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ABSTRACT

Prior research demonstrates that mortality rates increase during economic booms and decrease during economic busts, but little analysis has been conducted investigating the role of environmental risks as potential mechanisms for this relationship. We investigate the contribution of air pollution to the procyclicality of deaths by combining state-level data on overall, cause-specific, and age-specific mortality rates with state-level measures of ambient concentrations of three types of pollutants and the unemployment rate. After controlling for demographic variables and state and year fixed-effects, we find a significant positive correlation between carbon monoxide (CO) concentrations and mortality rates. Controlling for CO, particulate matter (PM10), and ozone (O3) attenuates the relationship between overall mortality and the unemployment rate by 30 percent. The attenuation is particularly large, although imprecisely measured, for fatalities from respiratory diseases and is frequently substantial for age groups unlikely to be involved in the labor market. Our results are consistent with those of other studies in the economics and public health literatures measuring the mortality effects of air pollution.

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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 slack 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 a strong but limited role for changes in driving behavior and traffic fatalities, but changes in environmental risk factors have not been studied.

Air pollution, which increases when the economy strengthens 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 first step towards filling this gap 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 1982-2009, we incorporate information on ambient concentrations of three air pollutants – carbon monoxide (CO), particulate matter less than 10 microns in diameter (PM10), and ozone (O_3) – into models that examine total, cause-specific, and age-specific mortality, while also controlling for state fixed-effects and unemployment rates, general year effects and supplementary location-specific demographic characteristics.

We substantiate prior findings that mortality is procyclical over the period studied: a one percentage point increase in unemployment is associated with a 0.35% decrease in the total mortality rate. However, after controlling for pollution, the estimated effect declines to 0.24%.

All three pollutant concentrations exhibit a procyclical variation, but only CO is estimated to strongly increase mortality, and it is the inclusion of CO that attenuates the estimated macroeconomic effect. Specifically, a one-standard deviation increase in the CO concentration is associated with a 2.0% increase in the mortality rate, after controlling for state and year effects, demographic characteristics and PM10 and O_3 levels (but not unemployment rates), and its inclusion in the full model attenuates the estimated unemployment coefficient by 30%.

The results for specific causes and ages of death 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 has a significant effect on deaths from respiratory and cardiovascular disease, and this is what we also find.[1] In our estimates, a one-standard deviation increase in the CO concentration is associated with a 5.2% rise in the respiratory mortality rate, and the inclusion of the pollution concentrations in our main model eliminates the correlation between respiratory fatalities and unemployment rates (although the estimates are imprecise). Adding controls for air pollution also attenuates (by smaller amounts) the unemployment rate coefficients for various types of cardiovascular and cerebrovascular disease, as well as for 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) and Stevens et al. (2011), 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, CO concentrations are associated with increased mortality for all groups. As a result, the procyclicality of fatalities is attenuated for youths and seniors – with a particularly strong

[1] For instance, Clancy et. al. (2002) identifies an association between particulate matter concentrations and respiratory and cardiovascular deaths, and Peters et. al. (2004) links traffic pollution exposure to heart attacks.

reduction for infant deaths – and the countercyclicality of deaths for 20-54 year olds becomes more pronounced when controlling for pollution levels.

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.[2] 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.[3]

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 accounted for, as can factors that vary over time in a uniform manner across locations. Death rates, the most

[2] See Ruhm (2012) for a full discussion of these issues and extensive references.
[3] For example, the variation in unemployment during the four decades (beginning in the 1930s) covered by much of Brenner's research was 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, changes in non-life-threatening health conditions may not be fully accounted for.

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 overall mortality, corresponding to an elasticity of –.02 to –.05, with significantly larger elasticity estimates sometimes obtained.[4]

In explaining why health improves during economic downturns, researchers have emphasized the role of changes in lifestyles, hypothesizing that 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 these mechanisms. There is strong evidence that alcohol *sales* are procyclical and several studies (Ruhm, 1995; Freeman, 1999; Cotti & Tefft, 2011) 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 (Ruhm, 2000; Neumayer, 2004; Miller, et al., 2009), and with particularly large

[4] Ruhm (2012) 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, as well as 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.

effects for deaths due to coronary heart disease (Ruhm, 2007), that are likely to be responsive to short-term changes in modifiable health behaviors (but also some environmental risk factors).

Other behaviors may also become healthier when economic conditions weaken. 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 lifestyle changes. For example, Böckerman et al. (2006) obtain 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, Dávlos et al. (2012) likewise do so for alcohol abuse and dependence among U.S. adults, Arkes (2007) for drug consumption among teenagers, while Colman & Dave (2011) suggest that increases in leisure-

time exercise during periods of economic weakness are more than offset by reductions in work-related physical exertion.

Other risk factors provide potential alternative explanations for why health may improve during economic downturns. One such risk – traffic fatalities – has been widely studied, with substantial and robust evidence provided that a one point increase in unemployment reduces such deaths by 1% to 3% (see Ruhm, 2012, for citations.) This reflects both decreases in driving during hard economic times and fewer fatalities per mile driven, partly because of less alcohol-involved driving (Cotti & Tefft, 2011).

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; Currie et. al. 2009). Pollution is an established contributor to cardiovascular deaths (Peters et al., 2004) but has not yet received attention in 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, and mortality rates, using panel data methods that, following Ruhm (2000), have become standard in this literature.[5] Studies based on aggregate data usually estimate some variant of:

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

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

[5] Although alternative estimation procedures have some desirable features, we use "standard" models to maximize the comparability of our results to those obtained in previous research.

general time effect, and ε is the regression error term. Following most previous literature, the natural log (rather than level) of mortality rates is used, and we report robust standard errors that are clustered at the state level.

Unemployment rates are the most common primary proxy for macroeconomic conditions, and the one focused upon (although we discuss some results using alternative measures). The supplementary characteristics include controls for the age-structure of the local population, and the shares in specified education and race/ethnicity subgroups, as well as location-specific measures of temperature and precipitation. The analysis covers 1982-2009. Detailed emissions data are unavailable prior to 1982 and location-specific mortality rates after 2009 (at the time of analysis). States have been the unit of observation is most previous related U.S. research, and are what we focus upon, although we also briefly summarize the results of a county-level analysis.[6]

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 state-fixed-effects, α_j, account for differences across locations that are time-invariant (such as persistent lifestyle disparities between residents of Nevada and Utah). The impact of the macroeconomy is then identified from within-location variations relative to the changes in other locations. Equation (1) does *not* account for unobservable factors that vary over time within states.[7] Although unemployment rates are the *proxy* for macroeconomic conditions, the mortality effects need not be restricted to individuals changing employment status. For instance, increases in air pollution due to growth in economic output may particularly affect the health of infants and senior citizens, who are not in the labor force.

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[6] Recent related research using county or MSA level data includes Charles & DeCicca (2008) and An & Liu (2012).
[7] 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.

The primary econometric strategy in the current analysis is to first estimate equation (1), with $\hat{\gamma}$ providing the overall macroeconomic effect, and then to run the augmented model:

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

where P_{jt} is the ambient pollution level at location *j* and time *t*. In this specification, $\hat{\gamma}'$ shows the partial effect of macroeconomic conditions after controlling for pollution levels, and the degree of attenuation, relative to $\hat{\gamma}$ from equation (1), shows the extent to which pollution is a mediating factor in explaining the overall macroeconomic effect. The direct impact of pollution, which is hypothesized to raise mortality, is estimated as $\hat{\delta}'$ in equation (2). This is likely to provide a lower bound on the true effect to the extent that pollution is only partially controlled for, or measured with error. For this reason, equation (2) is also estimated with the simultaneous inclusion of multiple pollution measures, while recognizing that multicollinearity may limit our ability to interpret the coefficients obtained for the individual emissions variables.[8] We also estimate first-stage models where pollution levels are the dependent variables and unemployment rates the key regressors, to confirm our hypothesis of a positive relationship between economic activity and emissions levels.

III. Data

Three primary data sources are used for this investigation: pollution levels from the Environmental Protection Agency's *Air Quality System (AQS)* database, unemployment rates from U.S. Department of Labor's *Local Area Unemployment Statistics (LAUS) Database*, and mortality rates from the Centers for Disease Control and Prevention's *Compressed Mortality*

[8] The correlation coefficient between our state-level measures of CO and PM10 is 0.582; between O_3 and PM10 it is 0.293; and between CO and O_3 it is 0.084.

Files (CMF). We also used additional sources, described below, to obtain data on state demographic and weather characteristics.

