Air Pollution and Procyclical Mortality

Garth Heutel, Christopher J. Ruhm

Abstract: Prior research demonstrates that mortality rates increase during economic booms and decrease during economic busts, but little is known about the role of environmental risks as a potential mechanism for this relationship. We investigate the contribution of air pollution to the procyclicality of deaths by combining county-level data on overall, cause-specific, and age-specific mortality rates with county-level measures of ambient concentrations of three types of pollutants and the unemployment rate. After controlling for demographic variables and state-by-year fixed effects, we find a significant positive correlation between pollution concentrations and mortality rates. Controlling for carbon monoxide, particulate matter, and ozone attenuates the relationship between overall mortality and the unemployment rate by 17%. The findings are robust to the use of state- rather than county-level data and to a variety of alternative specifications, although the attenuation of the unemployment-mortality relationship after controlling for pollution is insubstantial when including county-specific linear trends.

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Health is conventionally believed to deteriorate during macroeconomic downturns. However, substantial research conducted over the last decade 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 studies on this topic emphasized the

Garth Heutel (corresponding author) is at Georgia State University and the National Bureau of Economic Research (gheutel@gsu.edu). Christopher J. Ruhm is at the University of Virginia and the National Bureau of Economic Research (ruhm@virginia.edu). We thank Andrew Friedson, Matthew Neidell, Nicholas Sanders, and seminar and conference participants at the University of North Carolina at Greensboro, Appalachian State University, NBER, the 2012 AERE Summer Conference, and the 2013 and 2016 Allied Social Sciences Association meetings for helpful comments, and Kalee Burns, Will Mautz, Stephanie Norris, and James Small for excellent research assistance. Ruhm gratefully acknowledges financial support for this research from the University of Virginia Bankard Fund.
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 other 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 investigated in this context, probably because the data required to do so are difficult to analyze. This study provides a first step toward 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 county-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 (O₃)—into models that examine total, cause-specific, and age-specific mortality, while also controlling for county fixed effects and unemployment rates, state-specific 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 0.28%; this difference is statistically significant at the 10% level. All three pollutant concentrations exhibit a procyclical variation. CO is estimated to strongly increase mortality, and the inclusion of CO attenuates the estimated macroeconomic effect.¹ However, the high collinearity of the three pollution measures prevents us from making confident claims regarding differential effects between the pollutants. A one standard deviation increase in the CO concentration is associated with a 1.6% increase in the death rate, after controlling for county and year effects, demographic characteristics, and PM10 and O₃ levels (but not unemployment rates), and its inclusion in the full model attenuates the estimated unemployment coefficient by around 15%. This attenuation is insubstantial in models that include linear trends, though we argue that the inclusion of these trends may be a misspecification and that controlling for state-by-year effects is preferable.

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 that pollution has a significant effect on deaths from respiratory and cardiovascular disease,

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¹. The effect from CO only is consistent with results from Currie and Neidell (2005), Schlenker and Walker (2011), and Arceo, Rema, and Paulina (forthcoming).
and this is what we also find. In our estimates, a one standard deviation increase in the CO concentration is associated with a 3.0% rise in the respiratory mortality rate, and the inclusion of the pollution concentrations in our main model eliminates much of the correlation between respiratory fatalities and unemployment rates. Conversely, suicides are countercyclical and unaffected by pollution. The results for age-specific mortality are also revealing. Deaths are estimated to be procyclical for all age groups except those younger than 20, but only significantly so for > 84-year-olds. However, CO concentrations are associated with increased mortality for all groups. As a result, the procyclicality of fatalities is attenuated for these age groups—with a particularly strong but imprecisely measured reduction for the very old. We also measure the cyclicality of pollution itself, and we find that both PM10 and CO are significantly procyclical. A one percentage point increase in the unemployment rate is correlated with a 0.06 standard deviation drop in PM10 concentrations and a 0.04 standard deviation decrease in CO concentrations.

While these results are generally robust to alternate specifications, including running the analysis at the state-year level rather than the county-year level, several important caveats should be noted. As mentioned, it is difficult to identify differential effects between the three pollutants, and the attenuation of the unemployment coefficient after controlling for pollution is sensitive to the inclusion of linear trends. As described below, there are also concerns about bidirectional causality and migration bias that we cannot fully address using the available data. Future research should address these issues and consider extensions like controlling for more pollutants and elements beyond mean pollution levels (e.g., covariates for peak levels of pollutants), as well as possible instrumental variables such as the use of thermal inversions that temporarily raise pollution levels (Hicks, Marsh, and Oliva 2015).

1. BACKGROUND
Following Ruhm (2000), many studies examine the relationship between macroeconomic conditions and health by analyzing data for multiple locations and points in time. Panel data techniques can be used to control for many potential confounding factors. In particular, location-specific determinants of health that remain constant over time are easily accounted for, as are factors that vary over time in a uniform manner across locations. Death rates are useful to study because mortality represents the most severe negative health outcome and is objective and well measured and because diagnosis generally does not depend on access to the medical system (in contrast to many morbidities). Prior research provides strong evidence of a procyclical.

clical 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%–0.5% reduction in overall mortality, corresponding to an elasticity of $-0.02$ to $-0.05$, with significantly larger elasticity estimates sometimes obtained.\(^3\)

In explaining why health improves during economic downturns, researchers have emphasized the role of changes in lifestyles, hypothesizing that increased availability of nonmarket “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 and Tefft 2011) find that alcohol-involved vehicle mortality declines in such periods. Cardiovascular fatalities, which are strongly influenced by lifestyles, are also procyclical (Ruhm 2000; Neumayer 2004; Miller et al. 2009).

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 and Frakes (2006) and Xu (2013) 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. Consistent with these patterns, evidence that higher time prices correlate with increased obesity has been provided for adults and children (e.g., Courtemanche 2009).

However, changes in health behaviors are probably not the sole, or necessarily the most important, mechanism for procyclical variations in mortality. 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 related to obesity (Böckerman et al. 2006; Charles and DiCicca 2008; Arkes 2009), physical activity (An and Liu 2012; Colman and Dave 2013), and alcohol use (Dávalos et al. 2011).

Other risk factors provide potential alternative explanations for why health may improve during economic downturns. For example, traffic fatalities have been widely studied, with substantial and robust evidence provided that a one-point increase in unemployment reduces such deaths by 1%–3% (see Ruhm [2012] for citations). While these studies explore the mechanisms behind the procyclicality of mortality, no study attempts to quantify how much of the procyclicality can be explained by one particular mechanism.

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Another joint product of economic activity, air pollution, also presents health risks, especially for infants or senior citizens who do not participate in the labor force. A large economics literature examines the relationship between pollution exposure and health, accounting for potential confounders (Graff Zivin and Neidell 2013). Several studies link pollution to infant mortality. Currie and Neidell (2005) find that reductions in carbon monoxide in California over the 1990s saved approximately 1,000 infant lives. Chay and Greenstone (2003) use data from the 1981–82 recession to show that a 1% drop in total suspended particulates leads to a 0.35% reduction in infant mortality; TSP reductions nationwide from 1980 to 1982 led to 2,500 fewer infant deaths. Currie, Neidell, and Schmieder (2009), Greenstone and Hanna (2014), and Knittel, Miller, and Sanders (forthcoming) also find significant effects of pollution on infant deaths.

In addition to these economics papers, the epidemiological literature has linked pollution to mortality. Pollution is an established contributor to cardiovascular (Peters et al. 2004) and respiratory (Clancy et al. 2002) deaths. Mustafic et al. (2012) provides a meta-analysis linking both carbon monoxide and particulate matter to heart attack incidence, and Wellenius et al. (2012) tie particulate matter to strokes. Although pollution is correlated with mortality, it has not yet received attention in empirical research examining the effects of macroeconomic fluctuations on mortality. This analysis takes a first step toward rectifying this shortcoming.

