Air Pollution and Procyclical Mortality

Abstract

Prior research demonstrates that mortality rates increase during economic booms and decrease during economic busts. The procyclicality of environmental risks provides a possible mechanism for this pattern, but almost no analysis of this relationship has previously been conducted. We take a first step towards rectifying this by investigating the contribution of pollution to the procyclicality of deaths. We combine state-level data on overall, cause-specific, and age-specific mortality rates with state-level measures of two types of ambient pollution concentrations. After controlling for other demographic variables, state fixed-effects and general year effects, we find a significant positive correlation between carbon monoxide (CO) concentrations and mortality rates. Controlling for CO and emissions of particulate matter (PM10) attenuates the relationship between mortality and business cycles, measured by the unemployment rate, by more than half and renders it statistically insignificant. The attenuation is particularly large (more than 60 percent), although imprecisely measured, for fatalities from respiratory causes, but the reduction in estimated effects is also frequently substantial for other causes of death and for the mortality of persons who are not of prime working age.

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Health is conventionally believed to deteriorate during macroeconomic downturns. However, a substantial body of research conducted over the last decade instead suggests that physical health instead *improves* when the economy temporarily weakens. In particular, there is strong evidence of a procyclical variation in mortality, but the mechanisms for this relationship are poorly understood. Early research on this topic emphasized the role of individual behaviors, which may become healthier during weak economic periods because of increases in available time and reductions in income; however, recent analyses provide more mixed evidence on whether this occurs. There is also strong but limited role for changes in driving behavior and traffic fatalities but changes in other environmental risk factors have not been studied.

Air pollution, which is positively associated with short-term cyclical fluctuations in economic activity and so may be a source of procyclical fluctuations in mortality, has not been examined in this context, probably because the data required to do so are difficult to analyze. This study provides a step towards filling this gap in the literature by examining the extent to which controlling for pollution attenuates the estimated coefficient on unemployment rates (the proxy of macroeconomic conditions) in models that are otherwise similar to those used in previous related analyses. Specifically, using state-level data for 1982-2007, we incorporate data on ambient concentrations of two air pollutants (carbon monoxide and particulate matter) in models examining total, cause-specific and age-specific mortality, that also control for state fixed-effects and unemployment rates, general year effects and supplementary location-specific demographic characteristics.

We substantiate prior findings that mortality is procyclical: a one percentage point increase in the unemployment rate is associated with a 0.28% decrease in the total mortality rate. However, after controlling for pollution, the estimated effect declines to about -0.12%.

Particulate matter and carbon monoxide concentrations both exhibit a procyclical variation but only the latter is estimated to strongly increase mortality and so, among the pollutants that we measure, it is the inclusion of carbon monoxide that attenuates the estimated macroeconomic effect. Specifically, a one-standard deviation increase in the carbon monoxide concentration is associated with a 2.2% increase in the mortality rate, after controlling for state and year effects, demographic characteristics and particulate matter levels (but not unemployment rates), and its inclusion in the full model attenuates the estimated macroeconomic effect by 51 percent.

The estimates for specific causes and ages of death also provide suggestive evidence that environmental risks, like pollution, provide a mechanism for at least some of the procyclical fluctuation in mortality. In particular, previous research suggests pollution will have the largest effect on deaths from respiratory disease, and this is what we find. For instance, a one-standard deviation increase in the carbon monoxide concentration is associated with a 2.2% increase in this mortality rate, and the inclusion of the pollution concentrations in our main model attenuates the correlation between respiratory fatalities and unemployment rates an imprecisely estimated 60 percent. Controlling for air pollution emissions also leads to smaller reductions in the procyclicality of mortality from various types of cardiovascular and cerebrovascular disease, as well as accidental deaths. Conversely, suicides are countercyclical and unaffected by pollution. The results for age-specific mortality are also revealing. Consistent with recent findings by Miller et al. (2009), deaths are estimated to be procyclical for the young and old, but not for 20-54 year olds, who are of prime working age. However, carbon monoxide concentrations are associated with increased mortality for all groups, with the result that the procyclicality of fatalities is attenuated for youths and seniors – with a particularly strong reduction for infant

deaths – and the countercyclicality of deaths for 20-54 year olds becomes more pronounced when holding pollution levels constant.

I. Background

The relationship between macroeconomic conditions and health has been extensively examined using time series data for single geographic locations. Particularly influential have been the studies by M. Harvey Brenner and coauthors (e.g. Brenner, 1979) arguing that recessions increase mortality and health problems. However, many researchers (e.g. Gravelle, 1984) have pointed out serious flaws in Brenner's analysis. Recent time series analyses (e.g. McAvinchey, 1988; Joyce & Mocan ,1993; Laporte, 2004; Tapia Granados & Ionides, 2008) correct for some of these issues but, despite these innovations, the results remain ambiguous. Most time series research suggests that the contemporaneous effect of economic downturns is to improve health and reduce mortality, but some find countercyclical effects, no impact or variation across countries or time periods.[1] Such lack of robustness should not be surprising since any lengthy time series may yield biased estimates due to omitted variables that are spuriously correlated with economic conditions and affect health.[2]

Following Ruhm (2000), many recent studies address the omitted variables bias issue by analyzing data for multiple locations and points in time. The key advantage is that panel data techniques can then be used to control for many potential confounding factors. In particular, location-specific determinants of health that remain constant over time can be easily controlled for, as can factors that vary over time in a uniform manner across locations. Death rates, the most

[1] See Ruhm (forthcoming) for a full discussion of these issues and extensive references.
[2] For example, the variation in unemployment during the four decades (beginning in the 1930s) covered by much of Brenner's research is dominated by dramatic reductions in joblessness following the great depression, where mortality declined due to improved nutrition and increased availability of antibiotics.

common dependent variables, are useful to study because mortality represents the most severe negative health outcome, is objective and well measured, and diagnosis generally does not depend on access to the medical system (in contrast to many morbidities). However, death rates do not fully account for changes in non-life-threatening health conditions.