The *AQS* database (http://www.epa.gov/air/data/) contains air pollution concentration data from monitors in the 50 United States and the District of Columbia. Measures are available for a large number of pollutants, but the three that we focus on are carbon monoxide (CO), particulate matter less than 10 microns in diameter (PM10), and ozone (O_3). Each are among the six "criteria pollutants" designated by the Clean Air Act and are thus widely accepted as having negative health effects. CO, PM10, and O_3 were chosen among the criteria pollutants because of the large number of monitors measuring them in the AQS and because these pollutants have been linked with health problems and mortality in previous research.[9] For instance, Currie and Neidell (2005) and Currie et. al. (2009) find infant mortality is positively and significantly related to CO exposure, while Chay and Greenstone (2003) find a correspondingly significant effect for particulate matter.[10]

Data on CO concentrations were available from a total of 1,470 monitors over the 1980 to 2010 timespan; there were 4,144 monitors for PM10 between 1982 and 2010, and 2,799 O_3 monitors from 1980 to 2010. For each monitor-year, the *AQS* provides summaries of air pollution measurements, including arithmetic and geometric means, percentiles and days above specified limit values.[11] A challenge of using the *AQS* is that it provides an unbalanced panel, since pollution monitors change over time. For instance, the median CO monitor was only in the data for seven years, and just 65 CO monitors (4.4%) were available all 31 years. Similarly, the

[9] We also attempted to examine PM2.5 (particles smaller than 2.5 microns in diameter) but were unable to do so because of the small amount of monitoring (no more than 40 monitors annually) prior to 1999.
[10] Chay and Greenstone examine total suspended particulates (TSPs), an older EPA designation that has been replaced by PM10 and PM2.5.
[11] We use only monitors reporting CO or O_3 concentrations at an hourly duration, and PM10 concentrations for a 24-hour duration. These are the most commonly used durations for the respective pollutants.

median PM10 monitor was in the data for six years, and fewer than 1.2% were available in all 29 years. We account for monitors that enter or exit within a year in a manner described below.

Because each state's monitors are changing over time, considerable effort and experimentation were required to come up with meaningful location-specific pollution measures.[12] For states with only one monitor, we used the annual arithmetic mean of that monitor's pollution concentration readings as the state-level measure. For states with multiple monitors, we used 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 the monitor that were actually observed. For instance, a monitor that only reported daily observations for one half of the year was discounted by 50%. We dropped observations from 2010 and before 1982, since we lack mortality data for those years. After doing so, we are left with a dataset spanning 1982-2009 and containing 1,333 state-year level observations of CO concentrations (not every state is represented in all years), 1,270 observations of PM10 concentrations, and 1,378 observations of O_3 concentrations.[13] Our pollution concentration measures certainly contain errors because we are attempting to identify average levels for the entire state using monitors for a limited set of locations. We discuss alternative measures below, when describing our county-level analysis.

The *LAUS* data (http://www.bls.gov/lau/lauov.htm) came from a Federal-State cooperative effort in which monthly estimates of total employment and unemployment are

[12] Currie and Neidell (2005) use data just from California monitors, and their results are unaffected by whether or not they use the subsample of monitors that are in the panel for the entire period or whether they use the entire unbalanced panel (see their footnote 7).

[13] PM10 monitoring is quite limited in the early years, with between 2 and 15 states having monitors prior to 1985, but with the majority of states doing so in subsequent years. Chay and Greenstone (2003, p.419-420) address the issue of whether the monitors may be strategically placed by authorities to mislead about true environmental conditions. They note that the Code of Federal Regulations, which describes criteria that determine the siting of monitors, 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 frequency of entrance and exit of monitors in our panel, it remains possible that these regulations are not fully enforced.

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 official measure of the labor force for the nation. This analysis uses annual average state unemployment rates as the key proxy for macroeconomic conditions. State unemployment rates are available throughout the entire analysis period. County level unemployment rates are comparable over time starting in 1990.

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 2009 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.

Using the *CMF* mortality and population data, we constructed dependent variables: annual total mortality rates; annual mortality rates from 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 mortality rates for seven age groups – infants, 1-19, 20-54, 55-64, 65-74, 75-

84, and ≥85 year olds. These outcomes were chosen for consistency with the previous literature, to test rigorously for differences across age-categories (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 were obtained from a variety of sources. Multiple years of the Current Population Survey (CPS) March Annual Demographic Survey were used to provide state-level information on gender, race, and education population shares. Specifically, our regression models control for the share of the state population who were: female, black, other nonwhite, Hispanic and aged <1, 1-19, 55-64, 65-74, 75-84 and ≥85 years old. In addition, the estimates held constant the share of persons aged 25 and over in the state who were high school graduates (without college), had attended college, and who were college graduates. A state-year level measure of total highway miles driven per capita was generated with data obtained from the U.S. Department of Transportation Highway Statistics Series (http://www.fhwa.dot.gov/policyinformation/statistics.cfm). Using weather data from the National Climatic Data Center at the National Oceanic and Atmospheric Administration (http://www.ncdc.noaa.gov/temp-and-precip/time-series/), we constructed measures of average temperature and precipitation.[14]

We restrict our analysis to the 1,147 state-year observations from 1982-2009 containing information on all three pollutants. The number of states represented from 1985 through 2009 varied from 40 to 47, but before 1985 there were never more than 13 states represented.[15]

[14] These data are not available for Hawaii and so we omit it from our analysis. We also estimated and obtained robust results for models that also controlled for heating and cooling degree days and the Palmer drought severity index, for which data were available in all locations except Hawaii, Alaska and Washington DC.

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

Summary statistics are presented in Table 1. PM10 concentrations are measured in micrograms per cubic meter ($\mu g/m^3$), and CO and O_3 concentrations in parts per million (ppm).

IV. Results

a. Macroeconomic Conditions and Pollution

We begin the econometric analysis by testing whether ambient pollution rises during economic booms and falls during downturns, 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 (columns 1 and 2), CO (columns 3 and 4), and O_3 (columns 5 and 6). All regressions are weighted by the state's population and include state-fixed effects and year-fixed effects (not reported). Columns 2, 4, and 6 also control for state population shares in specified age, gender, race/ethnicity and education groups. The coefficients on these variables are shown in Appendix Table A.1.

As hypothesized, there is a significant negative relationship between the unemployment rate and all three pollution measures. 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 about a one-fifteenth of a standard deviation reduction in ambient CO and O_3 concentrations. Controlling for demographic measures modestly attenuates these correlations for PM10 and CO, while the coefficient magnitude for ozone falls by half. Nevertheless, all of the parameter estimates differ significantly from zero, and the results verify our expectation that emissions are procyclical.[16] Most coefficients on the demographic variables are statistically

[16] Surprisingly, we found few other papers directly investigating this issue. Heutel (2012) documents the procyclicality of carbon dioxide (CO_2) emissions at the quarterly level. Using ARIMA regressions, he estimates the

insignificant. Precipitation is correlated with significantly lower pollution, and temperature is with significantly higher pollution.

b. Total Mortality

We next turn to the main question of 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 specifications include controls for demographic variables and state- and year-fixed effects (reported in Appendix Table A.2), as well as the state 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 0.35% decrease in the total mortality rate. This is smaller than the 0.5% predicted reduction obtained by Ruhm (2000), but consistent with evidence by Stevens et. al. (2011) that the estimated procyclicality of mortality is somewhat attenuated when adding post-1991 observations to the model.[17] 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. Standardized PM10 concentrations are incorporated in column 2, CO concentrations in column 3, O_3 concentrations in column 4, and all three pollution measures simultaneously in column 5. Neither

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elasticity between U.S. GDP and CO_2 emissions levels to be between 0.5 and 0.9. Smith and Wolloh (2012) find that aggregate water quality in the U.S. is positively correlated with the national unemployment rate.

[17] Using data from 1978-2006 and a specification similar to that in column 1, they obtain an unemployment coefficient of -.0019. See Ruhm (2013) for a detail analysis confirming that the procyclical variation in mortality has weakened or disappeared in recent years.

PM10 or O_3 levels are highly predictive of mortality, nor do they substantially attenuate the predicted macroeconomic effect. The coefficients on them, in columns 2 and 4, suggest that one standard deviation increases are associated with statistically insignificant 0.2% and 0.4% increases in mortality, which is just a fraction of the 1.9% mortality reduction predicted by a one standard deviation increase in CO levels (in column 3). When controlling for all three pollutants together (column 5), the CO coefficient is the only significant one, with a one standard deviation increase again predicting a 1.9% drop in mortality. Given this pattern, we primarily focus on the results for CO below, although controlling for all three types of emissions in our models.[18]

Adding CO emissions to the model cuts the unemployment rate coefficient by about 30%, whether or not the other two pollutants are included in the regression. In the basic specification (column 1), a one percentage point increase in unemployment reduces predicted mortality by 0.35%; this falls to 0.24% when all three pollution concentrations are controlled for (column 5).[19]

c. Cause-specific mortality

The evidence that changes in air pollution provide a mechanism for the procyclical fluctuation in mortality will be strengthened if the unemployment coefficients are more sharply attenuated after controlling for pollution for sources of fatalities that we expect to be strongly related to emissions levels (such as those from respiratory diseases) than for those where the relationship is anticipated to be weaker (like cancer deaths) or nonexistent (like homicides). We

[18] Stronger results for CO than other pollutants are consistent with the findings of other researchers. Currie and Neidell's (2005) examination of infant mortality also uncovers significant effects of CO, but not PM10 or O_3, concentrations. Beatty and Shimshack (2012) examine all three pollutants' effects on childhood morbidity and find effects from CO but not PM10. Arceo et. al (2013) examines infant mortality in Mexico and finds significant effects from both PM10 and CO, but their estimated magnitudes of CO coefficients are larger than those found in the US.