2. 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. Studies based on aggregate data usually estimate some variant of:

\[ M_{jt} = \alpha_j + X_{jt}\beta + E_{jt}\gamma + \lambda_t + \epsilon_{jt}; \]

where \( M_{jt} \) is a health outcome (the log of the mortality rate) in location \( j \) at time \( t \), \( E \) measures macroeconomic conditions, \( X \) is a vector of covariates, \( \alpha \) is a location-specific fixed effect, \( \lambda \) a general time effect, and \( \epsilon \) is the regression error term. Following most previous research, the natural log (rather than level) of mortality rates is used.

Unemployment rates are the most common primary proxy for macroeconomic conditions, and the one focused upon here, although we also discuss results using an alternative measure. The supplementary characteristics include controls for the

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4. 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.
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 pollution data are unavailable prior to 1982, as is information on some of the covariates after 2009 (at the time of analysis). Our main analysis is at the county-year level; however, we also provide a corresponding state-level analysis in appendix B (apps. A–C available online). We report robust standard errors that are clustered at the state level.

The year effects, $\lambda_t$, 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 county fixed effects, $\alpha_j$, account for differences across locations that are time invariant (such as persistent lifestyle disparities between residents of counties in Nevada and Utah). We include state-by-year fixed effects, allowing the year effects $\lambda_t$ to vary across states. The impact of the macroeconomy is then identified from within-location variations relative to the changes in other areas. 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.

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

$$M_{jt} = \alpha_j + X_{jt}\beta + E_{jt}\gamma + A_{jt}\delta + \lambda_t + \epsilon_{jt};$$

where $A_{jt}$ 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), indicates 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

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5. Recent related research using county or MSA level data includes Charles and DeCicca (2008) and An and Liu (2012).

6. 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.

7. This specification will not pick up nonlinearities in the relationship between mortality and pollution. We are limited in how finely we can measure the shape of the dose-response function because of the aggregated nature of our data; however, we explore the effects of including quadratic terms of pollution in table 8.
on the true effect to the extent that pollution is only partially controlled for or is 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 pollution 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 pollution. Our estimates are for a log-linear model (the dependent variable is the log of the mortality rate and the right-hand-side variables are in levels) because this is consistent with most previous estimates and is likely to be the most appropriate specification.9

We test the significance of the change in the coefficient on unemployment between equations (1) and (2) using two methods. First, we calculated Wald statistics testing whether the unemployment coefficients differed in the models with and without pollution controls (using the Stata suest command). Second, we use the z-test statistic introduced by Gelbach (2016) to test the significance of this difference.10

Several potential issues with our estimation procedure deserve mention. First, including an endogenous variable (pollution) that is in the causal pathway from unemployment to mortality introduces a potential “bad control” bias (Angrist and Pischke 2009, 64–66). The issue here is that the coefficient estimated in the regression captures both the causal effect and potential selection bias. The latter occurs if, holding economic conditions constant, there are omitted determinants of mortality that are correlated with pollution levels. We test the significance of the change in the coefficient on unemployment between equations (1) and (2) using two methods. First, we calculated Wald statistics testing whether the unemployment coefficients differed in the models with and without pollution controls (using the Stata suest command). Second, we use the z-test statistic introduced by Gelbach (2016) to test the significance of this difference.10

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8. The correlation coefficient between our state-level measures of CO and PM10 is 0.582, between O₃ and PM10 it is 0.293, and between CO and O₃ it is 0.084.

9. In a log-linear specification, pollution changes are assumed to have common proportional rather than absolute effects on mortality (i.e., a 1% pollution increase is assumed to increase mortality by given percentage, whatever its initial level). By contrast, if mortality were specified in levels, an increase in pollution from 100 to 101 units would have the same effect as an increase from one to two units, which seems unlikely. In table 8 we investigate the robustness of our results to these and other alternate specifications.

10. The primary purpose of the Gelbach (2016) study is a critique of the use of sequentially adding control variables to a regression. While this does not apply to our analysis, the estimator that he provides (along with the Stata command b1x2) is an appropriate test. Intuitively, this approach runs the “base” regression (without pollution), then the “full” regression (with pollution), then provides a consistent estimate of the covariance matrix for the difference in the coefficient vector of the variables that are in both regressions (including unemployment). It is based on a Hausman test from a simple identity linking the base- and full-regression coefficients: \( \hat{\beta}_{1, \text{base}} = \hat{\beta}_{1, \text{full}} + (X_1'X_1)^{-1}X_1'X_2\hat{\beta}_2 \), where \( X_1 \) are the controls in both regressions and \( X_2 \) are the controls in just the full specification. Derivation of the covariance matrix is provided in Gelbach (2016), appendix B.
lated with pollution levels, which are in turn linked to unemployment rates. For instance, with cross-sectional data, it is easy to imagine how such confounding factors might be correlated with emissions. Though it cannot be measured, the selection bias is likely to be insubstantial given our use of panel data. We use extensive controls, including county fixed effects and state-by-year fixed effects, that capture variations in the pollution-specific treatment effects due to time-invariant location differences and all time-specific interstate variations.

There is also potential for bidirectional causality. Reduced economic activity, proxied by the unemployment rate, almost certainly decreases emissions. However, there is also some evidence that pollution negatively affects labor productivity (Graff Zivin and Neidell 2012) and work hours (Hanna and Oliva 2015). Pollution impacts on unemployment rates have not, to our knowledge, been examined but are possible. Our prior is that these effects are small and dominated by the effect of economic activity on pollution, particularly after including the extensive set of covariates.

Finally, our estimates could be vulnerable to migration bias. Migration could affect the estimates in three ways. First, migrants tend to move from areas of higher to lower unemployment rates, and healthy individuals are more likely to migrate than are those in poor health (Halliday 2007). This will introduce a countercyclical bias in the estimated average mortality effect. Second, these effects may vary with age. While such age variations have not been extensively studied, Halliday and Kimmitt (2008) offer tentative evidence that among older (≥60-year-old) men, but not women, migration is concentrated among both the top and bottom (versus the middle) of the health distribution. Since health shocks are most likely to cause death among those already in precarious health, it is possible that the estimated procyclicality of mortality among the elderly could be overstated. Third, migration that is unaccounted for

11. To show this, adopting Angrist and Pischke’s (2009, 64–66) notation: let \( y \) be the mortality rate, \( c \) be a binary indicator for unemployment rate (e.g., \( c = 1 \) represents low unemployment/good economy), and \( w \) be pollution. Unemployment then directly affects mortality and pollution according to \( y = cy_1 + (1 - c)y_0 \) and \( w = cw_1 + (1 - c)w_0 \), where \( y_1, y_0 \) is the mortality rate (pollution level) in locality \( i \) with low unemployment and \( y_0, w_0 \) are corresponding values with high unemployment rates. When regressing mortality on both unemployment and pollution, the unemployment coefficient measures: \( E[y_{1i} | w_1 = w] - E[y_{0i} | w_1 = w] = E[y_{1i} - y_{0i} | w_1 = w] \), \( E[y_{0i} | w_1 = w] = E[y_{0i} | w_1 = w] - E[y_{0i} | w_0 = w] \), \( E[y_{1i} - y_{0i} | w_1 = w] \). This selection bias cannot be definitively signed, without a fuller understanding of why the effects of pollution might differ in low- versus high-performing economies.

12. However, Halliday and Kimmitt (2008) also provide evidence of substantial attrition bias among unhealthy seniors and suggest that, for this reason, the finding of high migration rates among the unhealthy may be overstated or even incorrect.
in population estimates will lead to a bias, which may be positive or negative, in the
denominator of the mortality rate models. Migration to economically robust regions
will lead to a particularly large underestimation of population in these areas and an
overestimate of the procyclicality of mortality. To mitigate this issue, the data on
population estimates, described below, are the most accurate available and take partic-
ular account of noncensus year population flows. In addition, we note that even if
unaccounted-for migration patterns bias the estimates on the responsiveness of mor-
tality rates to economic conditions, they will not affect the attenuation in these result-
ing from adding pollution controls to the model, unless unaccounted-for migration flows
are independently correlated with emissions changes after controlling for observables.