This research provides strong evidence of a procyclical fluctuation in total mortality and several specific causes of death, using disparate samples and time periods. A one-percentage point increase in the unemployment rate (the most common macroeconomic proxy) is typically associated with a 0.3% to 0.5% reduction in total mortality, corresponding to an elasticity of –.02 to –.05, but with significantly larger decreases sometimes obtained.[3]

In explaining why health improves during economic downturns, researchers have emphasized the role of changes in lifestyles. Specifically increased availability of non-market "leisure" time makes it less costly for individuals to undertake health-producing activities such as exercise and cooking meals at home, while lower incomes are associated with reductions in unhealthy lifestyles like smoking and drinking. The data provide some support for this mechanism. There is strong evidence that alcohol *sales* are procyclical and several studies (e.g. Ruhm, 1995; Freeman, 1999) find that alcohol-involved vehicle mortality declines in such periods. Cardiovascular fatalities, which are strongly influenced by lifestyles, are also procyclical, with variations of similar or larger magnitude (in percentage terms) than for total mortality (e.g. see Ruhm, 2000; Neumayer, 2004; Miller, et al., 2009), and with particularly strong effects for deaths due to coronary heart disease (Ruhm, 2007), that are likely to be

[3] Ruhm (forthcoming) provides a detailed discussion of this evidence. Due to severe data restrictions, few analyses examine how macroeconomic conditions affect morbidity. Ruhm (2003) finds that increased unemployment reduces the prevalence of medical conditions (particularly for acute health problems), restricted-activity and bed-days, and ischemic heart disease or intervertebral disk problems. This contrasts with an increase in non-psychotic mental disorders. Consistent with this last result, Charles and DeCicca (2008) uncover a procyclical variation in mental health for less-educated and African-American males.

responsive to short-term changes in modifiable health behaviors (but also some environmental risk factors).

There is also other evidence that behaviors become healthier during economic downturns. Ruhm (2005) finds that severe obesity, smoking and physical inactivity decline, with especially large reductions in multiple risk factors. Gruber & Frakes (2006) and Xu & Kaestner (2010) provide further evidence of a procyclical variation in smoking. Ruhm (2000) shows that the consumption of dietary fat falls while the intake of fruits and vegetables rises. Dehejia & Lleras-Muney (2004) indicate that pregnant mothers consume less alcohol, with mixed effects for smoking. Consistent with these patterns, evidence that higher time prices correlate with increased obesity has been provided for adults and children (e.g. Courtemanche, 2009), individuals spend more time socializing and caring for relatives when the economy is weak (Edwards, 2008), and Germans exercise more when wages temporarily decline (Dustmann & Windmeijer, 2004).

However, changes in health behaviors are probably not the sole, or necessarily the most important, mechanism for procyclical variations in mortality. In a provocative study, Miller et al. (2009) find that working age adults are responsible for relatively little of the cyclical variation in deaths, suggesting that behavioral responses to changes in labor market conditions are unlikely to be a dominant factor. Some research also raises questions about the strength or direction of the behavioral changes. For example, Böckerman et al. (2006) obtains a countercyclical variation in obesity for Finnish adults in some (but not all) models, as do Charles & DiCicca (2008) for some U.S. adult males and Arkes (2009) for teenage girls. Johansson, et al. (2006) indicate a countercyclical pattern of some sources alcohol-related mortality in Finland, as do Dávlos et al. (forthcoming) for alcohol abuse and dependence among U.S. adults, and Arkes (2007) for drug consumption among teenagers.

Environmental risks provide an important potential alternative explanation for why health may change with macroeconomic conditions. One such risk – traffic fatalities – has been widely studied, with substantial and robust evidence provided that a one point increase in unemployment reduces traffic deaths by 1% to 3% (see Ruhm, forthcoming, for citations.) This reflects both reductions in driving during hard economic times and reductions in fatalities per mile driven, partly because of less alcohol-involved driving (Cotti & Tefft, 2011).

However, other joint products of economic activity, air pollution in particular, also present health risks, especially for infants or senior citizens who do not participate in the labor force (Chay & Greenstone, 2003; Currie and Neidell, 2005). Pollution is a cause of death from sources such as cardiovascular and respiratory disease (e.g. see Peters et al., 2004), yet has received no attention in previous empirical research examining the effects of macroeconomic fluctuations on mortality. This analysis takes a first step towards rectifying this shortcoming.

II. Research Design

We analyze the relationship between macroeconomic conditions, air pollution emissions, and mortality rates, using panel data methods that, following Ruhm (2000) have now become standard in this literature.[4] Studies based on aggregate data usually estimate some variant of:

$$M_{jt} = \alpha_j + X_{jt}\beta + E_{jt}\gamma + \lambda_t + \varepsilon_{jt}, \qquad (1)$$

where M_{jt} is a health outcome (mortality rates here) in location j at time t, E measures macroeconomic conditions, X is a vector of covariates, α is a location-specific fixed-effect, λ a general time effect, and ε is the regression error term.

[4] Although alternative estimation models have some desirable features, we use "standard" models to maximize the comparability of our results to those obtained previously.

Unemployment rates are the most common primary proxy for macroeconomic conditions, and the one used here. The supplementary characteristics include controls for the age-structure of the local population, and the shares in specified education and race/ethnicity subgroups. The time period for analysis is 1982-2007. Detailed emissions data are unavailable prior to 1982, while location-specific mortality rates are not currently available after 2007. States have been the unit of analysis is most previous related U.S. research and are what we use here. In future work, we also plan to conduct a county-level analysis, which would be desirable since air pollution is often a relatively local phenomenon.

The year effects (λ_t) in equation (1) hold constant determinants of death that vary uniformly across locations over time (e.g. advances in widely used medical technologies or behavioral norms); the fixed-effects (α_j) account for those that differ across locations but are time-invariant (such as persistent lifestyle differences between residents of Nevada and Utah); and the impact of the macroeconomy is identified from within-location variations relative to the changes in other locations. The model above does *not* account for unobservable factors varying within states over time, but the inclusion of location-specific time trends often substantially rectifies this.[5] Although unemployment rates are the *proxy* for macroeconomic conditions, the macroeconomic effects need not be restricted to individuals who are changing employment status. For instance, increases in air pollution due to growth in economic output will affect the health of persons not in the labor force.

The basic model, described by equation (1), is supplemented in several ways for the current analysis. The primary econometric strategy is to first estimate (1), with $\hat{\gamma}$ then providing the overall macroeconomic effect. The augmented equation

[5] The impact of national business cycles, which could differ from more localized fluctuations, is absorbed by the time effects. Discussions of macroeconomic effects therefore refer to changes within locations rather than at the national level.

$$M_{jt} = \alpha_j + X_{jt}\beta + E_{jt}\gamma + P_{jt}\delta + \lambda_t + \varepsilon_{jt}, \qquad (2)$$

is then be estimated, where P_{jt} is the ambient pollution level at location j and time t. In this model, $\hat{\gamma}$ shows the partial effect of macroeconomic conditions after controlling for pollution levels and the degree to which it is attenuated, relative to the corresponding coefficient in (1), shows the extent to which pollution is a mediating factor in explaining the overall macroeconomic effects. $\hat{\delta}$, in (2), shows the direct effect of pollution, which are hypothesized to be positively related to mortality rates. These estimates are likely to provide a lower bound on the true effect of pollution to the extent that the latter is measured incompletely or with error. With this in mind, the equation is also estimated with the simultaneous inclusion of multiple pollution measures, while recognizing potential limitations on the interpretation of the coefficients on the individual proxies, if there is substantial multicollinearity between them.[6] We also estimate a first-stage model where pollution levels are the dependent variables and unemployment rates the key regressor, to confirm our hypothesis of a positive relationship between economic activity and emissions levels. Following most previous literature, we use the natural logarithm of mortality rates as the dependent variable and report robust standard errors that are clustered at the state level.[7]

III. Data

Three primary data sources are used for this investigation: pollution levels from the Environmental Protection Agency's *Air Quality System (AQS)* database; location-specific unemployment rates from U.S. Department of Labor's *Local Area Unemployment Statistics*

[6] The correlation coefficient between our state-level measures of CO and PM10 is 0.532.
[7] At the state level, there are no zero mortality counts, so the logarithm of the mortality rate is never undefined. With county-level analysis this may not be true, in which case, following Miller et al. (2009), Poisson (or negative binomial) count data models can be used as an alternative.