[19] Appendix Table A.2 also presents results for a model that controls for the pollution measures but not the unemployment rate. When doing so, CO continues to be positively correlated with total mortality at the 5% level, while PM10 and O_3 are insignificantly related to it (see column 6 of Table A.2). Coefficients for the remaining right-hand-side variables are also quite similar to those in our main specifications.

examine this by considering 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, Table 4 presents 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 controls for the standardized emissions levels.

Mortality rates are negatively correlated with unemployment rates for all six causes of death when not controlling for pollution (column a), although the association is not significant for respiratory or cancer mortality (but is of substantial magnitude for the former). Heart attacks are the most procyclical – a one percentage point increase in the unemployment rate is predicted to decrease deaths from this source by two percent. The same rise in unemployment is estimated to reduce mortality from cardiovascular disease and stroke by about one percent, and ischemic heart disease by 1.2%. Cancer fatalities are unrelated to macroeconomic conditions, as has been found previously (Ruhm, 2000).

The pollution measures are added as controls in specification b. We hypothesized that pollution will increase respiratory deaths and probably also some cardiovascular fatalities. PM10 and O_3 do not have a significant effect in any of these cases, consistent with the results for total mortality above. (The only exception is the barely significant negative coefficient on PM10 for cardiovascular deaths.) Conversely, ambient CO levels are significantly and positively associated with deaths from respiratory causes, cardiovascular disease, ischemic heart disease, and stroke: a one-standard-deviation increase in CO concentrations is associated with 5.7%, 1.4% and 5.9% increases in deaths from respiratory, cardiovascular, and ischemic heart diseases.

This is consistent with earlier findings of a positive effect of pollution for these causes of death.[20]

Controlling for pollution switches the sign of the unemployment coefficient for respiratory deaths from negative to positive, although the original effect was of small magnitude and imprecisely estimated. It does not attenuate the predicted effect on cardiovascular fatalities, but when focusing on ischemic heart disease, which is likely to be more responsive to short-term triggers and changes in risk factors, the magnitude of the coefficient falls by 10%. The estimated unemployment effect declines by around 15% for stroke deaths but does not change for heart attack fatalities. This last result is surprising, since heart attacks account for over half of deaths due to ischemic heart disease.

Table 5 presents information on deaths from external causes including motor vehicle and other accidents (separately), suicides, and homicides. Mortality rates from motor vehicle accidents are procyclical, suicides are countercyclical, while homicides and other accidents are not significantly related to macroeconomic conditions.[21] We do not expect fatalities from these causes to be strongly related to pollution levels, and they are not for suicides, homicides or motor vehicle deaths. The last finding is especially comforting given the potential for variation in driving behavior to be a confounding omitted variable.[22] Surprisingly, pollution is positively correlated with the non-vehicle accident death rate, and controlling for it increases the magnitude and significance of the (positive) unemployment coefficient. A potential explanation is that jobs

[20] Similarly, Peters et. al. (2004) uncover a positive correlation between exposure to pollutants caused by traffic and heart attack. Mustafic et. al.'s (2012) meta-analysis indicates that short term exposure to several pollutants, including PM10 and CO, is significantly associated with heart attack risk. Clancy et. al. (2002) demonstrate a correlation between particulate matter concentrations and respiratory and cardiovascular deaths. Wellenius et. al. (2012) link daily levels of PM2.5 to strokes.

[21] These results are consistent with prior research findings (e.g. Ruhm, 2000), except that a procyclical variation in non-vehicle accidents and homicides has sometimes previously been found. Ruhm (2013) provides evidence that non-vehicle accidents have shifted from being procyclical to countercyclical over time.

[22] Controlling for per-capita miles driven, which are significantly correlated with the vehicle fatality rate, does not substantially alter these results.

in "polluting" industries are less safe, so that when production rises, both emissions and non-vehicle accidents increase. Although we cannot directly test this possibility, since the data do not distinguish whether deaths occurred on the job, evidence from the *Bureau of Labor Statistics Injuries, Illness, and Fatalities, Census of Occupational Injuries Database* confirms the high rates of fatal injuries in the cyclically sensitive manufacturing and construction industries.[23]

d. Age-Specific Mortality

Miller et al. (2009) provide evidence that procyclical variations in mortality are particularly pronounced among the young and old – who are unlikely 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, and negative shocks might be relatively likely to result in death.[24] We address this possibility in Table 6, which summarizes estimation results for the mortality rates of six age groups: <1, 1-19, 20-54, 55-64, 65-74, and 75-84 year olds. As before, the models control for unemployment rates, demographic characteristics (other than age), and state and year effects, both with and without the pollution measures.[25]

In the basic model (column a), unemployment is negatively correlated with the mortality rates of all age groups except 20-54 year olds, with particularly strong procyclicality observed for infants and youths (under 20). A one point increase in the unemployment rate is predicted to reduce the mortality of these two groups by 0.9% and 1.4%, which compares to decreases of

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[23] For instance, using data for 2009 from http://www.bls.gov/iif/oshwc/cfoi/cftb0241.pdf (accessed March 15, 2013), we calculate that construction industries had a non-vehicle fatal accident rate that was over four times as large as that for all industries (6.9 vs. 1.5 per 100,000 workers) and that this industry accounted for over 30 percent of such occupational mortality in that year.

[24] Much of the prior literature on the health effects of pollution focuses on infant mortality (Chay and Greenstone 2003, Currie and Neidell 2005, Currie et. al. 2009, Greenstone and Hanna 2011, Knittel et. al. 2011).

[25] Observations are weighted by state population in the specified age category. We also examined the mortality of persons ≥85 but obtained insignificant results for this age group and so omit these finding from Table 6.

0.5%, 0.7% and 0.3% for 55-64, 65-74 and 75-84 year olds, and a statistically insignificant 0.1% *increase* in the death rate of prime-working-age (20-54 year old) individuals.

When adding controls for pollution concentrations (specification b), we see the hypothesized attenuation of the macroeconomic coefficients for the most vulnerable groups – infants and older individuals. Specifically, the unemployment coefficient declines, in absolute value, by 31%, 19%, and 32% for <1, 55-64 and 75-84 year olds, and becomes statistically insignificant in the last case. It is noteworthy 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.[26] As above, neither PM10 nor O_3 is significantly related to the mortality rates of any of the groups, whereas CO concentrations are predicted to increase death rates for all six age categories.

e. County-Level Results

In Table 7, we replicate earlier regression results using the county, rather than the state, as the unit of observation. There are both advantages and disadvantages to using county-level data. The main advantage is that average state levels of pollution and unemployment may conceal significant within-state disparities that are lessened by using county data. The major disadvantage is that there may be more error in the measurement of both mortality and unemployment rates at smaller units of geographic aggregation.[27] There are also issues of data comparability across time, since a consistent county unemployment rate series is only available beginning in 1990. Limiting the analysis to this period would obviously reduce sample sizes and

[26] Consistent with this, accidents 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 mortality for these groups – but were much less important for infants or senior citizens, where they were responsible for 5% and 2% of fatalities (Murphy, et al., 2012).

[27] The greater measurement error in county than state unemployment rates is well known. Errors in classifying the county of residence at death have been less studied but Pierce and Denison (2006) provide evidence of substantial misrecording of counties using mortality data from Texas.

the timespan of analysis. As an alternative, we purchased county-level unemployment data for earlier years from the *Bureau of Labor Statistics* (BLS) and combined it with the post-1989 information on county unemployment rates. However, the *BLS* warns that these data are not fully comparable to those for later years and cautions against their use in this way. There are also no deaths for some causes or age groups in smaller counties in some years; we replace these zero values with one so that we can take the natural log.[28] Finally, as detailed next, the sample of counties is limited to those with pollution monitors located within them or nearby, possibly reducing the generalizability of the results. For all of these reasons, we treat our county-level analysis as a robustness check but not necessarily preferable to the main state-level investigation.