3. DATA
Four primary data sources are used for this investigation: pollution levels from the
Environmental Protection Agency’s Air Quality System (AQS) database, unemploy-
ment rates from US Department of Labor’s Local Area Unemployment Statistics
(LAUS) database, mortality rates from the Centers for Disease Control and Preven-
tion’s Compressed Mortality Files (CMF), and population estimates from the Sur-
veillance Epidemiology and End Results (SEER) program of the National Cancer
Institute. 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 con-
centration data from monitors in the 50 United States and the District of Colum-
bia. 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 (O3).13 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 O3 were chosen from among the criteria pollutants
because of the large number of monitors in the AQS and because they have been
linked with health problems and mortality in previous research.14 For instance, Currie

13. CO is a by-product of combustion, and the majority of CO emissions come from mobile
sources (cars and trucks). PM10 is a mixture of small particles and liquid droplets. It includes
primary particles emitted directly from sources like construction sites or unpaved roads, and
secondary particles formed by reactions in the atmosphere of chemicals emitted from power
plants, industry, and automobiles. O3 is not directly emitted but is created by chemical reactions
between emissions of nitrogen oxides (NOX) and volatile organic compounds (VOCs). Major
sources of NOX and VOC include emissions from industrial facilities, electric utilities, and
motor vehicle exhaust. See http://www.epa.gov/air/urbanair/.

14. We also attempted to examine PM2.5 (particles smaller than 2.5 microns in dia-
ter) but were unable to do so because of the small amount of monitoring (no more than 40
monitors annually) prior to 1999.
and Neidell (2005) and Currie et al. (2009) find that infant mortality is positively and significantly related to CO exposure, while Chay and Greenstone (2003) find a correspondingly significant effect for particulate matter.\footnote{Chay and Greenstone examine total suspended particulates (TSPs), an older EPA designation that has been replaced by PM10 and PM2.5.}

Data on CO concentrations were available from a total of 1,470 monitors from 1980 to 2010; there were 4,144 monitors for PM10 between 1982 and 2010, and 2,799 O₃ 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.\footnote{We use only monitors reporting CO or O₃ concentrations at an hourly duration, and PM10 concentrations for a 24-hour duration. These are the most commonly used durations for the respective pollutants.} 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 active for 7 years, and data for just 65 CO monitors (4.4%) were available all 31 years. Similarly, the median PM10 monitor was in the data for 6 years, and fewer than 1.2% were available in all 29 years.

Because each county’s monitors are changing over time, considerable effort and experimentation were required to come up with meaningful location-specific pollution measures.\footnote{Currie and Neidell (2005) use data just from California monitors, and their results are unaffected by whether they use the subsample of monitors in the panel for the entire period or the entire unbalanced panel (see their n. 7).} If we only included counties that had a pollution monitor located within them, the analysis would be restricted to just 279 counties. Instead, we take advantage of the known location of each monitor (latitude and longitude) and use 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.\footnote{County population centroids are calculated by the US Census Bureau, based on the 2000 Census.} This substantially increases the analysis sample to 8,876 observations for 542 counties.\footnote{By contrast, adopting this method at the state level would substantially reduce the number of observations, since many monitored states do not have a monitor within 20 miles of the state population centroid. County-level regressions that include only counties containing pollution monitors yield unemployment coefficients that are almost twice as high as those using the larger sample of counties or the state-level regressions.} Our pollution measures certainly contain errors because we are attempting to identify average levels for the county
using monitors for a limited set of locations. We discuss alternative measures in appendix B, when describing our state-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 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 provides the official measure of the labor force for the nation. Our main specifications use annual average county unemployment rates as the key proxy for macroeconomic conditions. A consistent county-level unemployment rate data series is available beginning in 1990. We supplemented this by purchasing county-level unemployment data for earlier years from the Bureau of Labor Statistics (BLS). However, the BLS warns that these data are not fully comparable and cautions against their use in this way. This is one reason why we will also report (in app. B) the results of a full state-level analysis, since consistent state unemployment rates are available throughout the entire analysis period.

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 publicly available; those from 1989 to 2009 were obtained through a special agreement with the CDC. The CMF include a record for every death of a US 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 census-based population estimates; however, instead of using these, we obtained population information from the SEER program (http://www.seer.cancer.gov/data), because these are designed to be more reliable than census estimates.

20. Chay and Greenstone (2003, 419–20) 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.

21. The SEER data are designed to provide more accurate population estimates than standard census projections for the intercensal years and provide additional adjustments for population shifts in 2005, resulting from Hurricanes Katrina and Rita. Differences between the SEER and CMF population estimates are miniscule prior to 2000 but, for some states, become reasonably large (up to 3%) after 2003.
Using the CMF mortality and SEER population data, we constructed dependent variables for the natural logs of annual total mortality rates; annual mortality rates for eight age groups—infants, 1–19, 20–44, 45–54, 55–64, 65–74, 75–84, and ≥85-year-olds; and mortality rates due to 11 specific causes—respiratory, cardiovascular, acute myocardial infarction (heart attack), ischemic heart disease, cerebrovascular disease (stroke), cancer, accidents (total, vehicular, and nonvehicle), suicide, and homicide. 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). From the SEER population data, we also constructed regression controls for the share of the county population who were female, black, other nonwhite, and aged <1, 1–19, 45–54, 55–64, 65–74, 75–84 and ≥85 years old.

Although our main estimates use counties as the unit of observation, there are several reasons why a state-year level analysis has advantages. First, we are interested in examining the extent to which pollution provides a mechanism explaining the procyclical variation in mortality found in many previous studies, most of which were conducted at the state level. Second, while there are potentially significant within-state disparities in pollution and unemployment rates, there is also likely to be more error in the measurement of both mortality and unemployment rates at smaller geographic units. Smaller counties will also sometimes have zero deaths for some causes or age groups in some years. Third, as mentioned, issues of data comparability across time also become more pronounced when using county data—for instance, a consistent unemployment rate series is only available beginning in 1990. Fourth, we can control for additional characteristics for which we are able to obtain data at the state but not the county level, as described below. (However, we cannot include state-year effects in these models.) Finally, there is a question about the level of geographic aggregation at which the macroeconomic effects actually occur. In this regard, Lindo’s (2015, 83) analysis is particularly instructive: he concludes that “more disaggregated analyses—particularly county-level analyses—routinely [produce] estimates that are smaller in magnitude.” For all of these reasons, we provide a full replication of results at the state level (see app. B) as well as a summary of findings obtained when using

22. The greater measurement error in county than state unemployment rates is well known (see, e.g., Ganong and Liebman 2013). 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.

23. We replace these zero values with one so that we can take the natural log. 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.
county-level mortality and pollution data but measuring macroeconomic conditions at the state level.

As mentioned, we included a number of additional location-specific characteristics in our state models. State-year level education shares were obtained from the Current Population Survey (CPS) March Annual Demographic Survey for the share of the population (25 and older) who were high school graduates (without college), had attended but not graduated from college, and who were college graduates (with high school dropouts the reference group). Since weather can affect mortality directly and the relationship between pollution and mortality (Schwartz 1994; Samet et al. 1998), we control for average temperature and precipitation using data from the National Centers for Environmental Information at the National Oceanic and Atmospheric Administration. Since the relationship between temperature and mortality is nonlinear, we include a set of indicator variables for 10-degree-Fahrenheit temperature bins, following Deschênes and Greenstone (2011). We also control for the state-year level Gini coefficient, the proportion of the state’s state representatives that are Democrat, and the urbanization rate in the state.

