475
OH	Cleveland	750.9	−10.29	1349047	−139	971
OH	Columbus	1685.4	−15.62	1087462	−170	1189
OH	Cincinnati	1659.9	−19.90	813639	−162	1133
OH	Elyria	288.5	−1.73	293532	−5	35
OH	Toledo	674.8	−7.33	450304	−33	231
OH	Youngstown	558.6	−6.63	255995	−17	119
OK	Oklahoma city	1013.2	−9.97	679498	−68	474
OK	Tulsa	1085.8	−10.65	568611	−61	424
OR	Eugene	52.5	−0.60	331567	−2	14
OR	Salem	213.9	−2.03	301702	−6	43
OR	Oregon city	143.5	−1.65	362681	−6	42
OR	Portland	1184.1	−10.65	671363	−71	500
OR	Hillsboro	405.3	−3.75	487548	−18	128
PA	Philadelphia	3875.4	−55.11	1471255	−811	5676
PA	Reading	342.6	−3.89	391447	−15	107
PA	Greensburg	311.5	−3.73	367937	−14	96
PA	York	2383.1	−30.59	401063	−123	859
PA	Doylestown	3663.28	−50.47	617214	−312	2181
PA	West chester	497.9	−4.74	466043	−22	155
PA	Pittsburgh	1142.2	−14.92	1247512	−186	1303
PA	Harrisburg	2115.72	−25.60	253060	−65	453
PA	Media	1784.8	−18.09	554426	−100	702
PA	Erie	407.34	−5.22	280844	−15	103
PA	Lancaster	2176.98	−27.20	486361	−132	926
PA	Allantown	847.5	−8.49	325570	−28	193
PA	Wilkes-barre	294.4	−3.37	313088	−11	74
PA	Norristown	1311.3	−11.14	773375	−86	603
PA	Easton	2676.05	−37.74	283333	−107	749
RI	Providence	912.7	−10.69	641874	−69	481
SC	Charleston	352.69	−2.79	327403	−9	64
SC	Greenville	968.11	−7.47	401019	−30	210
SC	Columbia	684.66	−5.64	335597	−19	132
SC	Spartanburg	748.86	−6.11	264106	−16	113
TN	Memphis	1086.7	−10.22	906287	−93	648
TN	Nashville	1824.6	−13.66	571948	−78	547
TN	Chattanooga	581.6	−5.76	309729	−18	125
TN	Knoxville	1296.46	−10.03	400340	−40	281
TX	Dallas	2190.2	−9.35	2291071	−214	1499
TX	Denton	401.2	−3.30	530982	−18	123
TX	El paso	395	−3.87	712617	−28	193
TX	Richmond	1435.75	−14.34	442389	−63	444
TX	Galveston	201.4	−2.20	272024	−6	42
TX	Houston	1571.2	−15.91	3641114	−579	4056
TX	Edinburg	284.59	−2.85	657310	−19	131
TX	Beaumont	215.3	−2.31	248308	−6	40
TX	San antonio	901.6	−8.33	1492361	−124	870
TX	Corpus christi	450.5	−4.47	317317	−14	99
TX	Fort worth	1364.1	−6.41	1587019	−102	712
TX	Austin	685.9	−5.46	868873	−47	332
TX	Brownsville	233.8	−2.34	370829	−9	61
TX	Mckinney	452.2	−2.81	628426	−18	124
UT	Salt lake city	788.4	−7.24	934838	−68	474
UT	Provo	161.4	−1.21	434114	−5	37
VA	Chesterfield	603.3	−5.14	282470	−15	102
VA	Virginia beach	473.3	−4.94	439224	−22	152
VA	Richmond	1046.3	−8.97	275962	−25	173
WA	Vancouver	1476.68	−13.37	392364	−52	367
WA	Seattle	863.43	−7.98	1777746	−142	993
WA	Tacoma	580.48	−5.32	745778	−40	277
WA	Everett	361.85	−3.29	644205	−21	148
WA	Spokane	219.31	−2.37	435146	−10	72
WI	Menomonie	1819.78	−18.90	376476	−71	498
WI	Madison	321.6	−2.98	453051	−14	95
WI	Millwaukee	636	−7.84	926764	−73	508
AL	Clanton	77.7	−0.73	3173202	−23	162
AR	Little rock	123.17	−0.78	2384772	−19	131
AZ	Flagstaff	29.68	−0.26	1334752	−4	25
CA	Red bluff	41.69	−0.53	3396503	−18	127
CO	Aspen	24.3	−0.23	2654316	−6	42

Table C1 (*continued*)