We calculate county pollution levels in two ways. The first, analogous to what we did at the state level, averages monitor readings in the county, weighted by the percentage of potential observations from the monitor that are actually observed.[29] PM10, CO and O_3 pollution monitors are available for 923, 394 and 957 counties respectively. We restrict the analysis to 4,148 county-year observations (from 279 counties) where we have measurements for all three pollutants. The median number of years that counties appear in the sample is 15 (out of 28 total).

The second method of measuring pollution concentrations takes advantage of the known location of each monitor (latitude and longitude) and uses all monitors close to a county, (not just those inside it). Specifically, we follow Currie and Neidell (2005) by calculating a weighted average of pollution readings from all monitors within 20 miles of the county's population centroid, weighting by the inverse of the monitor's distance from the centroid.[30] This

[28] An alternative would be to estimate negative binomial regression models, which can deal with zero death counts (Miller, et al., 2009), or to drop these observations.
[29] If the county contains only one monitor, pollution readings are obtained directly from it.
[30] County population centroids are calculated by the U.S. Census Bureau, based on the 2000 Census.

substantially increases the number of counties and observations in sample, to 8,944 observations for 542 counties, since many counties without a pollution monitor do have one nearby.[31]

The mortality measures and age shares are all available at the county level for all years from the CMF files; however, the demographic and weather variables, which were obtained from other sources, are available only at the state-year level and so are included in this way in the regressions. The models also control for county-fixed effects and state-by-year-fixed effects.

The top half of Table 7 replicates results from Tables 3 and 4, but at the county level.[32] In the first two columns, the negative correlation between total mortality and unemployment rates is about twice as high as it was at the state level – a one point increase in the county unemployment rate is predicted to reduce total mortality by around 0.8%. Controlling for the three pollution measures attenuates the unemployment rate coefficient by around 10%, (versus more than 30% in the state analysis). The correlation between CO and mortality is 28% higher than at the state level but we again see no significant predicted effect of the other two pollutants.

The third through sixth columns present results for deaths from respiratory and ischemic heart disease, the two causes for which pollution had the largest effect in the state level regressions (see Table 4). In that analysis, a one point rise in unemployment predicted a statistically insignificant 0.3% reduction in respiratory deaths. Here, the estimated decrease is a much larger (and statistically significant) 1.3% decrease. CO is again the only significant pollutant but its coefficient is smaller than at the state level. Conversely, ischemic heart disease deaths are estimated to be less procyclical at the county than state level (the unemployment

[31] By contrast, adopting this method at the state level substantially reduces the number of observations, since many monitored states do not have a monitor within 20 miles of the state population centroid.

[32] Replicating Table 2 at the county level yields results that are somewhat weaker than those at the state level. Specifically, when using average monitor readings in the county, the air pollution coefficients are about one-half to one-fifth as large as they were with state-level data. Somewhat stronger and more significant associations are obtained when using the alternative distance-based measure. Weaker results at the county than state level are consistent with the previously mentioned hypothesis that county-level data are more error-prone than state data.

coefficient is 0.007, rather than 0.014, and not significant), and the coefficient on CO is only 0.0132 (compared to 0.0587 at the state level) and not significant. Controlling for pollution attenuates the unemployment rate coefficient by 8% for respiratory deaths and 10% for ischemic heart disease, again suggesting that the overall macroeconomic effects could partially result from changes in emissions.[33]

The bottom panel of Table 7 presents county level results using the distance-based measure of pollution exposure. In these regressions, the evidence of the procyclicality of mortality is somewhat weaker than in the top panel, while the effects of pollution on mortality remain strong and largely consistent with prior results (with PM10 also here being positively associated with total and respiratory mortality).[34] In these models, the unemployment coefficients are attenuated by 29% for total mortality and 40% to 50% for deaths from respiratory causes and ischemic heart disease, again consistent with pollution providing a mechanism for the macroeconomic effects.

f. Comparison to Other Estimates

The preceding analysis demonstrates that air pollution provides a plausible mechanism for some of the procyclicality of mortality. Causality is difficult to prove because of the potential for uncontrolled confounding factors. What we can do, however, is examine whether our predicted pollution effects are plausible when compared to results from previous research providing micro-level estimates of the relationship between ambient concentrations and

[33] Mixed results were obtained for other types of mortality (not reported in the table). The county level estimates indicated procyclicality of deaths from cardiovascular disease, heart attacks, and stroke, but not those from external causes. CO was only significantly correlated with cardiovascular deaths, and the unexpected positive correlation between pollution and non-vehicle accident fatalities remained.

[34] Following Chay and Greenstone (2005), we also estimated models using the county's attainment status, defined by the Clean Air Act's National Ambient Air Quality Standards (NAAQSs), as an instrumental variable for pollution. Unfortunately, attainment status turned out to be a weak instrument, and so the second-stage coefficients (for mortality) were insignificant.

mortality. This comparison is summarized in Table 8. A detailed description of the methods and calculations is provided in Appendix B.

Although the basic procedure is straightforward, there are several issues need to be addressed. First, our estimates above examined the percent change in mortality predicted by a one standard deviation change in the specified pollution level. By contrast, earlier research reports effects of standard units of pollution (e.g. parts per million or $\mu g/m^3$), as well as alternative outcome measures (e.g. deaths per 100,000 or relative risk ratios). Therefore, our first task was to use conversion factors to make our estimates as comparable as possible to those of earlier investigations. Second, some previous studies focused on incidence (e.g. of strokes or heart attacks) rather than mortality rates. In these cases, we make the strong assumption that the incidence and mortality effects are comparable. Third, the prior research analyzes a limited set of mortality outcomes, using regression specifications that may be quite different than ours.

Given these issues, we present point estimates only (no confidence intervals or standard errors) to highlight that our "back-of-the-envelope" calculations are not meant to provide precise estimates, but instead a qualitative assessment of how our results compare to those obtained previously. We should also emphasize that the prior estimates are often imprecise, and that deviations between our results and those of earlier work do not necessarily indicate biases or errors in our predictions.

Table 8 provides consistent, although certainly not definitive, evidence corroborating our hypothesis that pollution may explain a substantial portion of the procyclicality of mortality. Based on prior estimates of the effects of pollution on mortality, combined with our results showing how emissions vary with macroeconomic conditions, pollution is estimated to account for between 7% to 30% of the procyclicality of mortality in most models, although with smaller

or larger estimates in three cases (columns 4, 6 and 7). Conversely, estimates based fully on our analysis (including those for the predicted mortality effects of pollution) suggest that air pollution accounts for 9% to 15% of the cyclical fluctuations, in those cases where we estimate that it has any effect.[35] The estimates using the two methods are sufficiently similar to suggest that our anlaysis provides plausible estimates of the role of pollution in accounting for procyclical variations in mortality.

V. Discussion

Recent research indicates that mortality increases during times of economic strength and declines when the economy weakens. This relationship is strongest for the young and old, rather persons of prime-working age, suggesting that the direct effects of changes in labor market conditions are unlikely to fully explain these patterns.[36] A plausible alternative is that variations in other risks explain some of the macroeconomic fluctuation in deaths. One such risk, traffic fatalities, has been widely studied and universally found to increase when the economy strengthens. However, a different potential health risk – air pollution – is also likely to depend on the state of the economy but has not been previously studied. We begin to remedy this shortcoming by providing an initial investigation of how three types of emissions, carbon monoxide (CO), particulate matter (PM10), and ozone (O_3), fluctuate with macroeconomic conditions, and whether these variations help to explain observed fluctuations in mortality rates.

Specifically, we used panel data for 1982-2009 to identify the effect of the macroeconomy on mortality rates, with and without controls for ambient pollution

[35] We did not find a significantly positive effect of PM10 on deaths from strokes, respiratory or cardiovascular diseases, so Table 8 reports a zero effect in these cases.