Table 1 presents summary statistics separately for in-sample and out-of-sample counties. The former have lower unemployment and mortality rates but similar observable demographic characteristics. PM10 concentrations are measured in micrograms per cubic meter (μg/m³), and CO and O₃ concentrations in parts per million.

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24. Available at http://www.ncdc.noaa.gov/. These data are not available for Hawaii and so we omit them from our analysis. We also estimated and obtained robust results from models that controlled for heating and cooling degree days and the Palmer drought severity index, with data available in all states except Hawaii, Alaska, and Washington, DC. Our state-level measures of weather are calculated by the National Centers for Environmental Information (formerly the National Climatic Data Center), who provide data at the state level, and for some US cities and climate divisions, but not all counties. Rather than redefine county-level measures using, for instance, monitor data, we allow the state-by-year fixed effects to account for weather in our county-level regressions. Likewise, the education variables from the CPS are calculated at the state, not county, level, so rather than interpolating from census years we omit them. Because of the relative insignificance of education and weather variables in the state-level mortality regressions, we do not think these exclusions are important.

25. The state-level urbanization rate is calculated in census years only; we linearly interpolate the values for other years. Gini coefficients are available at http://www.shsu.edu/eco_mwf/inequality.html; the political data are from Klarner (2003) and updates are available at http://www.indstate.edu/polisci/klarnerpolitics.htm. The District of Columbia does not have a State House, so its political measure is based on its US House delegate (always Democrat). Unicameral Nebraska’s political measure is based on the party of its governor.

26. The in-sample counties include counties from all states except Alaska, Hawaii, and Wyoming and represent 57% of the total US population. The large number of counties, and the fact that nearly half of the US population lives in counties without a pollution monitor within 20 miles, highlights the noise in the state-level pollution measure.
Table 1. Selected Variable Means for In-Sample and Out-of-Sample Counties

<table>
<thead>
<tr>
<th></th>
<th>In Sample</th>
<th>Standard Error</th>
<th>Out of Sample</th>
<th>Standard Error</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>County unemployment rate (%)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>5.489</td>
<td>.027</td>
<td>6.754</td>
<td>.013</td>
</tr>
<tr>
<td><strong>Mortality rates (per 1,000):</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>8.196</td>
<td>.023</td>
<td>10.301</td>
<td>.010</td>
</tr>
<tr>
<td>Respiratory {466–96}, [J20–J47, U04]</td>
<td>.570</td>
<td>.003</td>
<td>.727</td>
<td>.001</td>
</tr>
<tr>
<td>Cardiovascular {390–448}, [I00–I178]</td>
<td>3.253</td>
<td>.012</td>
<td>4.332</td>
<td>.005</td>
</tr>
<tr>
<td>Acute myocardial infarction {410}, [I21–22]</td>
<td>.727</td>
<td>.004</td>
<td>1.195</td>
<td>.003</td>
</tr>
<tr>
<td>Ischemic heart disease {410–14}, [I20–I125]</td>
<td>1.316</td>
<td>.009</td>
<td>1.813</td>
<td>.004</td>
</tr>
<tr>
<td>Cerebrovascular disease {430–38}, [I60–I69]</td>
<td>.544</td>
<td>.002</td>
<td>.757</td>
<td>.001</td>
</tr>
<tr>
<td>Cancer {140–208}, [C00–C97]</td>
<td>1.922</td>
<td>.005</td>
<td>2.257</td>
<td>.003</td>
</tr>
<tr>
<td>Accident {E800–E869, E880–E929}, [V01–X59, Y85–Y86]</td>
<td>.353</td>
<td>.001</td>
<td>.551</td>
<td>.001</td>
</tr>
<tr>
<td>Vehicle accident {E810–E825}, [V02–V89]</td>
<td>.158</td>
<td>.001</td>
<td>.29</td>
<td>.001</td>
</tr>
<tr>
<td>Suicide {E950–E959}, [X60–X84, Y87.0]</td>
<td>.121</td>
<td>.001</td>
<td>.171</td>
<td>.000</td>
</tr>
<tr>
<td>Homicide {E960–E978}, [X85–Y09, Y87.1, Y35, Y89.0]</td>
<td>.072</td>
<td>.001</td>
<td>.112</td>
<td>.000</td>
</tr>
<tr>
<td>&lt; 1 year old</td>
<td>8.314</td>
<td>.061</td>
<td>12.21</td>
<td>.040</td>
</tr>
<tr>
<td>1–19 years old</td>
<td>.381</td>
<td>.003</td>
<td>.623</td>
<td>.002</td>
</tr>
<tr>
<td>20–44 years old</td>
<td>1.438</td>
<td>.006</td>
<td>1.721</td>
<td>.003</td>
</tr>
<tr>
<td>45–54 years old</td>
<td>4.303</td>
<td>.015</td>
<td>4.879</td>
<td>.008</td>
</tr>
<tr>
<td>55–64 years old</td>
<td>10.446</td>
<td>.030</td>
<td>11.257</td>
<td>.014</td>
</tr>
<tr>
<td>65–74 years old</td>
<td>24.852</td>
<td>.056</td>
<td>25.514</td>
<td>.024</td>
</tr>
<tr>
<td>75–84 years old</td>
<td>58.500</td>
<td>.094</td>
<td>59.168</td>
<td>.046</td>
</tr>
<tr>
<td>≥ 85 years old</td>
<td>154.63</td>
<td>.245</td>
<td>156.607</td>
<td>.138</td>
</tr>
<tr>
<td><strong>State population shares:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 1 year old</td>
<td>.014</td>
<td>.000</td>
<td>.014</td>
<td>.000</td>
</tr>
<tr>
<td>1–19 years old</td>
<td>.271</td>
<td>.000</td>
<td>.277</td>
<td>.000</td>
</tr>
<tr>
<td>20–44 years old</td>
<td>.381</td>
<td>.000</td>
<td>.336</td>
<td>.000</td>
</tr>
<tr>
<td>45–54 years old</td>
<td>.125</td>
<td>.000</td>
<td>.121</td>
<td>.000</td>
</tr>
<tr>
<td>55–64 years old</td>
<td>.090</td>
<td>.000</td>
<td>.102</td>
<td>.000</td>
</tr>
<tr>
<td>65–74 years old</td>
<td>.066</td>
<td>.000</td>
<td>.082</td>
<td>.000</td>
</tr>
</tbody>
</table>
Figure 1 presents scatter plots of each of the three pollution levels versus the overall mortality rate and does not provide evidence of a strong relationship between the two; this is not surprising since potentially important confounding factors have not been controlled for. Appendix figures A1–A3 show state-specific annual average ambient concentrations for each of the three pollutants. PM10 and CO trend downward over time in most states, while O3 is relatively flat, but with substantial year-by-year deviations from trends for all three pollutants.

4. RESULTS

4.1. 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. In addition to providing supporting evidence for our main question about mortality over the business cycle, the relationship between emissions and business cycles is interesting in and of itself. Surprisingly, we found few other papers directly investigating this issue.27

Table 2 presents regression results where the dependent variables are standardized pollution measures (with mean zero and standard deviation one) for PM10 (cols. 1 and 2), CO (cols. 3 and 4), and O3 (cols. 5 and 6). All regressions are weighted by

<table>
<thead>
<tr>
<th></th>
<th>In Sample</th>
<th></th>
<th>Out of Sample</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>Standard Error</td>
<td>Mean</td>
<td>Standard Error</td>
</tr>
<tr>
<td>75–84 years old</td>
<td>.040</td>
<td>.000</td>
<td>.051</td>
<td>.000</td>
</tr>
<tr>
<td>≥ 85 years old</td>
<td>.013</td>
<td>.000</td>
<td>.018</td>
<td>.000</td>
</tr>
<tr>
<td>Female</td>
<td>.511</td>
<td>.000</td>
<td>.506</td>
<td>.000</td>
</tr>
<tr>
<td>Black</td>
<td>.111</td>
<td>.001</td>
<td>.088</td>
<td>.001</td>
</tr>
<tr>
<td>Other nonwhite</td>
<td>.037</td>
<td>.000</td>
<td>.025</td>
<td>.000</td>
</tr>
</tbody>
</table>

Note.—Summary statistics are over the county-year observations, from 1982 to 2009. The left half of the table includes those 8,876 observations for which we have PM10, CO, and O3 concentrations and data on all control variables. The right half includes all of the other observations. ICD-9 codes for specific causes of death categories applying from 1982 to 1998 are shown in braces; corresponding ICD-10 codes, used from 1999 on are displayed in brackets.