(LAUS) Database; mortality rates from the Center for Disease Control and Preventions' *Compressed Mortality Files (CMF)*. We also obtain data on state demographic characteristics from these sources and from the Current Population Survey (CPS) March Annual Demographic Survey.

The *AQS* data base (http://www.epa.gov/air/data/) contains measurements of air pollution concentrations from monitors in the 50 United States and the District of Columbia. Pollution measures are available for a large number of pollutants, but the two that we focus on here are carbon monoxide (CO) and particulate matter less than 10 microns in diameter (PM10). Both of these are among the six "criteria pollutants" designated by the Clean Air Act and are thus are widely accepted as having negative health effects. Because of this designation, data on both emissions and ambient concentrations is plentiful, relative to other pollutants. CO and PM10 were chosen among the criteria pollutants because of the large number of monitors measuring them in the AQS and because these pollutants have been previously found to be strongly associated with health problems and mortality. For instance, Currie and Neidell (2005) find a significant effect from CO exposure on infant mortality in California, while Chay and Greenstone (2003) find a correspondingly significant effect from particulate matter exposure.[8]

Data on CO concentrations are available from 1980 to 2010 from 1,463 pollution monitors, and those on PM10 from 1982 to 2010 from 4,502 monitors. For each monitor-year, the *AQS* provides summaries of air pollution measurements, including arithmetic and geometric means, percentiles and days above specified limit values. A challenge of using the *AQS* data is that they provide an unbalanced panel, since pollution monitors change over time. For instance, the median number of years that a CO monitor is in the data is seven, and only 65 CO monitors

[8] Chay and Greenstone examine total suspended particulates (TSPs), an older EPA designation that has been replaced by PM10 and PM2.5 (particles smaller than 2.5 microns in diameter).

(4.4%) are available all 31 years. Similarly, the median PM10 monitor is in the data for 6 years, and fewer than 1.2% are available in all 29 years of the panel. We account for monitors that enter or exit within a year in a manner described below.

Because each state's monitors are changing each year, considerable effort and experimentation were required to come up with meaningful location-specific pollution measures. For states with only one monitor, we use the annual arithmetic mean of that monitor's pollution concentration readings as the state-level measure. For states with multiple monitors, the state-level measure is a weighted average of each monitor's annual arithmetic mean, weighted by the population of the county in which it is located times the percent of total potential observations from a monitor that are actually observed (for instance, a monitor that only reports daily observations for one half of the year is discounted by 50%). After the aggregation, we are left with a dataset containing 1,484 state-year level observations of CO concentrations from 1980-2010 (not every state is represented in all years) and 1,337 observations of PM10 concentrations from 1982-2010 (for PM10, the number of states before 1985 ranges from just 2 to 15, and afterwards levels off with the majority of states).[9]

The *LAUS* data (http://www.bls.gov/lau/lauov.htm) come from a Federal-State cooperative effort in which monthly estimates of total employment and unemployment are prepared for approximately 7,300 areas including: census regions and divisions, states, metropolitan statistical areas, counties, and some cities. Concepts and definitions underlying the *LAUS* data come from the Current Population Survey (CPS), the household survey that is the

[9] Chay and Greenstone (2003, p.419-420) address the issue of the reliability of data from these pollution monitors, given that the monitors may be strategically placed by authorities to mislead about true environmental conditions. They appeal to the Code of Federal Regulations, which describes criteria that determine the siting of monitors. It appears that legislation specifically forbids this type of strategic siting and that the EPA can enforce this by overseeing and authorizing localities' monitor siting plans. However, given the high frequency of entrance and exit of monitors in our panel, it remains possible that these regulations are not in fact enforced.

official measure of the labor force for the nation. This analysis uses annual average unemployment rates for states as the key proxy for macroeconomic conditions. We have the unemployment rate for each state from 1981-2009 (i.e. we are missing just the first and the final year for which we have pollution data).

The *CMF* (http://www.cdc.gov/nchs/data_access/cmf.htm) include county- and state-level mortality and population counts. Data prior to 1988 are publically available while those from 1989 to 2007 were obtained through a special agreement with the CDC. The *CMF* include a record for every death of a U.S. resident, with source data condensed by retaining information on the state and county of residence, year (rather than exact date) of death, race and sex, Hispanic origin (after 1998), age group (16 categories), underlying cause of death (ICD codes and CDC recodes). The number of records is reduced in the *CMF* by aggregating those with identical values for all variables and adding a count variable indicating the number of such records. The file also contains population estimates, based on Census data, for US, State, and county resident populations, as well as for subsamples stratified by race, sex, Hispanic origin, and 13 age groups. The number of live-births is also included to permit the calculation of infant mortality rates.

The *CMF* mortality and population data are used to construct the dependent variables: total annual mortality rates and selected age-specific and cause-specific death rates. The outcomes analyzed include total mortality rates and mortality rates for eleven specific causes (respiratory, cardiovascular, acute myocardial infarction (heart attack), ischemic heart disease, cerebrovascular disease (stroke), cancer, accidents (total, vehicular, and non-vehicle), suicide, and homicide) and for six age groups (infants, 1-19, 20-54, 55-64, 65+, 85+). These are chosen for consistency with the previous literature, to test rigorously for differences across age-groups (since pollution affects groups with low or no participation in the labor force) and to distinguish

between sources of death expected to be strongly influenced by pollution levels (e.g. respiratory diseases) versus those anticipated to be unrelated to them (e.g. suicides).

State-year level demographic controls are obtained from a variety of sources. Data from the March *CPS* were used to provide state-level information on gender, race, and education population shares. Information from the U.S. Department of Transportation Highway Statistics Series (http://www.fhwa.dot.gov/policyinformation/statistics.cfm) are used to generate a state-year level measure of total miles driven per capita.