[36] However, there could be indirect effects. For example, working age individuals may have more time during economic downturns to care for young children or aged parents, resulting in health benefits for these groups.

concentrations. Consistent with previous research, we uncovered a significant negative correlation between state unemployment and mortality rates, after controlling for state demographic characteristics and fixed-effects, as well as general year effects. Adding the three air pollutants to the model attenuated the predicted unemployment rate effect by 30%, consistent with a substantial role for air pollution. CO concentrations were estimated to be more important than PM10 or O_3 concentrations, but we do not know whether this represents differences in true health effects, the accuracy of emissions measurement, or correlations with other types of pollution or uncontrolled confounding factors.[37]

The results for specific causes of death were also largely (but not entirely) consistent with an important role for air pollution as a mechanism explaining procyclical changes in mortality. In particular, CO levels had large positive direct estimated effects on fatalities from respiratory causes, ischemic heart disease and stroke, and their inclusion substantially attenuated the unemployment rate coefficients in these models. However, the macroeconomic estimates were imprecise for several causes of death, and the attenuation resulting from including the pollution controls was modest for some types of cardiovascular mortality. Pollution levels were also positively associated with non-vehicle accidental deaths for reasons that are not well understood but could be related to the growth, during economic rebounds, of risky cyclically-sensitive jobs such as those in construction industries. As a check on the plausibility of our results, we combined our estimates of the responsiveness of pollution to macroeconomic conditions with the findings of detailed previous investigations of how pollution affects mortality. The results of this

[37] The toxicity of exposure to high CO levels has long been understood, and recent epidemiological studies also suggest the dangers of exposure at lower levels, for even relatively brief periods of time (USEPA 2010). Consistent with this, some recent research (Arceo et. al. 2013, Beatty and Shimshack 2012, Currie and Neidell, 2005) finds a key role for CO. However, other investigations obtain negative health effects of total suspended particulates (Chay and Greenstone, 2003) or, in some specifications, PM10 (Knittel et al., 2011). We also link PM10 to mortality in our estimates using county level data and a distance-based pollution measure (see Table 7). Thus, disentangling between CO and PM10 mortality effects remains an area with unanswered questions.

analysis further suggest that changes in pollution levels may explain a portion of the observed procyclical variation in mortality.

This research should be considered preliminary, rather than definitive, because of some unexplained results and since many extensions would be desirable. At the most basic level, we cannot be sure that we are not underestimating or overestimating the true effects of pollution. On the one hand, our emissions measures are crude, being limited to just three of many types of pollutants, and may be measured with error, leading to an understatement of the true effects. In future work it would be useful to control for additional pollutants and to go beyond average ambient concentrations (e.g. by examining peak levels and fluctuations around the mean). The use of state level data is also potentially problematic since pollution monitoring is provided for only a limited set of locations. We partially addressed this by conducting a county-level investigation, which provided results that were largely but not entirely consistent with findings at the state-level.

Furthermore, other robustness checks of our main results met with mixed success. For instance, in regressions replicating Table 3, but using gross state product (GSP) or average state personal income as the macroeconomic measure, rather than unemployment rates, the coefficients on these proxies were positive but not quite significant (the p-values were 0.24 and 0.19), weakly suggesting the procyclicality of mortality.[38] However, controlling for pollution did not attenuate the estimated macroeconomic effects in these models, suggesting the need for further research using alternate measures of the business cycle.[39]

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[38] Data were from the Bureau of Economic Analysis: http://www.bea.gov/iTable/iTable.cfm?ReqID=70&step=1. Both GSP and personal income exhibit a trend, we detrended each state's annual real GSP and personal income series (in logs) using the Hodrick-Prescott filter (Hodrick and Prescott 1997), with a smoothing parameter of 6.25 for annual data (Ravn and Uhlig 2002).

[39] CO was again the only pollutant that was significantly correlated with mortality.

We also ran regressions separately for the 1982-1994 and 1995-2009 periods.[40] Recent studies (Stevens et al., 2011; Ruhm, 2013) indicate that the procyclicality of mortality has become less pronounced in recent years. Consistent with this, we find that mortality is highly procyclical from 1982-1994 but not at all, or even slightly countercyclical, from 1995 on. On the other hand, the positive correlation between CO emissions and mortality is only found after 1995. This suggests that the cyclical behavior of both pollution and emissions has changed over time, and that additional study is needed to understand these changes.

Finally, we cannot be sure that an attenuation of the estimated macroeconomic effects occurring when pollution controls are added to the model reflects a causal relationship rather than a spurious correlation between emissions and unobserved factors. This may lead to an overstatement of the true effect of pollution and suggests that alternative strategies, such as instrumental variables techniques, might be useful.

Notwithstanding these caveats, our results suggest potential implications for policy. The findings should certainly *not* be taken to imply that recessions are beneficial (although they may slightly less costly than is commonly understood), or used to argue for (or against) macroeconomic stabilization policies. Indeed, the procyclicality of pollution-induced mortality could be irrelevant to optimal emissions policy, if the marginal external damages (to which the marginal price of pollution should be equalized) do not vary over the business cycle. Conversely, it may be useful to moderate the cyclical fluctuations in pollution if the damages are nonlinear, or if the elimination or moderation of mortality spikes during expansions is a public policy goal in its own right. A tradable emissions permits scheme might assist in accomplishing this goal

[40] We break the sample in this way because 1995, which is roughly the midpoint of the full sample period, is the first year for which PM10 data become available in the majority of states.

since the costs of polluting would rise in periods of economic strength, when the demand for permits is high.

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Table 1: Sample Means for Selected Variables

	Mean	Standard Error
Emissions		
PM10 concentration ($\mu g/m^3$)	27.394	0.224
CO concentration (ppm)	0.885	0.013
O_3 concentration (ppm)	0.052	0.000
State unemployment rate (%)	5.588	0.051
Mortality Rates (per 1000)		
Total	8.670	0.036
Respiratory {466-496}, [J20-J47, U04]	0.590	0.005
Cardiovascular {390-448}, [I00-I78]	3.403	0.022
Acute Myocardial Infarction {410}, [I21-I22]	0.763	0.009
Ischemic Heart Disease {410-414}, [I20-I25]	1.298	0.022
Cerebrovascular Disease {430-438}, [I60-I69]	0.572	0.004
Cancer (140-208}, [C00-C97]	1.991	0.009
Accident {E800-E869, E880-E929}, [V01-X59, Y85-Y86]	0.399	0.003
Vehicle Accident {E810-E825}, [V02-V89]	0.178	0.002
Non-vehicle Accident {E800-E807, E826-E949}, [V90-X59, Y85-Y86]	0.221	0.002
Suicide {E950-E959}, [X60-X84, Y87.0]	0.126	0.001
Homicide {E960-E978}, [X85-Y09, Y87.1, Y35, Y89.0]	0.073	0.002
< 1 year old	8.275	0.075
1-19 years old	0.402	0.003
20-54 years old	2.260	0.016
55-64 years old	10.680	0.063
65-74 years old	24.753	0.111
75-84 years old	57.510	0.172
≥ 85 years old	151.246	0.274
State Population Shares		
< 1 year old	0.014	0.000
1-19 years old	0.271	0.001
20-54 years old	0.497	0.001
55-64 years old	0.092	0.000
65-74 years old	0.069	0.000
75-84 years old	0.042	0.000
≥ 85 years old	0.015	0.000
Female	0.514	0.000
Black (non-Hispanic)	0.120	0.004
Other nonwhite (non-Hispanic)	0.040	0.001
Hispanic	0.070	0.003
High school incomplete	0.176	0.002
High school graduate/12th grade completed	0.353	0.002
Some college/<4 years completed	0.231	0.002
College graduate/4+ years completed	0.240	0.002
Weather		
Annual precipitation (mm, 00s)	37.772	0.440
Temperature (°F)	52.894	0.224

Note: Summary statistics are over the state-year observations, from 1982-2009, including only those 1147 observations for which we have PM10, CO, and O_3 concentrations. ICD-9 codes for specific causes of death categories applying from 1982-1998 are shown in curly brackets; corresponding ICD-10 codes, used from 1999 on are displayed in square brackets.

Table 2: Relationship between Pollution and Unemployment Rates

	State Emissions Level					
Regressor	PM 10 (1)	PM 10 (2)	CO (3)	CO (4)	O_3 (5)	O_3 (6)
State unemployment rate (%)	-0.100**	-0.0930***	-0.0611**	-0.0529*	-0.0704**	-0.0372*
	(0.0382)	(0.0276)	(0.0274)	(0.0264)	(0.0325)	(0.0219)
Demographic controls	No	Yes	No	Yes	No	Yes
Observations	1,147	1,147	1,147	1,147	1,147	1,147
R-squared	0.816	0.836	0.862	0.887	0.699	0.808

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. Even-numbered columns also include controls for the share of state residents who are female, black, other nonwhite, in one of six age groups (<1, 1-19, 55-64, 65-75, 75-84 and ≥85 years old), as well as the share of ≥25 year olds who are in three education categories (high school graduate without college, some college, college graduate). Regressions are weighted by the state population. Robust standard errors, clustered at the state level, are reported in parentheses.