27. Heutel (2012) documents the procyclicality of carbon dioxide (CO2) emissions at the quarterly level. Using ARIMA regressions, he estimates the elasticity between US GDP and CO2 emissions levels to be between 0.5 and 0.9. Smith and Wolloh (2012) find that aggregate water quality in the United States is positively correlated with the national unemployment rate.
Figure 1. Unadjusted scatter plots of pollution levels versus mortality rates. Each panel plots the state-year observations of the average (nonstandardized) pollution level versus the total mortality rate for the 1,160 state-year observations included in the state-level data set.
the county’s population and include county fixed effects and state-by-year fixed effects (not reported). Columns 2, 4, and 6 also control for the county-year level population shares in the specified age, gender, and race/ethnicity categories.

As hypothesized, there is a negative relationship between the unemployment rate and all three pollution measures. A one percentage point increase in joblessness is associated with about a 0.06 standard deviation decrease in the ambient PM10 concentration and about 0.04 standard deviations reduction in ambient CO concentrations. The relationship between O3 and unemployment is of similar magnitude but not quite statistically significant. Controlling for the demographic covariates increases the size and significance of these correlations. These estimates verify our expectation that pollution is procyclical.

In appendix tables A1 and A2, we present results of an extensive sensitivity analysis that compares specifications varying according to the level of the observations (state or county), the inclusion of trends and fixed effects, and the level of clustering standard errors. Each reported coefficient in appendix table A1 is from a separate regression where the dependent variable is the specified pollution measure and the reported coefficient is the coefficient on the unemployment rate. In each specification there is a significant correlation between unemployment and at least one of the pollutants, though which pollutant enters significantly depends on the

28. Columns 7 and 8 of appendix table A1 present results from regressions run at the county level but with the unemployment rate measured at the state level, as suggested by Lindo (2015).
specification. Because of the high collinearity between the pollutants, we should interpret with caution any differences between the effects of the three pollutants. In the specifications with linear trends, the correlation between pollution and unemployment is the weakest, and in column 8 there is a significant positive correlation between ozone and unemployment. We suspect that this arises from a misspecification when including linear trends. This claim is bolstered by recent findings in the literature examining the effect of minimum wage laws on employment (Neumark, Salas, and Wascher 2014; Sabia 2014), suggesting that including linear time trends in these models eliminates potentially valid sources of identifying variation. Last, there is little difference between the magnitudes of the state-clustered and the county-clustered standard errors. For these reasons, we choose for our main results throughout the rest of the paper to present the county-level regressions with state-by-year fixed effects, rather than state-specific or county-specific linear trends, and errors clustered at the state level (col. 6).  

Most coefficients on the demographic variables are statistically insignificant (app. table A3). The percentage of the population that is nonwhite is not significantly related to pollution after controlling for unemployment. Higher population shares in younger age brackets (below 20 years old) and middle-aged age brackets (45–64 years old), relative to the excluded bracket (20–44 years old), are correlated with higher PM10 and CO pollution. A higher female population share is negatively correlated with all three pollutants but only significantly so for CO. The age population shares are economically significant, when comparing their magnitudes to those of the unemployment rate. For instance, a one percentage point increase in the 1–19 years old age share is correlated with a 0.223 standard deviation increase in CO concentrations, compared to the 0.04 standard deviation increase correlated with a one percentage point increase in the unemployment rate.  

4.2. 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 death

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29. State-by-year fixed effects are more flexible than state-specific linear trends. Including both state-by-year fixed effects and county-specific linear trends yields results that are generally unstable and inconsistent because of limited degrees of freedom (there are 542 counties, so 542 county fixed effect variables plus 542 county-specific linear trend, and 48 states and 28 years, implying 1,344 state-by-year fixed effects, in a total sample of 8,876 county-year observations). We also included both linear and quadratic trends, and the results were generally comparable to including just linear trends.

30. More demographic and control variables are available at the state-year level, which we can control for in our state-year level analysis that does not include state-by-year fixed effects (see sec. 4.5 and app. B).
rate. All specifications control for demographic variables and county fixed effects, as well as the county 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, although the coefficient is not statistically significant.31 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 Stevens et al.’s (2011) finding that the estimated procyclical variation of mortality is somewhat attenuated when adding post-1991 observations to the model.32 Among the demographic coefficients,

Table 3. Econometric Estimates of the Determinants of Total Mortality

<table>
<thead>
<tr>
<th>Regressor</th>
<th>(1)</th>
<th>(2)</th>
<th>(3)</th>
<th>(4)</th>
<th>(5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>County unemployment rate (%)</td>
<td>−.00346</td>
<td>−.00316</td>
<td>−.00285</td>
<td>−.00349</td>
<td>−.00286</td>
</tr>
<tr>
<td></td>
<td>(.00330)</td>
<td>(.00317)</td>
<td>(.00301)</td>
<td>(.00328)</td>
<td>(.00306)</td>
</tr>
<tr>
<td>PM10</td>
<td>.00544***</td>
<td>.00285*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(.00108)</td>
<td>(.00157)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CO</td>
<td>.0156***</td>
<td>.0160***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(.00436)</td>
<td>(.00526)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>O3</td>
<td></td>
<td>−.000771</td>
<td>−.00594*</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(.00223)</td>
<td>(.00228)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>F-statistic from Wald test</td>
<td>4.51</td>
<td>2.63</td>
<td>.17</td>
<td>3.76</td>
<td></td>
</tr>
<tr>
<td>(P-value)</td>
<td>(.0337)</td>
<td>(.1052)</td>
<td>(.6845)</td>
<td>(.0524)</td>
<td></td>
</tr>
<tr>
<td>Z-statistic from Gelbach test</td>
<td>2.09</td>
<td>1.59</td>
<td>.36</td>
<td>1.90</td>
<td></td>
</tr>
<tr>
<td>(P-value)</td>
<td>(.037)</td>
<td>(.112)</td>
<td>(.717)</td>
<td>(.057)</td>
<td></td>
</tr>
<tr>
<td>R-squared</td>
<td>.988</td>
<td>.988</td>
<td>.988</td>
<td>.988</td>
<td>.988</td>
</tr>
</tbody>
</table>

Note.—The dependent variable is the natural log of the total mortality rate (n = 8,876). State-by-year fixed effects are included but not reported. All models also include controls for the share of county residents who are female, black, other nonwhite, and seven age groups (<1, 1–19, 45–54, 55–64, 65–75, 75–84, and ≥85 years old). Regressions are weighted by the county population. Standard errors, clustered at the state level, are reported in parentheses. The F-statistic and Z-statistic refer to tests on whether the unemployment coefficients in models 2 through 5 differ significantly from that in model 1. The Z-statistic uses the procedure described in Gelbach (2016).

* p < .10.
** p < .05.
*** p < .01.