Our initial data set contains 1,309 state-year level observations from 1982-2007, with at least 48 states in each year and all 50 states and the District of Columbia in every year from 1985 on (except 1988, where data are available for 49 states plus DC). However, we restrict analysis to a panel of 1,109 state-year observations containing information on *both* PM10 and CO concentrations. The number of states represented from 1985 through 2007 varies from 42 to 49, but before 1985 there are never more than 14 states represented.[10] Summary statistics are presented in Table 1. PM10 concentrations are measured in micrograms per cubic meter ($\mu g/m^3$), and CO concentrations are measured in parts per million (ppm).

IV. Results

a. Macroeconomic Conditions and Pollution

Before investigating the relationship between mortality, pollution, and business cycles, we first test our hypothesis that ambient pollution is higher during economic booms and lower during recessions, a necessary first-stage for this to provide a mechanism for the procyclical fluctuation in death rates. Table 2 presents regression results where the dependent variables are standardized pollution measures (with mean zero and standard deviation one) for PM10

[10] All of the regression results presented below are robust to dropping observations from before 1985.

(columns 1 and 2) and CO (columns 3 and 4) emissions. All regressions are weighted by the state's population and include state-fixed effects and year-fixed effects (not reported). Columns 2 and 4 also state population shares in specified age, gender, race/ethnicity and education groups. The coefficients on these variables are shown in Appendix Table 1.

There is a significant negative relationship between the unemployment rate and both PM10 and CO emissions. A one percentage point increase in the unemployment rate is associated with about a one-tenth of a standard deviation decrease in the ambient PM10 concentration and around a one-fifteenth of a standard deviation reduction in ambient CO concentrations. Controlling for demographic measures modestly attenuates these correlations but the results continue to verify our expectation that emissions are procyclical.[11] Most coefficients on the demographic variables are statistically insignificant.

b. Total Mortality

We next begin the examination of our main question, which is whether pollution provides a possible mechanism for the procyclical variation in mortality. Table 3 summarizes the results of models where the dependent variable is the natural logarithm of the overall mortality rate. All models include controls for the demographic variables and state- and year-fixed effects (reported in Appendix Table 2), as well as on the unemployment rate – our proxy for macroeconomic conditions. The basic model, in column 1, verifies earlier findings by Ruhm (2000) and others showing that unemployment rates are negatively correlated with mortality. Specifically, a one percentage point increase in the unemployment rate is associated with a decrease in the total mortality rate of 0.3%. This is smaller than the 0.5% predicted reduction obtained by Ruhm (2000), but consistent with evidence recently presented by Stevens et al. (2011) showing that the

[11] Surprisingly, we can find few other papers directly investigating this issue. Heutel (forthcoming) documents the procyclicality of carbon dioxide (CO_2) emissions at the quarterly level. Using ARIMA regressions, he estimates the elasticity between U.S. GDP and CO_2 emissions levels to be between 0.5 and 0.9.

procyclicality of mortality is somewhat attenuated when adding post-1991 observations to the model.[12] Among the demographic coefficients, age has the expected effect on mortality, with higher shares of both infants and senior citizens being correlated with higher mortality rates. Race, gender, and education do not exhibit clear patterns, possibly because of they are highly correlated with the dominant age effects.

The reminder of Table 3 adds controls for pollution to the basic model. The standardized measure of PM10 concentrations is incorporated in model (2), that of CO concentrations in column (3) and both are included in the fourth specification. PM10 levels are neither highly predictive of mortality, nor do they substantially attenuate the predicted macroeconomic effect. For instance, the coefficient on PM10 in column (2) suggests that a one-standard-deviation increase in the concentration is associated with a statistically insignificant 0.5% increase in mortality, which is just one-fourth the size of the statistically significant CO concentration effect (2.3%) obtained in column (3). When both are included together (column 4), the coefficient on CO is roughly ten times the size of that on PM10 (2.2% vs, 0.2%). Given this pattern, we will primarily focus on the results for CO below, although we will control for both types of emissions in our estimation models.

Adding CO emissions to the model cuts the unemployment rate coefficient in half and when PM10 is also held constant the macroeconomic coefficient falls by 56%. Thus, in our basic specification (column 1), a one percentage point increase in the unemployment rate is predicted to reduce total mortality by 0.28% but this falls to 0.12% when both pollution concentrations are controlled for (column 4).[13]

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[12] Using data from 1978-2006 and a specification similar to that in column 1, they obtain an unemployment coefficient of -.0019.

[13] In Appendix Table 2, we also present a column that includes both pollution measures but not the unemployment rate. In that regression, the coefficients on all of the remaining right-hand-side variables are quite similar to those

c. Cause-specific mortality

These results just presented are consistent with the hypothesis that changes in air pollution levels provide a mechanism for the procyclical fluctuation in mortality. The evidence that the effects of pollution are causal would be strengthened if the unemployment coefficients are more sharply attenuated by their inclusion in the model for sources of fatalities (such as those from respiratory diseases) that we expect to be strongly related to emissions levels than for those (such as cancer deaths) where the relationship is anticipated to be weaker. We examine these possibilities next by extending the analysis to consider specific causes of death.

In Table 4 we examine deaths from respiratory, cardiovascular, and cerebrovascular diseases, from cancer, and from two subcategories of cardiovascular disease – ischemic heart disease and acute myocardial infarction (heart attacks). For each cause of death, we present the results of two specifications. The first (column a) is the basic model that controls for state unemployment rates, state and year effects, and demographic variables, but not air pollution. The second (column b) adds in the standardized pollution measures as right-hand-side variables. Mortality rates from all six causes of death are negatively correlated with the unemployment rate in the basic specification (column a) without pollution controls, although the association is significant for only three of the six. Heart attacks are the most procyclical – a one percentage point increase in the unemployment rate is predicted to decrease deaths from this source by more than two percent. A corresponding reduction in the unemployment rate is estimated to decrease deaths from cardiovascular disease and stroke by just under one percent. Cancer fatalities are unrelated to macroeconomic conditions, as has been found previously (Ruhm, 2000).

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that appear in Table 3. CO is positively correlated with mortality at the 5% level, and PM10 is insignificantly correlated with mortality. Currie and Neidell (2005) also examine the effect of both CO and PM10 on infant mortality, and in most specifications CO is found to be significant while PM10 is not.

The other columns (column b) include the pollution measures as controls. We expect pollution to have an effect on respiratory deaths, and probably on some of the cardiovascular categories. PM10 does not have a significant effect on any of these mortality rates, consistent with the results for total mortality obtained above. Conversely, ambient CO levels are significantly and positively associated with deaths from causes, cardiovascular disease, ischemic heart disease and stroke. A one-standard-deviation increase in CO concentrations is associated with a 3.7% increase in the respiratory mortality rate and a 1.4% increase in the cardiovascular mortality rate. This is consistent with earlier findings of an effect of pollution on these causes of death (Peters et. al. 2004).