State	County seat	Fuel use	Mortality change	Population	Deaths Avoided	
		gal/mi^2	Deaths per 100,000		Number	Value (mil $)
CT	Willimantic	219.7	−2.21	612819	−14	95
DE	Dover	1841.15	−23.40	311341	−73	510
FL	Bristol	215.91	−1.98	4115738	−82	571
GA	Hawkinsville	116	−1.07	5982766	−64	448
IA	Des moines	142.28	−1.35	2558873	−34	241
ID	Arco	13.96	−0.14	1062595	−1	10
IL	Taylorville	1029.46	−5.03	3568492	−180	1257
IN	Lebanon	143.6	−1.43	4267067	−61	426
KS	Great bend	74.47	−0.72	1773422	−13	89
KY	Beattyville	230.11	−1.98	3176481	−63	439
LA	Colfax	59.8	−0.57	2930024	−17	117
MA	Northampton	712.24	−7.40	611873	−45	317
MD	Easton	178.5	−1.81	1866589	−34	236
ME	Bath	712.88	−9.64	1041363	−100	703
MI	Harrison	236.74	−2.77	4399918	−122	852
MN	Grand rapids	420.99	−4.26	2779583	−118	828
MO	Lebanon	65.1	−0.73	3769165	−28	193
MS	Hattiesburg	63.9	−0.59	2650940	−16	109
MT	Lewistown	7.31	−0.08	926920	−1	5
NC	Carthage	1879.54	−12.71	6062415	−771	5396
ND	Mcclusky	9.9	−0.11	636308	−1	5
NE	Broken bow	107.98	−1.02	1004759	−10	72
NH	Lancaster	564.86	−7.44	608468	−45	317
NJ	Belvidere	2032.25	−29.83	708030	−211	1478
NM	Carrizozo	15.22	−0.14	1310468	−2	13
NV	Eureka	19.62	−0.24	303762	−1	5
NY	Auburn	96.2	−1.37	12142711	−166	1165
OH	Up sandusky	127.8	−1.43	5377335	−77	539
OK	Coalgate	551.82	−2.61	2275437	−59	416
OR	Canyon city	16.3	−0.18	1436502	−3	18
PA	Clearfield	462.44	−5.91	4161943	−246	1723
RI	Newport	773.49	−12.02	438042	−53	369
SC	Oraneburg	345	−2.60	2869767	−74	521
SD	Pierre	10.6	−0.12	770621	−1	6
TN	Woodbury	104.8	−0.98	3704994	−36	253
TX	Baird	39	−0.41	8299183	−34	237
UT	Loa	14.7	−0.12	1051756	−1	9
VA	Cumberland	156.3	−1.48	6483676	−96	674
VT	Montpelier	499.386	−6.65	621233	−41	289
WA	Ephrata	104.781	−0.96	2211807	−21	149
WI	Wausau	454.25	−4.59	3747242	−172	1204
WV	Sutton	76.2	−0.78	1812548	−14	99
WY	Casper	183.243	−1.68	505887	−8	59
					−28915	202406

overall net benefit change from the joint technology change, lead ban, and ethanol substitution: area $B+E$ in Fig. 3.[9]

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 cardiovascular disease rate reduction. A parallel set of external cost reductions for introducing the clean diesel engine/ biodiesel also exists.[8] They are also included in the $203 billion estimate of net benefits. Besides CC and diesel engine components of the externality reductions, conceptualization of the HDS 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 HDS rate reductions. It excludes short-run health effects.[10]

In principle, mortality function estimates also define the environmental cost of some incremental changes in fuel policy

[9] Fig. 3 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 requires only requires a specific definition of D_s^e to represent the social demand with the appropriate new technology/fuel blending situation.

[10] A reviewer notes analysis of an increase in ethanol's concentration inin fuel will require separate analysis of emission, air quality, and health components. Some of the components for long-run HDS impacts are available now, at least qualatively. The complex model of emissions (Federal Register, 1995) allows our comparision of a 10% ethanol blend to a baseline fuel with the same qualities but no ethanol—the calculations show the EPA's criteria pollutant groups, volatile organic compounds (VOCs), nitric oxides (NOX), and toxic chemicals (TOX), decline. There is a composition change within the toxic chemicals group: benzene and butadiene decline, formaldehyde and particulates do not change, but acetheldyde increases. Similiarly, an emissions test of 20% ethanol blends in post-2002 vehicles found that the broad chemical categories (non-methane oxygenated gas(nmog) and non-methane hydrocarbons(nmhc)) and carbon monoxide declined (West et al., 2008, p. 3-3, table 3.1). Within the toxic chemicals group, though, there were some increases for acetylhedyde and formaldehyde (West et al., 2008). Generally, reductions in ozone precursors, such as NOX and CO, should reduce HDS (Gryparis et al., 2004). Aldehyde increases do not appear to aggravate HDS risk. Aldehydes may aggravate cancer risk, but benzene reductions have an offsetting cancer effect.