Air Pollution and Procyclical Mortality

31. When the standard errors are clustered at the county level, this coefficient is significant at the 10% level (see appendix table A2).
32. Using data from 1978–2006 and a specification similar to that in column 1, they obtain an unemployment coefficient of −.0019. See Ruhm (2015) for a detailed analysis confirming that the procyclical variation in mortality has weakened in recent years.
Age has the expected effect on mortality, with higher shares of senior citizens being correlated with higher mortality rates. The coefficient on the “other” race category (nonwhite and nonblack) is negative, and the coefficient on the black share is positive. Gender is insignificant (see app. table A4).

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, O3 concentrations in column 4, and all three pollution measures simultaneously in column 5. PM10 and CO, each when only one pollutant is included, are positively correlated with mortality and attenuate the predicted macroeconomic effect. The point estimate on CO suggests that a one standard deviation increase in CO predicts a 1.6% rise in the mortality rate. O3 when included alone is uncorrelated with mortality and does not affect the relationship between unemployment and mortality. Controlling for all three pollutants together (col. 5), the CO coefficient is the only one that is positive and statistically significant at the 1% level, with a one standard deviation increase still predicting a 1.6% rise in mortality. O3 has a negative relationship with mortality that is just barely significant, and the relationship between PM10 and mortality is positive but just half as big as in column 2. Given this pattern, we primarily focus on the results for CO below, while controlling for all three types of pollution in our models.33 However, as mentioned earlier, the results in appendix tables A1 and A2 demonstrate that we should interpret with caution any difference in the predicted effects of the three pollutants.

Adding CO pollution to the model cuts the unemployment rate coefficient by 17%–18%, depending on whether the other two pollutants are included in the regression. In the basic specification (col. 1), a one percentage point increase in unemployment reduces predicted mortality by 0.35%; this falls to 0.29% when all three pollution concentrations are controlled for (col. 5).34 Both test statistics and p-values are presented in the respective columns in table 3. A Wald test shows that the difference between the two coefficients is significant at the 10% level when all three

33. Stronger results for CO than other pollutants are consistent with the findings of other researchers. Currie and Neidell’s (2005) study of infant mortality also uncovers significant effects of CO, but not PM10 or O3, concentrations. Beatty and Shimshack (2014) investigate all three pollutants’ effects on childhood morbidity and find effects from CO but not PM10. Arceo et al. (forthcoming) examines infant mortality in Mexico and obtains significant effects from both PM10 and CO, but their estimated magnitudes of CO coefficients are larger than those found in the United States.

34. Appendix table A4 also presents results for a model that controls for the pollution measures but not the unemployment rate. When doing so, CO is positively correlated with total mortality at the 1% level, while PM10 is insignificantly related to it and O3 is barely negatively related (see col. 6 of table A4). Coefficients for the remaining right-hand-side variables are also quite similar to those in our main specifications.
pollutants are controlled for. The Gelbach (2016) z-statistic likewise indicates that
the inclusion of pollutants attenuates the unemployment coefficient at the 10% level.
Thus, our results should be interpreted as providing reasonably strong evidence that
pollution affects the relationship between business cycles and mortality.

Appendix table A2 repeats the same sensitivity analysis that was presented in
appendix table A1, though here it presents regression results of the relationship
between unemployment and mortality, with and without pollution controls (as in
table 3). In all specifications we observe the expected negative relationship between
unemployment and mortality, but its magnitude and significance varies across spec-
fications. In our preferred specification (col. 6), this correlation is not quite signifi-
cant (although it is significant based on the standard errors clustered at the county
level), and in this respect our results presented in the main tables throughout the
paper are conservative. In the specifications that include linear trends, the attenuation
of the unemployment coefficient after including pollution controls is often insubstan-
tial. As described above, we argue that including linear trends, rather than state-by-
year fixed effects, is a misspecification. Nevertheless, we emphasize that our finding
that including pollution controls attenuates the relationship between unemployment
and mortality is sensitive to the inclusion of linear trends, rather than state-year fixed
effects.35

Attenuation bias may plague these estimates, especially because of the potential
for measurement error in the pollution variables.36 We conduct errors-in-variables
regressions, allowing for different levels of reliability in the pollution measures to
see how attenuation bias affects the pollution and unemployment coefficients.37 The
results are reported in appendix table A5. This test is limited in important ways.38
But the results suggest that measurement error may be causing a downward bias

35. See table 8 for a model that includes both linear and quadratic time trends.
36. Measurement error in the pollution variables is likely to be more severe at the state
than at the county level, while the opposite is likely to be true for measurement error in the
unemployment rate. See appendix B.
37. Errors-in-variables regression is a method for accounting for attenuation bias. For a
specified level of reliability in a set of independent variables, an errors-in-variables regression
will provide updated coefficient estimates for all independent variables. While it is known
that attenuation bias pushes the coefficients of the mismeasured variables to zero, errors-in-
variables regression gives information about the direction of bias of the other variables.
38. First, clustering of standard errors is only possible with bootstrapping. With boot-
strapping, population weighting is not allowed, and it is not possible to include the large
number of county and state-by-year fixed effects. So, we do not bootstrap and instead use
regressions with nonrobust standard errors. Second, the reliability ratios cannot be lower than
the R-squared values from regressions of the pollution measures on all other controls (0.89 for
PM10, 0.92 for CO, and 0.94 for ozone), so we are unable to test under low reliability ratios.
(more negative) on our estimated unemployment coefficients, and therefore that pollution may attenuate the unemployment coefficient by a larger amount than appears in our estimates. This implies that our reported results are conservative.

4.3. Age-Specific Mortality

Miller et al. (2009) find that procyclical variations in mortality are particularly pronounced among the young and old—who are seldom directly involved in the labor market. Changes in pollution levels could explain some of these patterns, since the health of these groups might be especially vulnerable to environmental risks, and negative shocks might be relatively likely to result in death. We address this possibility in table 4, which summarizes estimation results for the mortality rates of eight age groups: <1, 1–19, 20–44, 45–54, 55–64, 65–74, 75–84, and >84-year-olds. For each age group, table 4 presents the results of two specifications. Column a controls for county unemployment rates, county and state-by-year effects, and demographic variables, but not air pollution. Column b adds controls for the standardized pollution levels.

In the basic model (col. a), unemployment is negatively correlated with the mortality rates of all age groups except infants and 1–19-year-olds, although the relationship is significant only for the oldest age group. A one point increase in the unemployment rate is predicted to reduce the mortality of >84-year-olds by 0.44%. This compares to statistically insignificant decreases of 0.62%, 0.29%, 0.22%, 0.09%, and 0.22% for 20–44, 45–54, 55–64, 65–74, and 75–84-year-olds and insignificant increases of 0.28% and 0.46% for infants and 1–19-year-olds.

When adding controls for pollution concentrations (specification b), we see the hypothesized attenuation of the macroeconomic coefficients for elderly: the unemployment coefficient declines, in absolute value, by 23%, 12%, and 9% for 65–74, 75–84, and >84-year-olds. However, none of these changes are statistically significant. The only statistically significant difference between unemployment coefficients occurs for 20–44-year-olds. It is noteworthy that the large (though insignificant) macroeconomic fluctuations in deaths of 1–19-year-olds are not substantially affected by the inclusion of the pollution variables, which makes sense if these deaths occur for reasons that are largely unrelated to environmental risks.

As above, PM10 and

39. Compared to the least squares regressions, with a reliability ratio of 0.96 on all three pollution measures, the unemployment coefficient drops by 11%, from −0.00286 to −0.00254.

40. Much of the prior literature on the health effects of pollution focuses on infant mortality (Chay and Greenstone 2003; Dehejia and Llenas-Muney 2004; Currie and Neidell 2005; Currie et al. 2009; Greenstone and Hanna 2014; Knittel et al., forthcoming).