Controlling for pollution attenuates the unemployment coefficient for deaths from respiratory causes by more than 60%, although the original effect is of small magnitude and imprecisely estimated. It attenuates the predicted effect on cardiovascular fatalities by a substantially smaller 8%, but when focusing on ischemic heart disease, which is likely to be more responsive to short-term triggers or changes in risk factors, the coefficient is attenuated by one-third. The estimated unemployment effect declines by around one-quarter for stroke deaths, when moving from the basic to augmented models barely at all when examining heart attack fatalities. This last result is surprising, since health attacks are an important component of ischemic heart disease.

Table 5 presents information on deaths from external causes including accidents (and separately for motor vehicle and other accidents), suicides, and homicides. Generally, we would not expect deaths from these causes to be strongly related to pollution levels. Death rates from

both motor vehicle and other accidents are procyclical, suicides are countercyclical, while homicides are unrelated to macroeconomic conditions.[14]

As expected, neither pollution measure is significantly correlated with the suicide or the homicide rate. However, PM10 concentrations are positively correlated with accidents, and CO emissions are marginally significant for deaths from non-vehicle accidents. We suspect that these pollution measures, especially PM10, are highly correlated with driving behavior in ways that our models have not fully accounted for, and note that including the pollution variables attenuates the unemployment coefficient on motor vehicle accidents by 11 percent, while switching the sign on the (insignificant) parameter estimate for non-vehicle accidents from negative to positive. To provide some further information on this issue, we added per-capita highway miles driven in our accident mortality regressions. Mileage was positively correlated with the vehicle death rate (and significant at the 1% level), but its inclusion did not reduce the predicted PM10 effect in this model.[15] One possibility, which we are unable to investigate, is that other aspects of driving behavior – like aggressive driving or speeding – are procyclical and positively correlated with both emissions and mortality, after controlling for miles driven.

d. Age-Specific Mortality

Miller et al. (2009) provides evidence that procyclical variations in mortality are particularly pronounced among the young and old – who are less likely to be directly involved in the labor market. Changes in pollution levels could explain some of these patterns, since the health of these groups might be particularly vulnerable to environmental risks while related

[14] These results are consistent with prior research findings (e.g. Ruhm, 2000), except that a procyclical variation in homicides has sometimes previously been found.

[15] The attenuation of the coefficient on unemployment in the regressions on vehicular accident mortality is almost exactly the same whether including the pollution measures (attenuated from –0.0266 to –0.0237) or including just miles per capita (attenuated from –0.0266 to –0.0239). When including both pollution measures and miles per capita, the coefficient on unemployment is attenuated to –0.0212.

health shocks of a given size might be more likely to result in death for them, if they are in initially more precarious health. We address these issues by using the *CMF* data to calculate mortality rates for six age groups: less than 1 year olds, 1-19 year olds, 20-54 year olds, 55-64 year olds, older than 64 year olds, and older than 84 year olds. Table 6 summarizes the results obtained when we regress (log) mortality rates on unemployment rates, demographic characteristics, and state and year effects, both with and without the pollution measures. Regressions are weighted by the state's population in each age category.

In the basic model (column a), unemployment is negatively correlated with the mortality rates of most age groups, with particularly strong procyclicality observed for infants and youths (under 20). Interestingly, a positive, although insignificant, predicted unemployment effect is found for prime-age (20-54 year old) individuals, and little relationship is obtained for those 85 or older, possibly because the relative small sample sizes reduce precision of the estimates.

When adding in the pollution measures (specification b), we see the hypothesized attenuation of the macroeconomic coefficients for the groups anticipated to be most vulnerable to these environmental risks – infants and older individuals. Specifically, the predicted unemployment rate effect declines, in absolute value, by 51%, 38%, and 38% for <1, 55-64 and ≥65 year olds, and becomes statistically insignificant in each of these cases. It is also particularly interesting to note that the large macroeconomic fluctuations in deaths of 1-19 year olds are *not* substantially affected by the inclusion of the emissions variables, which makes sense if these deaths occur for reasons that are largely unrelated to environmental risks.[16] Finally, we note that PM10 is not significantly related to the mortality rates of any of the groups, once the other

[16] Consistent with this, accidents, which are unlikely to be strongly related to pollution levels, were the leading cause of death in 2010 for 1-4, 5-14, and 15-24 year olds – accounting for 32%, 31% and 41% of all deaths for these groups in 2010 – but were much less important for infants or senior citizens, where they were responsible for 5% and 2% of such deaths (Murphy, et al., 2012).

variables are controlled for, whereas CO concentrations are predicted to increase death rates for all six age categories.

V. Discussion

Recent research indicates that mortality is procyclical – increasing during times of economic strength and declining during periods when the economy is weak. Surprisingly, this relationship is strong for the young and old, not for persons of prime-working age, suggesting that behavioral changes among prime-age adults are unlikely to fully explain these patterns.[17] A plausible alternative is that variations in environmental risks provide an important mechanism for the macroeconomic fluctuation in death rates. One of these risks, traffic fatalities, has been widely studied and is universally found to increase when the economy strengthens. However, a second such health risk – air pollution – is also likely to vary with the state of the economy but has not been previously studied. This analysis seeks to remedy this shortcoming by providing an initial investigation of how two types of emissions, carbon monoxide (CO) and particulate matter (PM10), vary with macroeconomic conditions, and how these emissions might explain the observed fluctuations in mortality rates.

Specifically, we use panel data methods and state level data for 1982-2007 to identify the effect of the macroeconomy (proxied by the unemployment rate) on mortality rates, with and without controls for ambient concentrations of CO and PM10. Consistent with the results of previous research, we uncover a significant negative correlation between state unemployment rates and total mortality rates in models that control for state demographic characteristics and fixed-effects, as well as general year effects. When adding the pollution measures to the model,

[17] However, some changes in their behaviors could affect other groups. For example, working age individuals may have more time to provide care to their young children or aged parents, during economic downturns, resulting in indirect health benefits for these groups.

the negative predicted unemployment rate effect is attenuated by 56%, consistent with an important role for air pollution. CO concentrations appear much more important than PM10 concentrations but we do not know whether this represents a difference in true health effects or in the accuracy with which these emissions are measured. This is consistent with Currie and Neidell (2005), which controls for both CO and PM10 concentrations (along with a third pollutant, ozone) and finds only significant effects on infant mortality from CO. However, Chay and Greenstone (2003) find a significant effect on infant mortality from total suspended particulates (TSP), a pollution designation that includes PM10.