given today's technology and CAA policy. Specifically, $\hat{\beta}_{04}$ defines the mortality increment associated with a unit fuel expansion, given todays technology and CAA policy. In turn, the product of the mortality increment and the VSL define the welfare area, ΔD—ΔD measures the incremental environmental cost of another unit of blended-fuel with today's technology and policy. Estimation results for the level of $\hat{\beta}_{04}$ are somewhat mixed. But according to the result in Appendix D, it may approach$\hat{\beta}_{04} = +0.003$. Using this estimate, we calculate that there is a \$3.1 billion annual HDS cost associated with a nationwide increase in fuel consumption of 5%. Hence, policies that allow increasing fuel consumption in congested areas likely offset some of the Broad environmental policy and technology gains.[10]

8. Conclusions

Our analysis suggests that there is a statistical relation between the HDS 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 60 cardiovascular disease deaths per 100,000 residents in the largest urban areas with high fuel consumption per square mile. The initial period estimated the cardiovascular disease 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 cardiovascular disease 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 cardiovascular disease mortality difference before and after clean air regulations is substantial. In New York City, for instance, the mortality reduction may be worth about \$30.3 billion annually. Across the US, the benefit is \$203 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 A

To illustrate when the change in fuel intensity response is unbiased, 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:

$$y_i^k = \beta_1^k x_{i2}^k + \beta_2 x_{2i}^k + \varepsilon_i$$

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:

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

Table D1
SUR estimate of heart disease and stroke mortality function: using only 2004 data and including health variables. rural observations are excluded.

$$d_{it} = 110.044\ \mathrm{Dt}_{04} \quad (4.62)$$

$$+86.218\ \mathrm{Dal}_{it} + 34.846\ \mathrm{Dar}_{it} + 35.914\ \mathrm{Dca}_t - 14.803\ \mathrm{Dco}_{it} + 44.722\ \mathrm{Dde}_{it} + 27.233\ \mathrm{Dfl}_{it} + 21.514\ \mathrm{Dga}_{it}$$
$$(4.95) \quad (1.20) \quad (4.21) \quad (-0.96) \quad (1.54) \quad (3.00) \quad (1.36)$$

$$+20.979\ \mathrm{Dia}_{it} + 36.389\ \mathrm{Dil}_{it} + 40.653\ \mathrm{Din}_{it} + 33.304\ \mathrm{Dky}_{it} + 60.119\ \mathrm{Dla}_{it} + 13.461\ \mathrm{Dma} + 30.855\ \mathrm{Dmd}_{it} - 16.226\ \mathrm{Dme}$$
$$(0.73) \quad (3.30) \quad (2.63) \quad (1.59) \quad (3.93) \quad (1.17) \quad (2.19) \quad (-0.56)$$

$$+50.487\ \mathrm{Dmi}_{it} + 48.080\ \mathrm{Dmo}_{it} + 70.135\ \mathrm{Dms}_{it} + 24.154\ \mathrm{Dnc}_{it} + 15.396\ \mathrm{Dnh}_{it} + 28.689\ \mathrm{Dnj}_t - 2.099\ \mathrm{Dnm}_t$$
$$(4.15) \quad (2.76) \quad (2.42) \quad (1.77) \quad (0.74) \quad (3.03) \quad (-0.07)$$

$$+39.841\ \mathrm{Dnv} + 44.618\ \mathrm{Dny}_t + 27.218\ \mathrm{Doh}_{it} + 96.308\ \mathrm{Dok}_{it} + 32.942\ \mathrm{Dpa}_{it} + 34.636\ \mathrm{Dri}_{it} + 46.205\ \mathrm{Dsc}_{it} + 68.116\ \mathit{Dtn}_{it}$$
$$(1.89) \quad (4.22) \quad (2.28) \quad (4.59) \quad (3.50) \quad (1.20) \quad (3.01) \quad (4.46)$$

$$+28.972\ \mathrm{Dtx}_{it} + 20.499\ \mathrm{Dva}_{it} + 10.979\ \mathrm{Dwa}_{it} + 209.841\ \mathrm{smoke} + 118.24667\ \mathrm{overweight} + 0.0033\ g_{i04}$$
$$(3.05) \quad (1.17) \quad (0.79) \quad (3.73) \quad (2.78) \quad (1.23)$$

(Rural states of reference): (MT, ND, SD, VT, WV, WY) **adj.R^2_{04}= .3634**
States without statistically significant Ds_i;
i=AZ, CT, DC, ID, KS, NE, OR, UT, WI, IA, NH
States with statistically significant and positive Ds_i;
i=AL, AR, CA, DE, FL, GA, IL, IN, KY, LA, MA, MD, MI, MO, MS, NC, NJ, NV, NY, OH, OK, PA, RI, SC, TN, TX, VA, WA
States with statistically significant and negative Ds_i;
i=CO, NM, ME

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:

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

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,

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

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

Appendix B

See (Table B1) for details.

Appendix C

See (Table C1) for details.

Appendix D

See (Table D1) for details.

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