Table 3: Econometric Estimates of the Determinants of Total Mortality

Regressor	(1)	(2)	(3)	(4)	(5)
State unemployment rate (%)	-0.00354**	-0.00334**	-0.00253**	-0.00338**	-0.00243**
	(0.00142)	(0.00134)	(0.00120)	(0.00142)	(0.00118)
PM10		0.00211			-0.000733
		(0.00277)			(0.00244)
CO			0.0191**		0.0192**
			(0.00879)		(0.00870)
O_3				0.00420	0.00433
				(0.00254)	(0.00272)
R-squared	0.984	0.984	0.985	0.984	0.985

Note: *** p<0.01, ** p<0.05, * p<0.1. The dependent variable is the natural log of the total mortality rate (n=1,147). State- and year-fixed effects are included but not reported. All models also include controls for the share of state residents who are female, black, other nonwhite, in one of six age groups (<1, 1-19, 55-64, 65-75, 75-84 and ≥85 years old), as well as the share of ≥25 year olds who are in three education categories (high school graduate without college, some college, college graduate). Regressions are weighted by the state population. Standard errors, clustered at the state level, are reported in parentheses.

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 (%)	-0.00328	0.000284	-0.00695***	-0.00687***	-0.0198***	-0.0198***
	(0.00458)	(0.00395)	(0.00223)	(0.00231)	(0.00412)	(0.00346)
PM10		0.00510		-0.00770*		-0.0124
		(0.00698)		(0.00400)		(0.0108)
CO		0.0568***		0.0142*		0.0289
		(0.0191)		(0.00757)		(0.0238)
O_3		0.00223		0.00150		-0.0117
		(0.00705)		(0.00312)		(0.00858)
	Ischemic Heart Disease		**Stroke**		**Cancer**	
	(a)	(b)	(a)	(b)	(a)	(b)
State unemployment rate (%)	-0.0142**	-0.0129**	-0.0107***	-0.00915***	-0.000492	0.000204
	(0.00533)	(0.00606)	(0.00352)	(0.00297)	(0.00115)	(0.000898)
PM10		-0.0166		-0.00755		-0.000833
		(0.0143)		(0.00547)		(0.00239)
CO		0.0587*		0.0377**		0.0129*
		(0.0312)		(0.0174)		(0.00687)
O_3		-0.00820		0.00716		0.00249
		(0.0127)		(0.00462)		(0.00221)

Note: *** p<0.01, ** p<0.05, * p<0.1. The dependent variables are natural logs of the specified cause-specific mortality rate (n=1,147). All models control for state- and year-fixed effects and demographic characteristics. Observations are weighted by the state population. Standard errors, clustered at the state level, are reported in parentheses.

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

Regressor	Vehicle Accident		Non-Vehicle Accident	
	(a)	(b)	(a)	(b)
State unemployment rate (%)	-0.0306***	-0.0288***	0.00309	0.00871*
	(0.00372)	(0.00394)	(0.00521)	(0.00488)
PM10		0.0111		0.0299***
		(0.00942)		(0.00993)
CO		0.0137		0.0371**
		(0.0127)		(0.0163)
O_3		0.00191		0.0236*
		(0.0116)		(0.0131)
	Suicide		**Homicide**	
	(a)	(b)	(a)	(b)
State unemployment rate (%)	0.0153***	0.0159***	0.00345	0.00403
	(0.00507)	(0.00481)	(0.00977)	(0.00864)
PM10		0.00442		-0.0107
		(0.00728)		(0.0214)
CO		-0.00551		0.0271
		(0.0108)		(0.0427)
O_3		0.0116		0.00366
		(0.00780)		(0.0180)

Note: *** p<0.01, ** p<0.05, * p<0.1. The dependent variables are the natural logs of the specified cause-specific mortality rate (n=1,147). All models control for state- and year-fixed effects and demographic characteristics. Observations are weighted by state population. Standard errors, clustered at the state level, are reported in parentheses.

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 (%)	-0.00876***	-0.00608**	-0.0138***	-0.0131***	0.00128	0.00414
	(0.00266)	(0.00236)	(0.00428)	(0.00393)	(0.00346)	(0.00290)
PM10		0.00608		-0.00452		0.00151
		(0.00625)		(0.00803)		(0.00702)
CO		0.0389***		0.0238*		0.0480*
		(0.0132)		(0.0124)		(0.0241)
O_3		-0.00299		-0.00466		0.00453
		(0.00879)		(0.00821)		(0.00706)
VARIABLES	55-64 Year Olds		65-74 Year Olds		75-84 Year Olds	
	(a)	(b)	(a)	(b)	(a)	(b)
State unemployment rate (%)	-0.00463**	-0.00373**	-0.00678***	-0.00579***	-0.00284*	-0.00194
	(0.00211)	(0.00178)	(0.00139)	(0.00128)	(0.00159)	(0.00142)
PM10		-0.00290		-0.00227		-0.00195
		(0.00415)		(0.00357)		(0.00218)
CO		0.0232**		0.0216**		0.0185**
		(0.00925)		(0.00824)		(0.00734)
O_3		-0.000736		0.000713		0.00404
		(0.00448)		(0.00379)		(0.00245)

Note: *** p<0.01, ** p<0.05, * p<0.1. The dependent variables are natural logs of the specified age-specific mortality rate. All models control for state- and year-fixed effects and demographic characteristics. Observations are weighted by the state population within each age group. Standard errors, clustered at the state level, are reported in parentheses

Table 7: County-Level Analysis

Regressor	Total Mortality		Respiratory		Ischemic Heart Disease	
	(a)	(b)	(a)	(b)	(a)	(b)
Counties with Pollution Monitors (n=4,148)						
County unemployment rate (%)	-0.00759***	-0.00685***	-0.0131*	-0.0120*	-0.00719	-0.00644
	(0.00260)	(0.00241)	(0.00713)	(0.00693)	(0.00876)	(0.00863)
PM10		0.00165		-0.00194		0.0104
		(0.00366)		(0.00702)		(0.00884)
CO		0.0246***		0.0390**		0.0132
		(0.00728)		(0.0165)		(0.0143)
O_3		0.00176		-0.000519		-0.0101
		(0.00648)		(0.00938)		(0.0111)
Counties with Pollution Monitors within 20 Miles of Population Centroid (n=8,944)						
County unemployment rate (%)	-0.00380*	-0.00270	-0.00602	-0.00346	-0.00166	-0.000854
	(0.00220)	(0.00212)	(0.00557)	(0.00528)	(0.00706)	(0.00680)
PM10		0.00481*		0.0139**		-0.000410
		(0.00262)		(0.00582)		(0.00792)
CO		0.0233***		0.0480***		0.0277**
		(0.00484)		(0.0129)		(0.0129)
O_3		-0.00570		-0.00757		-0.0190**
		(0.00422)		(0.00990)		(0.00905)

Note: *** p<0.01, ** p<0.05, * p<0.1. The dependent variables natural logs of the total or cause-specific mortality rate. All models control for county- and state-by-year fixed effects and demographic characteristics. Regressions are weighted by the county population. In the top panel, analysis is restricted to counties monitoring all three pollutants. In the bottom panel, analysis includes counties with monitors within 20-miles of the population centroid, with distance-weighted pollution measures calculated, using procedures detailed in the text. Standard errors, clustered at the county level, are reported in parentheses.

Table 8: Comparison of Estimated Pollution Effects Based on Previous Research and Current Analysis

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Paper	Currie and Neidell (2005)	Chay and Greenstone (2003)	Knittel et. al. (2011)	Knittel et. al. (2011)	Mustafic et. al. (2012)	Mustafic et. al. (2012)	Clancy et. al. (2002)	Clancy et. al. (2002)	Wellenius et. al. (2012)
Pollutant	CO	TSP/PM10	CO	PM10	CO	PM10	PM10	PM10	PM2.5/PM10
Outcome	Infant mortality	Infant mortality	Infant mortality	Infant mortality	Heart Attacks	Heart Attacks	Respiratory Deaths	Cardiovascular Deaths	Stroke
				Estimated Effect of a One Unit Pollution Increase on Mortality[1] Based On:					
Previous Research	18.1	1.00	17.1	18.0	0.0550	0.000600	0.00443	0.00294	0.00103
Current Research	34.7	1.41	34.7	1.41	0.0715	0	0.000653	0	0
				Change in Cyclical Deaths Explained By Pollution Based On:					
Previous Research	7.2%	11.0%	6.8%	197.24%	7.1%	2.0%	96.6%	30.2%	6.9%
Current Research	13.7%	15.4%	13.7%	15.4%	9.30%	0%	14.2%	0%	0%

[1] Units are deaths per 100,000 live births (change in death rate), in response to a one-unit change in pollution, in columns 1 through 4 (5 through 9). CO units are parts per million (ppm); PM10 units are $\mu g/m^3$.