We next extended the analysis to consider cause-specific and age-specific death rates. The results were largely (but not entirely) consistent with an important role for air pollution as a mechanism for explaining procyclical changes in mortality. In particular, we found that CO levels had large positive direct estimated effects on deaths from respiratory causes, ischemic heart disease and stroke, and that the inclusion of pollution controls substantially attenuated the unemployment rate coefficients in these models. However, the estimated effects of macroeconomic conditions were imprecisely estimated for several of these causes of death and for overall cardiovascular mortality and deaths from one key component of it – heart attacks – the attenuation was more modest. Pollution levels were also estimated to lead to unexplained reductions in accidental death rates, possibly because of unobserved confounding factors.

Finally, the direct effects of pollution and attenuation of the estimated macroeconomic effects was larger for the young and old, than for persons of prime-working age, which is what we would expect if pollution provides a causal mechanism, since the health of infants and seniors is likely to be particularly vulnerable to changes in emissions.

We view this research to be preliminary rather than definitive both because of some unexplained results described above and because many extensions of the research would be desirable. At the most basic level, we cannot say with confidence whether we are underestimating or overestimating the true effects of pollution. On the one hand, our pollution measures are quite crude and limited to just two of many types of emissions, which works in the direction of understating the true effects. In future work it would be useful to include controls for additional pollutants and to more fully specify the range of pollution outcomes. For example, rather than just examining ambient concentration levels, it might be useful to include information on fluctuations around the mean, peak levels and so forth. Also, our use of state level data is potentially problematic particularly since we obtain ambient concentration levels from a limited number of (local) pollution monitors. For this reason, a county level analysis might be useful. Second, even in cases where inclusion of the pollution measures attenuates the unemployment rate coefficients we cannot be sure that we are measuring a true causal mechanism rather than a spurious correlation between emissions and other unobserved factors. This may lead to an overstatement of the true effect of pollution and suggests that alternative strategies, such as the use of instrumental variables techniques, might be useful.[18]

Notwithstanding these caveats, or results suggest potential implications for policy. We certainly would not take our findings to imply that recessions are beneficial (although they may slightly less costly than is commonly understood), or claim that these results substantially affect arguments in favor of (or against) macroeconomic stabilization policies. However, they may indicate a role for policies that raise the costs of emitting pollutants during good economic times, since pollution levels rise when the economy strengthens. Interestingly, systems with tradable

[18] We have attempted instrumenting for pollution using a state-level instrument based on county attainment status, but preliminary results suggest that the instrument is very weak.

pollution permits are likely to accomplish this goal to at least some degree, since price will tend to increase with production levels.

Bibliography

Arkes J (2007) Does the Economy Affect Teenage Substance Use? Health Econ 16, 19-36.

Arkes J (2009) How the Economy Affects Teenage Weight. Soc Sci Med 68, 1943-1947.

Böckerman P, et al. (2006) Does a Slump Really Make You Thinner? Finnish Micro-Level Evidence 1978-2002. Health Econ 10(5), 9-13.

Brenner MH (1979) Mortality and the National Economy. Lancet 314 (8142), 568-73.

Charles KK, DeCicca P (2008) Local Labor Market Fluctuations and Health: Is there a Connection and For Whom? J Health Econ 27, 1532-1550.

Chay K, Greenstone M (2003) The Impact of Air Pollution on Infant Mortality: Evidence from Geographic Variation in Pollution Shocks Induced by A Recession. Q J Econ 118, 1121-67.

Cotti C, Tefft N (2011) Decomposing the Relationship Between Macroeconomic Conditions and Fatal Car Crashes During the Great Recession: Alcohol- and Non-Alcohol-Related Accidents. B.E. Journal of Economic Analysis and Policy 11(1 – Topics), Article 48.

Courtemanche C (2009) Longer Hours and Larger Waistlines? The Relationship Between Work Hours and Obesity, Forum for Health Economics and Policy 12 (2 – Obesity), Article 2.

Currie J, Neidell M (2005) Air Pollution and Infant Health: What Can We Learn from California's Recent Experience?, Quarterly Journal of Economics 120, 1003-1030.

Currie J, Neidell M, Schmieder J (2009) Air Pollution and Infant Health: Lessons from New Jersey, Journal of Health Economics 28, 688-703.

Dávlos M, et al. (forthcoming) Easing the Pain of an Economic Downturn: Macroeconomic Conditions and Excessive Alcohol Consumption. Health Econ.

Dehejia R, Lleras-Muney A (2004) Booms, Busts, & Babies' Health. Q J Econ 119, 1091-1130.

Dustmann C, Windeijer F (2004) Wages and the Demand for Health – A Lifecycle Analysis. Mimeo, University College London, May.

Edwards R (2008) American Time Use Over the Business Cycle. Mimeo, CUNY.

Freeman DG (1999) A Note on 'Economic Conditions and Alcohol Problems. J Health Econ 18, 661-70.

Gravelle HSE (1984) Time Series Analysis of Mortality and Unemployment. J Health Econ 3, 297-305.

Gruber J, Frakes M (2006) Does Falling Smoking Lead to Rising Obesity? J Health Econ 25, 183-197.

Heutel, G. (forthcoming) How Should Environmental Policy Respond to Business Cycles? Optimal Policy under Persistent Productivity Shocks. Review of Economic Dynamics.

Johansson E, et al. (2006) Alcohol-Related Mortality, Drinking Behavior and Business Cycles' Eur J Health Econ 7, 215-220.

Joyce TH, Mocan HN (1993) Unemployment and Infant Health: Time-Series Evidence from the State of Tennessee. J Hum Res 28, 185-203.

Laporte A (2004) Do Economic Cycles Have a Permanent Effect on Population Health? Revisiting the Brenner Hypothesis. Health Econ 13, 767-79.

McAvinchey ID (1988) A Comparison of Unemployment, Income and Mortality Interaction for five European Countries. Appl Econ, 20 (4), 453-71.

Miller DL, et al. (2009) Why Are Recessions Good for Health. Am Econ Rev 99, 122-127.

Murphy, Sherry L, Jiaquan Xu and Kenneth D. Kochanek (2012) Deaths: Preliminary Data for 2010. *National Vital Statistics Reports* 60(4), January 11.

Neumayer E (2004) Recessions Lower (Some) Mortality Rates. Soc Sci Med 58, 1037-47.

Peters A, et al. (2004) Exposure to Traffic and the Onset of Acute Myocardial Infarction. New Engl J Med 351(17), 1721-30.

Ruhm CJ (1995) Economic Conditions and Alcohol Problems. J Health Econ 14, 583-603.

Ruhm CJ (2000) Are Recessions Good For Your Health? Q J Econ 115, 617-50.