41. Observations are weighted by county population in the specified age category.

42. 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 and Kochanek 2012).
Table 4. Econometric Estimates of Determinants of Age-Specific Mortality

<table>
<thead>
<tr>
<th>Regressor</th>
<th>&lt; 1-Year-Olds</th>
<th>1–19-Year-Olds</th>
<th>20–44-Year-Olds</th>
<th>45–54-Year-Olds</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(a)</td>
<td>(b)</td>
<td>(a)</td>
<td>(b)</td>
</tr>
<tr>
<td>County unemployment rate (%)</td>
<td>.00272 (.00602)</td>
<td>.00435 (.00533)</td>
<td>.00411 (.00651)</td>
<td>.00346 (.00650)</td>
</tr>
<tr>
<td>PM10</td>
<td>-.000862 (.00904)</td>
<td>-.0103* (.00608)</td>
<td>-.00312 (.00231)</td>
<td>-.00283 (.00214)</td>
</tr>
<tr>
<td>CO</td>
<td>.0501*** (.0150)</td>
<td>.0106 (.0186)</td>
<td>.0532** (.0214)</td>
<td>.0193 (.0127)</td>
</tr>
<tr>
<td>O3</td>
<td>-.00835** (.00623)</td>
<td>-.00393* (.00474)</td>
<td>-.00835** (.00400)</td>
<td>-.00393* (.00200)</td>
</tr>
<tr>
<td>55–64-Year-Olds</td>
<td>.00268 (.0295)</td>
<td>.00295* (.00175)</td>
<td>.00203 (.00183)</td>
<td>.00238 (.00655)</td>
</tr>
<tr>
<td>65–74-Year-Olds</td>
<td>.00923 (.00623)</td>
<td>.00659 (.00474)</td>
<td>.00659 (.00474)</td>
<td>.00659 (.00474)</td>
</tr>
<tr>
<td>75–84-Year-Olds</td>
<td>.00923 (.00623)</td>
<td>.00659 (.00474)</td>
<td>.00659 (.00474)</td>
<td>.00659 (.00474)</td>
</tr>
<tr>
<td>&gt;84-Year-Olds</td>
<td>.00923 (.00623)</td>
<td>.00659 (.00474)</td>
<td>.00659 (.00474)</td>
<td>.00659 (.00474)</td>
</tr>
</tbody>
</table>

Note.—The dependent variables are natural logs of the specified age-specific mortality rate. All models control for state-by-year fixed effects and county-year level demographic characteristics. Observations are weighted by the county population within each age group. Standard errors, clustered at the state level, are reported in parentheses. For 20–44-year-old deaths, the unemployment coefficient is significantly different between columns a and b. In all other columns, there is no statistically significant difference between the unemployment coefficient in columns a and b.

* p < .10.
** p < .05.
*** p < .01.
O3, are with a few exceptions, not significantly related to the mortality rates of any of the groups, whereas CO concentrations are predicted to significantly increase death rates for all eight age categories.

4.4. Cause-Specific Mortality
The evidence that changes in air pollution explain a portion of the procyclical fluctuation in mortality will be strengthened if the unemployment coefficients are more sharply attenuated after controlling for emissions for fatalities that we expect to be strongly related to pollution 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).43 We examine this in table 5 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).

Mortality rates are negatively correlated with unemployment rates for four of the six causes of death in the basic model (col. a), without controlling for pollution, although the association is not significant.44 Cardiovascular deaths are the most procyclical—a one percentage point increase in the unemployment rate decreases predicted cardiovascular deaths by 0.66%. The same rise in unemployment is estimated to reduce mortality from respiratory disease, heart attacks, ischemic heart disease, and stroke by 0.3%–0.4%. Cancer fatalities are unrelated to macroeconomic conditions, as has been found previously (Ruhm 2000).

The pollution measures are added as controls in specification b. Based on the literature, we hypothesize that pollution will increase respiratory deaths and possibly cardiovascular deaths. PM10 and O3 usually do not have a significant effect, consistent with the results for total mortality, except for a barely significant positive coefficient on PM10 for cardiovascular fatalities. Conversely, ambient CO levels are significantly positively associated with respiratory, cardiovascular, and ischemic heart disease deaths: a one standard deviation increase in CO concentrations predicts 3.0%, 0.9%, and 2.6% increases in mortality from these causes. The coefficient on CO is also positive and significant for heart attack and stroke deaths. This is consistent with earlier findings of a positive effect of pollution for these causes of death. The coefficient on CO for cancer deaths is barely significant and small.

43. 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.

44. When errors are clustered at the county level, the coefficient for cardiovascular deaths is significant.
Controlling for pollution reduces the magnitude of the unemployment coefficient for respiratory deaths by about 40%, although the original effect was of small magnitude and imprecisely estimated. A Wald test shows that this difference is significant at the 5% level. It slightly attenuates the predicted effect on cardiovascular fatalities, but when focusing on ischemic heart disease, which is likely to be responsive to economic conditions, the unemployment coefficient is significantly different between columns a and b. In all other columns, there is no statistically significant difference between the unemployment coefficients in columns a and b.

* $p < .10$.
** $p < .05$.
*** $p < .01$.

Note.—The dependent variables are natural logs of the specified cause-specific mortality rate ($n = 8,876$). All models control for state-by-year fixed effects and county-year level demographic characteristics. Observations are weighted by the county population. Standard errors, clustered at the state level, are reported in parentheses. For respiratory deaths, the unemployment coefficient is significantly different between columns a and b. In all other columns, there is no statistically significant difference between the unemployment coefficient in columns a and b.
to short-term triggers and changes in risk factors, the magnitude of the coefficient actually increases. The estimated unemployment effect declines by 19% for stroke deaths and by 10% for heart attack fatalities. With the exception of respiratory deaths, none of these changes are statistically significant.

As a placebo test, table 6 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, and the others are countercyclical, though none of the relationships is significant. 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. Surprisingly, PM10 pollution is positively correlated with nonvehicle accidental deaths, and controlling for it increases the magnitude of the (positive) unemployment coefficient. A potential explanation is that many deaths in this category occur at work and employment in “polluting” industries is less safe, so that when production rises, both emissions and nonvehicle accidents increase. The data do not distinguish whether deaths occurred on the job, but 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. We replicated the regressions for nonvehicle accidental

45. Pope et al. (2006) find a link between short-term pollution exposure and ischemic heart disease.

46. We also replicate table 5’s regressions restricting the deaths to just infants or to just those 85 years old or older, since mortality rates among these groups may be the most sensitive to pollution and to the business cycle. The causes of death from table 5 are not major contributors to infant mortality, so there was little relationship between those death rates and unemployment. The correlation between CO and infant respiratory mortality is about five times as large as the correlation between CO and overall respiratory mortality. For those 85 years and older, cardiovascular deaths and stroke deaths were significantly procyclical.

47. These results are consistent with prior research findings (e.g., Ruhm 2000), except that a procyclical variation in nonvehicle accidents and homicides has sometimes previously been found. Ruhm (2015) provides evidence that nonvehicle accidents have shifted from being procyclical to countercyclical over time.

48. Controlling for per capita miles driven (using data from http://www.fhwa.dot.gov/policyinformation/statistics.cfm), which are significantly correlated with the vehicle fatality rate, does not substantially alter these results.

49. For instance, using data for 2009 from http://www.bls.gov/iif/oshwc/cfoi/cftb0241.pdf (accessed September 21, 2015), we calculate that construction industries had a nonvehicle fatal accident rate that was over four times as large as that for all industries (6.9 versus 1.5 per 100,000 workers) and that this industry accounted for over 30% of such occupational mortality in that year.
deaths separately for working age (20–64) and non–working age people. In concor-
dance with our hypothesis, the coefficient on PM10 for working age deaths is about
50% larger than the coefficient for non–working age deaths, but both are significantly
positive.