Appendix A: Supplementary Tables

Appendix Table A.1: Relationship between Pollution and Unemployment Rates

	State Emissions Level					
Regressor	PM 10	PM 10	CO	CO	O_3	O_3
	(1)	(2)	(3)	(4)	(5)	(6)
state unemployment rate (%)	-0.100**	-0.0930***	-0.0611**	-0.0529*	-0.0704**	-0.0372*
	(0.0382)	(0.0276)	(0.0274)	(0.0264)	(0.0325)	(0.0219)
<1 year old		109.7**		39.52		161.8*
		(49.83)		(47.00)		(84.84)
1-19 years old		-11.00		0.844		-22.60**
		(7.531)		(5.701)		(10.17)
55-64 years old		-27.67**		1.574		10.83
		(12.54)		(16.13)		(10.72)
65-74 years old		22.58		-14.70		-18.74
		(20.85)		(15.50)		(11.30)
75-84 years old		-13.11		15.43		-33.87
		(25.76)		(29.41)		(21.99)
≥85 years old		98.54*		-19.31		93.21*
		(54.31)		(51.08)		(53.28)
female		-0.620		0.924		-1.325
		(2.277)		(2.129)		(2.566)
black		0.0164		-2.981		0.454
		(1.663)		(1.836)		(1.836)
other nonwhite		-3.024		-7.334***		-5.121*
		(2.755)		(2.133)		(2.827)
Hispanic		1.530		1.226		-1.974
		(2.275)		(1.749)		(1.434)
high school graduate/12th		-1.242		3.641*		-1.246

grade completed (age 25+)		(1.655)		(1.906)		(1.385)
some college/<4 years completed (age 25+)		0.532		6.111***		0.809
		(1.996)		(2.022)		(1.840)
college graduate/4+ years completed (age 25+)		1.928		2.174		1.281
		(1.832)		(1.983)		(2.098)
average annual precipitation		-0.0114***		-0.00248**		-0.0234***
		(0.00255)		(0.00100)		(0.00244)
average temperature		0.0302**		0.00933		0.122***
		(0.0135)		(0.0118)		(0.0212)
constant	1.966***	4.878	1.536***	-1.311	0.304	6.646*
	(0.595)	(3.256)	(0.357)	(4.040)	(0.378)	(3.482)
Observations	1,147	1,147	1,147	1,147	1,147	1,147
R-squared	0.816	0.836	0.862	0.887	0.699	0.808

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

Appendix Table A.2: Econometric Estimates of the Determinants of Total Mortality

Regressor	(1)	(2)	(3)	(4)	(5)	(6)
state unemployment rate (%)	-0.00354**	-0.00334**	-0.00253**	-0.00338**	-0.00243**	
	(0.00142)	(0.00134)	(0.00120)	(0.00142)	(0.00118)	
PM10		0.00211			-0.000733	-4.69e-05
		(0.00277)			(0.00244)	(0.00247)
CO			0.0191**		0.0192**	0.0200**
			(0.00879)		(0.00870)	(0.00881)
O_3				0.00420	0.00433	0.00460
				(0.00254)	(0.00272)	(0.00281)
<1 year old	13.14***	12.91***	12.39***	12.46***	11.76***	10.79***
	(3.581)	(3.357)	(3.205)	(3.739)	(3.258)	(3.383)
1-19 years old	-1.495***	-1.472***	-1.511***	-1.400***	-1.421***	-1.577***
	(0.355)	(0.347)	(0.376)	(0.391)	(0.403)	(0.389)
55-64 years old	2.785**	2.843**	2.755***	2.739**	2.687***	2.648***
	(1.053)	(1.082)	(0.927)	(1.072)	(0.964)	(0.980)
65-74 years old	2.419*	2.371*	2.699**	2.498*	2.799**	2.661**
	(1.288)	(1.295)	(1.089)	(1.312)	(1.107)	(1.072)
75-84 years old	7.165***	7.193***	6.870***	7.307***	7.005***	7.043***
	(2.070)	(2.069)	(1.748)	(2.023)	(1.716)	(1.730)
≥85 years old	8.275***	8.067***	8.643***	7.884***	8.315***	7.874***
	(2.802)	(2.753)	(2.662)	(2.743)	(2.569)	(2.598)
female	0.126	0.128	0.109	0.132	0.114	0.139
	(0.153)	(0.154)	(0.136)	(0.153)	(0.135)	(0.135)
black	-0.109	-0.109	-0.0520	-0.111	-0.0535	-0.0559
	(0.158)	(0.157)	(0.113)	(0.159)	(0.114)	(0.114)
other nonwhite	-0.220	-0.213	-0.0799	-0.198	-0.0588	-0.0532
	(0.199)	(0.200)	(0.194)	(0.198)	(0.192)	(0.198)
Hispanic	-0.0169	-0.0201	-0.0403	-0.00863	-0.0308	-0.0221
	(0.156)	(0.152)	(0.123)	(0.152)	(0.120)	(0.122)

high school graduate/12th	0.0777	0.0803	0.00824	0.0830	0.0122	0.0209
grade completed (age 25+)	(0.0868)	(0.0873)	(0.0904)	(0.0833)	(0.0870)	(0.0891)
some college/<4 years	0.318**	0.317**	0.202*	0.315**	0.198*	0.193*
completed (age 25+)	(0.138)	(0.136)	(0.103)	(0.138)	(0.103)	(0.104)
college graduate/4+ years	-0.0651	-0.0691	-0.107	-0.0705	-0.111	-0.109
completed (age 25+)	(0.132)	(0.130)	(0.116)	(0.132)	(0.116)	(0.117)
average annual precipitation	5.81e-05	8.21e-05	0.000106	0.000156	0.000199*	0.000210*
	(0.000112)	(0.000100)	(0.000105)	(0.000138)	(0.000117)	(0.000116)
average temperature	-0.000397	-0.000461	-0.000575	-0.000908	-0.00108	-0.00132
	(0.000761)	(0.000796)	(0.000797)	(0.000728)	(0.000798)	(0.000809)
Observations	1,147	1,147	1,147	1,147	1,147	1,147
R-squared	0.984	0.984	0.985	0.984	0.985	0.985

Note: *** p<0.01, ** p<0.05, * p<0.1. Dependent variable is the natural log of the total mortality rate. State- and year-fixed effects are included but not reported. All regressors other than the unemployment rate refer to state population shares. Regressions are weighted by state population. Robust standard errors clustered at the state level are in parentheses.

Appendix B: Estimated Pollution Effects from Our Analysis and Previous Research

In Table 8, we combined micro-level estimates of the effects of pollution on mortality rates, obtained from other studies, with our estimates of the relationship between pollution and the business cycle to predict how a one-percentage-point increase in unemployment would affect the mortality rate. We then compared these results to those obtained entirely based on estimates using our analysis sample and procedures. The methods for doing so are described more fully in this appendix.

We draw on six previous studies. Currie and Neidell (2005) examine the effect of PM10, CO, and O_3 on infant mortality rates in California. In their preferred specification, a one part per million (ppm) reduction in CO concentration is correlated with an 18.125 decrease infant deaths per 100,000 live births.[41] Chay and Greenstone (2003) estimate that a 1 unit ($\mu g/m^3$) reduction of total suspended particulates (TSPs) is asscociated with 4-7 fewer infant deaths per 100,000 live births.[42] In OLS regressions, Knittel et. al. (2011) find significant negative effects of CO concentrations on infant mortality, but after instrumenting for pollution using traffic and weather data, they instead obtain significant effects for PM10 concentrations, where a one unit ($\mu g/m^3$) reduction is associated with 18 fewer deaths per 100,000 live births. Mustafic et. al.'s (2012) meta-analysis indicates that a 1 mg/m^3 increase in CO concentration increases the relative risk of heart attacks to 1.048 (a 4.8% increase), and a 10 $\mu g/m^3$ increase in PM10 concentration increases the relative risk to 1.006. Clancy et. al. (2002) find that a 35.6 $\mu g/m^3$ increase in PM10

[41] Like us, they find that only CO is significantly correlated with mortality. In a closely related study of New Jersey, Currie et al. (2009) estimate that the same CO reduction would eliminate 17.6 infant deaths per 100,000 live births.
[42] To compare our results for PM10 to the results for TSP (an older designation of particulates that includes more pollutants), we use rule-of-thumb EPA guidelines for comparing different types of particulates, cited in Dockery and Pope (1994), suggesting that one unit of TSP is equivalent to 0.55 units of PM10.

concentration decreases respiratory deaths by 15.5% and cardiovascular deaths by 10.3%.[43] Wellenius et. al. (2012) estimate that the odds ratio of stroke onset is 1.11 per 6.4 $\mu g/m^3$ increase in PM2.5 concentration.[44]

Table B.1 provides details on the calculations summarized in Table 8. For greater clarity, we carefully describe the methods and findings in column 1 of that table, which uses results from Currie and Neidell's (2005) analysis of the effects of CO exposure on infant mortality in California. The first row of the table presents our estimate of the cyclicality of pollution, where a one-percentage-point increase in unemployment predicts a 0.0258 ppm reduction in CO. This unemployment rate coefficient differs from that shown in Table 2 because the units of CO are ppm, so as to be comparable with Currie and Neidell (2005). The second row provides our estimate of the cyclicality of mortality – the unemployment coefficient from a model where infant mortality is the outcome. We estimate that a one percentage point increase in unemployment is correlated with 6.53 fewer infant deaths per 100,000 live births. This result differs from the corresponding estimate in Table 6 because the log (rather than level) of mortality rates was used there.