Ruhm, Christopher J. (2003), 'Good Times Make You Sick', Journal of Health Economics, 22 (4), 637-658.

Ruhm CJ (2005) Healthy Living in Hard Times. J Health Econ 24, 341-63.

Ruhm CJ (2007) A Healthy Economy Can Break Your Heart', Demography 44, 829-848.

Ruhm, CJ (forthcoming) Understanding the Relationship Between Macroeconomic Conditions and Health. In AM Jones (ed.) Elgar Companion to Health Economics, 2nd Edition. Cheltenham, UK: Edward Elgar.

Stevens, AH, DL Miller, M Page and M Filipski (2011) The Best of Times, the Worst of Times: Understanding Pro-cyclical Mortality, National Bureau of Economic Research Working Ppaer No. 17657, December.

Tapia Granados JA, Ionides EL (2008) Reversal of the Relationship Between Economic Growth and Health Progress: Sweden in the 19th and 20th Centuries. J Health Econ 27, 544-563.

Xu X, Kaestner R (2010) The Business Cycle and Health Behaviors. NBER Work Pap 15737.

Table 1: Sample Means for Selected Variables

	Mean	Standard Error
Emissions		
PM10 concentration ($\mu g/m^3$)	27.877	0.228
CO concentration (ppm)	0.933	0.013
State unemployment rate (%)	5.466	0.049
Mortality Rates (per 1000)		
Total	8.594	0.04
Respiratory	0.733	0.005
Cardiovascular	3.382	0.023
Acute Myocardial Infarction (Heart Attack)	0.775	0.009
Ischemic Heart Disease	1.344	0.022
Cerebrovascular Disease (Stroke)	0.576	0.004
Cancer	1.975	0.01
Accident	0.396	0.003
Vehicle Accident	0.178	0.002
Non-vehicle Accident	0.218	0.002
Suicide	0.126	0.001
Homicide	0.073	0.002
< 1 year old	8.418	0.075
1-19 years old	0.41	0.004
20-54 years old	2.246	0.016
55-64 years old	10.764	0.063
≥ 65 years old	50.253	0.122
≥ 85 years old	150.251	0.304
State Population Shares		
< 1 year old	0.014	0.000
1-19 years old	0.272	0.001
20-54 years old	0.499	0.001
55-64 years old	0.090	0.000
≥ 65 years old	0.124	0.001
Female	0.514	0.000
Black (non-Hispanic)	0.117	0.004
Other nonwhite (non-Hispanic)	0.054	0.003
Hispanic	0.066	0.003
High school incomplete	0.180	0.002
High school graduate/12th grade completed	0.356	0.002
Some college/<4 years completed	0.229	0.002
College graduate/4+ years completed	0.236	0.002

Note: Summary statistics are over the state-year observations, from 1982-2007, including only those 1109 observations for which we have PM10 and CO concentrations.

Table 2: Relationship between Pollution and Unemployment Rates

	State Emissions Level			
Regressor	PM 10 (1)	PM 10 (2)	CO (3)	CO (4)
State unemployment rate (%)	-0.0959**	-0.0827**	-0.0709**	-0.0628**
	(0.0429)	(0.0323)	(0.0311)	(0.0281)
Demographic controls?	No	Yes	No	Yes
Observations	1,109	1,109	1,109	1,109
R-squared	0.798	0.819	0.854	0.877

Note: *** p<0.01, ** p<0.05, * p<0.1. Standard errors clustered at the state are in parentheses. Dependent variable is the ambient pollution measure (PM10 or CO), standardized (mean zero, standard deviation one). State- and year-fixed effects are included but not reported. Regressions are weighted by state population.

Table 3: Econometric Estimates of the Determinants of Total Mortality

Regressor	(1)	(2)	(3)	(4)
State unemployment rate (%)	-0.00276*	-0.00234	-0.00136	-0.00121
	(0.00157)	(0.00148)	(0.00139)	(0.00139)
PM10		0.00505		0.00215
		(0.00402)		(0.00307)
CO			0.0224**	0.0218**
			(0.0110)	(0.0108)
Demographic controls?	Yes	Yes	Yes	Yes
Observations	1,109	1,109	1,109	1,109
R-squared	0.980	0.980	0.982	0.982

Note: *** p<0.01, ** p<0.05, * p<0.1. The dependent variable is the natural log of the total mortality rate. Standard errors clustered at the state are in parentheses. State- and year-fixed effects are included but not reported. Regressions are weighted by state population.

Table 4: Econometric Estimates of the Determinants of Mortality from Specific Diseases

Regressor	Respiratory		Cardiovascular		Heart Attack	
	(a)	(b)	(a)	(b)	(a)	(b)
State unemployment rate (%)	-0.00337	-0.00133	-0.00769***	-0.00711***	-0.0225***	-0.0213***
	(0.00419)	(0.00352)	(0.00228)	(0.00245)	(0.00514)	(0.00436)
PM10		-0.00328		-0.00402		-0.0114
		(0.00620)		(0.00385)		(0.0110)
CO		0.0368**		0.0144*		0.0341
		(0.0141)		(0.00780)		(0.0213)
	Ischemic Heart Disease		**Stroke**		**Cancer**	
	(a)	(b)	(a)	(b)	(a)	(b)
State unemployment rate (%)	-0.00873	-0.00593	-0.00923**	-0.00709**	-0.000258	0.000732
	(0.00777)	(0.00820)	(0.00388)	(0.00317)	(0.00126)	(0.00109)
PM10		-0.0119		-0.00468		0.00154
		(0.0156)		(0.00636)		(0.00272)
CO		0.0601**		0.0403*		0.0137*
		(0.0277)		(0.0210)		(0.00708)

Note: *** p<0.01, ** p<0.05, * p<0.1. Dependent variables are the natural logs of the specified cause-specific mortality rate. Standard errors clustered at the state are in parentheses. State- and year-fixed effects and all demographic controls shown in Table 2 are included here in all columns but not reported (n=1109). Regressions are weighted by state population.

Table 5: Econometric Estimates of the Determinants of External Causes of Death

Regressor	Accident		Vehicle Accident		Non-Vehicle Accident	
	(a)	(b)	(a)	(b)	(a)	(b)
State unemployment rate (%)	-0.00994**	-0.00580	-0.0266***	-0.0237***	-0.000333	0.00501
	(0.00428)	(0.00411)	(0.00420)	(0.00446)	(0.00664)	(0.00617)
PM10		0.0303***		0.0227**		0.0322***
		(0.00955)		(0.00990)		(0.0108)
CO		0.0260		0.0162		0.0426*
		(0.0166)		(0.0135)		(0.0227)
	Suicide		**Homicide**			
	(a)	(b)	(a)	(b)		
State unemployment rate (%)	0.0194***	0.0198***	0.00636	0.00810		
	(0.00566)	(0.00528)	(0.0117)	(0.00970)		
PM10		0.00751		-0.00642		
		(0.00749)		(0.0248)		
CO		-0.00241		0.0362		
		(0.0121)		(0.0498)		

Note: *** p<0.01, ** p<0.05, * p<0.1. Dependent variables are the natural logs of the specified cause-specific mortality rate. Standard errors clustered at the state are in parentheses. State- and year-fixed effects and all demographic controls shown in Table 2 are included here in all columns but not reported (n=1109). Regressions are weighted by state population.