The third row displays Currie and Neidell's (2005) previously described estimate that a one ppm increase in CO causes 18.125 additional deaths per 100,000 live births. The fourth row, "Δ in Cyclical Deaths Explained" is the product of the first and third rows (-0.0258 × 18.125), and indicates how the pollution reduction induced by a one percentage point increase in the unemployment rate is expected to decrease infant mortality, based on Currie and Neidell's (2005)

[43] Clancy et. al. (2002) actually study a pollutant called "black smoke," or "British smoke." The EPA finds that black smoke is equivalent to PM10 (Dockery and Pope 1994).

[44] The EPA's guidelines for comparing PM2.5 to PM10 suggest that one unit of PM2.5 is equivalent to 1.67 units of PM10 (Dockery and Pope 1994). Wellenius et. al. (2012) reports the effect of a 6.4 $\mu g/m^3$ change in PM2.5 because that is the interquartile range of pollution exposure in their study. We convert this to an impact of a 1 $\mu g/m^3$ change by simply dividing the effect by 6.4.

estimate of how CO is related to the infant death rate. The resulting expected reduction of 0.468 deaths per 100,000 live births is then divided by the estimated total reduction of 6.53 deaths per 100,000 live births from a one point increase in unemployment (shown in row 2), to imply that the share of the procyclicality of infant deaths due to the cyclicality of pollution is 7.2% (-0.468/6.53). This is shown in the fifth row and labeled "Pollution Share of Cyclical Deaths".

The sixth through eighth rows of Table B.1 present corresponding results based purely on the estimates developed in this analysis. Row 6 shows our prediction that a one unit increase in pollution (in the same units as the comparison paper) will increase infant mortality by almost twice as much as was found by Currie and Neidell (34.7 vs. 18.1 deaths per thousand live births).[45] The seventh and eighth rows repeat the calculations in rows (4) and (5), except using our result for how pollution affects mortality. Thus, the "Δ in Cyclical Deaths Explained" is -0.8953 (-0.0258 × 34.70) and the "Pollution Share of Cyclical Deaths" is 13.7% (-0.8953/6.53).

The remaining columns of Table 8 show results using other research on the link between pollution and health. Using Chay and Greenstone's (2003) results, PM10 pollution accounted for 11.0% of the procyclicality of infant mortality. The point estimate based on our analysis is about 40% larger, although not statistically significant. Knittel et. al.'s (2011) OLS regressions find CO effects comparable to those in Currie and Neidell (2005). However, their IV regressions instead show a PM10 effect, and one that is an order of magnitude greater than our estimates or those of Chay and Greenstone (2003). Using this result, the predicted contribution of pollution-induced mortality cyclicality is twice as big as the total change in deaths (see column 4). If correct, this finding implies that other cyclical changes, besides that of PM10, would make the infant mortality rate countercyclical.

[45] Currie and Neidell (2005), and the papers in columns 2 through 4, consider death rates per 100,000 live births; we evaluate death rates per 100,000 <1 year olds. Here we assume that these death rates are equivalent.

Columns 5 through 9 compare our results with those from medical studies, where mortality effects are reported in terms of relative risk or odds ratio, which we describe as percentage changes (i.e. a relative risk of 1.05 equals a 5% increase). In column 5, Mustafic et. al. (2012) report that a one-unit increase in CO raises heart attack incidence by 5.5%.[46] Our estimate (for heart attack deaths) is 7.2%, or about one-and-a-half times as large. When combining our estimate of the procyclicality of CO with Mustafic et. al.'s (2012) estimate of heart attack incidence, pollution accounts for 7.1% of the overall procyclicality of heart attack mortality. Using just our estimates, 9.3% of the procyclicality is accounted for. Mustafic et. al. (2012) also report results for PM10 on heart attacks (column 6) which, combined with our estimates of the effects of the economic conditions on PM10 levels, indicates that the cyclicality of PM10 accounts for 2.0% of the change in deaths from heart attacks. Clancy et. al. (2002) find significantly positive effects of PM10 on both respiratory and cardiovascular deaths. According to our calculations, these account for 97% and 30% of the procyclical variations in mortality from these causes. Conversely, our analysis fails to uncover a significantly positive effect of PM10 on deaths from either cause (unemployment coefficient is negative for the latter), so we report zero effects. We do estimate that changes in PM10 are responsible for around 14% of the procyclical variation in cardiovascular mortality, one-sixth as large as that predicted using Clancy et al.'s 2002 estimates. Finally, Wellenius et. al. (2012) find a significant effect of PM2.5 on stroke incidence, which appears to explain around 7% of the procyclicality of stroke mortality.[47] Once again, we do not find any relationship between PM10 (our closest equivalent to PM2.5) and deaths from strokes, so that none of the macroeconomic variation in stroke mortality is explained.

[46] Mustafic et. al. (2012) reports relative risk on heart attack *incidence* (not *mortality*) for a 1 mg/m^3 increase in CO concentrations; we adjust to the standard units of CO with the conversion factor 1 mg/m^3 = 0.873 ppm.
[47] Wellenius et. al. (2012) also report odds ratios for stroke incidence rather than mortality.

Table B.1: Estimated Pollution Effects Using Results from Previous Research

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Paper	Currie and Neidell (2005)	Chay and Greenstone (2003)	Knittel et. al. (2011)	Knittel et. al. (2011)	Mustafic et. al. (2012)	Mustafic et. al. (2012)	Clancy et. al. (2002)	Clancy et. al. (2002)	Wellenius et. al. (2012)
Pollutant	CO	TSP/PM10[6]	CO	PM10	CO	PM10	PM10	PM10	PM2.5/PM10[7]
Outcome	Infant mortality	Infant mortality	Infant mortality	Infant mortality	Heart Attacks	Heart Attacks	Respiratory Deaths	Cardiovascular Deaths	Stroke
Estimate of Cyclicality of Pollution and Mortality, Based on Current Analysis									
Cyclicality of Pollution[1]	–0.0258	–0.716	–0.0258	–0.716	–0.0258	–0.716	–0.716	–0.716	–0.716
Cyclicality of Mortality[2]	–6.53	–6.53	–6.53	–6.53	–0.0199	–0.0199	–0.00328	–0.00695	–0.0107
Estimate of Pollution as Mechanism for Cyclical Variation in Mortality, Based on Previous Research									
Pollution on Mortality[3]	18.1	1.00	17.1	18.0	0.0550	0.000600	0.00443	0.00294	0.00103
Δ in Cyclical Deaths Explained[4]	–0.468	–0.716	–0.441	–12.9	–0.00142	–.000400	–0.00317	–0.00210	–0.000737
Pollution Share of Cyclical deaths[5]	*7.17%*	*11.0%*	*6.75%*	*197%*	*7.14%*	*2.01%*	*96.6%*	*30.2%*	*6.89%*
Estimate of Pollution as Mechanism for Cyclical Variation in Mortality, Based on Current Analysis									
Pollution on Mortality[1]	34.7	1.41	34.7	1.41	0.0715	0	0.000653	0	0
Δ in Cyclical Deaths Explained[4]	–0.896	–1.01	–0.896	–1.01	–0.00185	0	–0.000467	0	0
Pollution Share of Cyclical deaths[5]	*13.7%*	*15.4%*	*13.7%*	*15.4%*	*9.30%*	*0%*	*14.2%*	*0%*	*0%*

[1] Unemployment rate coefficient from regression where dependent variable is pollution concentration (not normalized).
[2] Unemployment rate coefficient from regression where dependent variable is mortality rate (in deaths per 100,000) in columns 1 through 4 and log mortality rate in columns 5 through 9).
[3] Units are deaths per 100,000 live births (change in death rate), in response to a one-unit change in pollution, in columns 1 through 4 (5 through 9). CO units are parts per million (ppm); PM10 units are $\mu g/m^3$.
[4] Calculated as the product of "Cyclicality of Pollution" and "Pollution on Mortality".
[5] Calculated as "Δ in Cyclical Deaths Explained" divided by "Cyclicality of Mortality".
[6] Chay and Greenstone study TSP. We compare to our results from PM10, using the EPA guidelines for comparing the two pollutants (Dockery and Pope 1994).
[7] Wellenius et. al. study PM2.5. We compare to our results from PM10, using the EPA guidelines for comparing the two pollutants (Dockery and Pope 1994).