Table 6: Econometric Estimates of Determinants of Age-Specific Mortality

Regressor	< 1 Year Olds		1-19 Year Olds		20-54 Year Olds	
	(a)	(b)	(a)	(b)	(a)	(b)
State unemployment rate (%)	-0.00629*	-0.00306	-0.0148***	-0.0136***	0.00436	0.00815**
	(0.00353)	(0.00326)	(0.00452)	(0.00421)	(0.00395)	(0.00328)
PM10		0.00594		-0.00170		0.00641
		(0.00552)		(0.00801)		(0.00823)
CO		0.0420***		0.0218*		0.0522*
		(0.0142)		(0.0129)		(0.0287)
VARIABLES	55-64 Year Olds		≥65 Year Olds		≥85 Year Olds	
	(a)	(b)	(a)	(b)	(a)	(b)
State unemployment rate (%)	-0.00405*	-0.00253	-0.00290**	-0.00181	0.00146	0.00214
	(0.00216)	(0.00206)	(0.00130)	(0.00120)	(0.00255)	(0.00238)
PM10		0.000861		0.000135		0.000389
		(0.00436)		(0.00222)		(0.00242)
CO		0.0229**		0.0167*		0.0106**
		(0.00944)		(0.00831)		(0.00522)

Note: *** p<0.01, ** p<0.05, * p<0.1. Dependent variables are the natural logs of the specified age-specific mortality rate. Standard errors clustered at the state are in parentheses. State- and year-fixed effects and all demographic controls shown in Table 2 are included here in all columns but not reported (n=1109). Regressions are weighted by state population within each age group.

Appendix Table A1: Relationship between Pollution and Unemployment Rates

	State Emissions Level			
Regressor	PM 10	PM 10	CO	CO
	(1)	(2)	(3)	(4)
State unemployment rate (%)	-0.0959**	-0.0827**	-0.0709**	-0.0628**
	(0.0429)	(0.0323)	(0.0311)	(0.0281)
share of population <1 year old		101.0		54.59
		(60.66)		(51.15)
share of population 1-19 year old		-16.57*		2.123
		(9.785)		(5.583)
share of population 55-64 year old		-31.78**		1.972
		(12.21)		(16.17)
share of population >=65 year old		13.72		0.877
		(11.77)		(13.51)
share female		-0.586		0.981
		(2.493)		(2.226)
share black		0.169		-2.908
		(1.863)		(1.933)
share other nonwhite		-5.514*		-7.241***
		(3.180)		(2.625)
share Hispanic		3.129		1.423
		(2.648)		(1.555)
share high school graduate/12th grade completed (age 25+)		-0.559		2.879
		(1.928)		(2.121)
share some college/<4 years completed (age 25+)		2.162		5.852***
		(2.051)		(1.997)
share college graduate/4+ years completed (age 25+)		2.261		1.732
		(2.133)		(1.862)
Constant	1.969***	6.672*	1.531***	-2.214
	(0.679)	(3.784)	(0.385)	(4.714)
Observations	1,109	1,109	1,109	1,109
R-squared	0.798	0.819	0.854	0.877

Note: *** p<0.01, ** p<0.05, * p<0.1. Standard errors clustered at the state are in parentheses. Dependent variable is the ambient pollution measure (PM10 or CO), standardized (mean zero, standard deviation one). State- and year-fixed effects are included but not reported. All regressors other than unemployment rate refer to state population shares. Regressions are weighted by state population.

Appendix Table A2: Econometric Estimates of the Determinants of Total Mortality

Regressor	(1)	(2)	(3)	(4)	(5)
State unemployment rate (%)	-0.00276*	-0.00234	-0.00136	-0.00121	
	(0.00157)	(0.00148)	(0.00139)	(0.00139)	
PM10		0.00505		0.00215	0.00238
		(0.00402)		(0.00307)	(0.00306)
CO			0.0224**	0.0218**	0.0222**
			(0.0110)	(0.0108)	(0.0107)
share of population <1 year old	9.814**	9.303**	8.592**	8.407**	7.870*
	(4.792)	(4.426)	(4.005)	(3.856)	(3.952)
share of population 1-19 year old	-1.212***	-1.128***	-1.259***	-1.223***	-1.294***
	(0.301)	(0.304)	(0.346)	(0.342)	(0.334)
share of population 55-64 year old	2.565*	2.726*	2.521**	2.591**	2.575**
	(1.286)	(1.357)	(1.195)	(1.237)	(1.240)
share of population >=65 year old	4.625***	4.556***	4.606***	4.577***	4.541***
	(0.839)	(0.811)	(0.741)	(0.734)	(0.746)
share female	0.157	0.160	0.135	0.137	0.149
	(0.176)	(0.174)	(0.154)	(0.154)	(0.149)
share black	-0.0998	-0.101	-0.0347	-0.0367	-0.0397
	(0.188)	(0.187)	(0.127)	(0.128)	(0.127)
share other nonwhite	-0.438	-0.410	-0.276	-0.268	-0.268
	(0.263)	(0.253)	(0.208)	(0.209)	(0.210)
share Hispanic	-0.0531	-0.0690	-0.0850	-0.0909	-0.0894
	(0.139)	(0.132)	(0.108)	(0.105)	(0.105)
share high school graduate/12th grade completed (age 25+)	0.0257	0.0285	-0.0388	-0.0359	-0.0314
	(0.0967)	(0.0952)	(0.103)	(0.101)	(0.103)
share some college/<4 years completed (age 25+)	0.233*	0.223*	0.103	0.101	0.0977
	(0.125)	(0.120)	(0.0950)	(0.0954)	(0.0962)
share college graduate/4+ years completed (age 25+)	-0.0240	-0.0354	-0.0628	-0.0666	-0.0627
	(0.114)	(0.111)	(0.106)	(0.106)	(0.107)
Observations	1,109	1,109	1,109	1,109	1,109
R-squared	0.980	0.980	0.982	0.982	0.982

Note: *** p<0.01, ** p<0.05, * p<0.1. The dependent variable is the natural log of the total mortality rate. Standard errors clustered at the state in parentheses. State- and year-fixed effects are included but not reported. Regressions are weighted by state population.