4.5. State Analysis
We replicated our analysis using state- rather than county-level data. The proce-
dures for doing so, issues raised in such an analysis, and the results obtained are

### Table 6. Econometric Estimates of the Determinants of External Causes of Death

<table>
<thead>
<tr>
<th>Regressor</th>
<th>Vehicle Accident</th>
<th>Nonvehicle Accident</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(a)</td>
<td>(b)</td>
</tr>
<tr>
<td>County unemployment</td>
<td>–.00471 (.00323)</td>
<td>–.00534 (.00338)</td>
</tr>
<tr>
<td>rate (%)</td>
<td>–.00398 (.00665)</td>
<td>.0232*** (.00748)</td>
</tr>
<tr>
<td>PM10</td>
<td>–.00117 (.0136)</td>
<td>.0148</td>
</tr>
<tr>
<td></td>
<td>–.0114 (.00979)</td>
<td>–.00517 (.00844)</td>
</tr>
<tr>
<td>CO</td>
<td>–.0114 (.00979)</td>
<td>–.00517 (.00844)</td>
</tr>
<tr>
<td>O3</td>
<td>–.00117 (.0136)</td>
<td>.0148</td>
</tr>
<tr>
<td></td>
<td>–.0114 (.00979)</td>
<td>–.00517 (.00844)</td>
</tr>
</tbody>
</table>

**Note.**—The dependent variables are the natural logs of the specified cause-specific mortality rate (*n* = 8,876). All models control for state-by-year fixed effects and county-year level demographic characteristics. Observations are weighted by county population. Standard errors, clustered at the state level, are reported in parentheses. There is no statistically significant difference between the unemployment coefficient in columns *a* and *b*.

* *p < .10.
** *p < .05.
*** *p < .01.
detailed in appendix B. The results for total mortality are remarkably similar to those found at the county level. Pollution is procyclical with only CO (and not PM10 or O3) positively predicting death rates. The overall macroeconomic effect and the attenuation from controlling for pollution are also quite similar to those found above: a one percentage point rise in unemployment predicts 0.32% reduction in total mortality and this effect is attenuated by 13% with the addition of pollution covariates.

For age and cause of death, the findings are also generally qualitatively consistent with those from the county-level analysis, but the predicted unemployment rate effects are often larger at the state level, in accord with results obtained in Lindo (2015). Conversely, the significance of the correlation between CO and mortality decreased in most specifications at the state level, which we attribute to the coarser geographic detail of the CO measure. We also followed Lindo’s suggestion to estimate county-level models but controlling for state rather than county unemployment rates. In part, this may account for measurement error, if unemployment is better measured at the state level and pollution better measured at the county level. The results of this analysis, for total mortality, are presented in table 7 (standard errors are clustered at the state level). This table typically reveals larger unemployment rate coefficients than in the pure county-level analysis (table 3), while the pollution coefficients were little changed, and the attenuation in the unemployment effect when including controls for all three pollution measures is marginally larger (19% versus 17%).

4.6. Robustness Checks
Table 8 summarizes additional robustness tests. County-level data are used, and all models control for demographic variables as well as county- and state-by-year fixed effects. Each table entry shows the unemployment rate coefficient from a separate regression. The second row for each model also holds constant the three pollution measures. Thus these replicate the specifications in columns 1 and 5 of table 3, other than the changes that are detailed in the third row. Of primary interest is the extent to which the addition of pollution controls attenuates the unemployment coefficient.50 In table 8, the standard errors are clustered at the county, not the state, level. Although this specification is less conservative, we choose it in this table to make clearer how the various robustness checks affect the significance of the unemployment coefficients. With county-clustered errors, the basic specification (model 1 in table 8) yields coefficients on unemployment that are significant at the 10% level. Comparing the significance level across the models in table 8 shows how the specifications affect the main results, although the significance will generally be lower with state-clustered standard errors, as shown in appendix table A2.

50. All other analyses, at both the state and county level, have also been subjected to these robustness tests, with results available upon request.
Model 1 repeats the results of the basic specification (but with standard errors clustered at the county level), showing that the three pollution measures “explain” (in a statistical sense) around one-sixth of the total macroeconomic effect. Model 2 is the same, except that the data are not weighted by county population. This might be desirable if, for example, pollution is more accurately measured in a smaller and less populous county, because it is geographically small and more of the population is close to the pollution monitors. The overall macroeconomic effect is considerably weaker in this case—the unemployment coefficient is \(-0.00222\) rather than \(-0.00346\) and is not statistically significant—but the attenuation when controlling for pollution is still around 15%. In model 3, we do not weight the pollution monitor data by the percent of total potential monitor days actually observed, which might make sense if the monitor days are chosen randomly so that, for instance, a monitor with observations during 183 days should count just as much as one operating every day of the year. Such reweighting has virtually no impact on the results. In model 4, we drop the years 1982–84, where complete data were available for only a few counties, again without changing the predicted effects.

51. More generally, unweighted estimates are often preferred to those that are weighted (e.g., see Wooldridge 1999; Butler 2000; Solon, Haider, and Wooldridge 2015).

52. At the state level, the results are considerably stronger when not weighting the regressions by (state) population.
Table 8. Robustness Checks: Total Mortality Estimates

<table>
<thead>
<tr>
<th>Specification</th>
<th>Model</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(1)</td>
<td>(2)</td>
<td>(3)</td>
<td></td>
</tr>
<tr>
<td>Without pollution controls</td>
<td>-.00346**</td>
<td>-.00222</td>
<td>-.00344**</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(.00169)</td>
<td>(.00179)</td>
<td>(.00169)</td>
<td></td>
</tr>
<tr>
<td>With pollution controls</td>
<td>-.00286*</td>
<td>-.00195</td>
<td>-.00303*</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(.00164)</td>
<td>(.00179)</td>
<td>(.00166)</td>
<td></td>
</tr>
<tr>
<td>Description</td>
<td>Main model</td>
<td>No population weights</td>
<td>No pollution monitor weighting</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(4)</td>
<td>(5)</td>
<td>(6)</td>
<td></td>
</tr>
<tr>
<td>Without pollution controls</td>
<td>-.00394**</td>
<td>-.00346**</td>
<td>-.00305*</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(.00171)</td>
<td>(.00169)</td>
<td>(.00168)</td>
<td></td>
</tr>
<tr>
<td>With pollution controls</td>
<td>-.00330**</td>
<td>-.00294*</td>
<td>-.00259</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(.00167)</td>
<td>(.00162)</td>
<td>(.00164)</td>
<td></td>
</tr>
<tr>
<td>Description</td>
<td>1985–2009</td>
<td>Quadratic pollution controls</td>
<td>Age-adjusted mortality</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(7)</td>
<td>(8)</td>
<td>(9)</td>
<td></td>
</tr>
<tr>
<td>Without pollution controls</td>
<td>-.00295***</td>
<td>-3.58e-05***</td>
<td>-.00555**</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(.000893)</td>
<td>(1.32e-06)</td>
<td>(.00217)</td>
<td></td>
</tr>
<tr>
<td>With pollution controls</td>
<td>-.00252***</td>
<td>-3.17e-05***</td>
<td>-.00510**</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(.000829)</td>
<td>(8.58e-06)</td>
<td>(.00208)</td>
<td></td>
</tr>
<tr>
<td>Description</td>
<td>State-specific time trends included</td>
<td>Mortality in levels (not logs)</td>
<td>Only counties with monitors</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(4)</td>
<td>(5)</td>
<td>(6)</td>
<td></td>
</tr>
</tbody>
</table>

Note.—See the text for a description of each column’s specification. Table shows the unemployment rate coefficient. The dependent variables are natural logs of the total mortality rate, except for model 9, where the mortality rate is not logged. All models control for county- and state-by-year fixed effects and demographic characteristics. The second row of each model also includes controls for the three pollution measures, whereas the first row does not. Observations are weighted by the county population except in model 2. Standard errors, clustered at the county level, are reported in parentheses.

* \( p < .10 \)

** \( p < .05 \)

*** \( p < .01 \)
Model 5 captures potential nonlinearities in the pollution-mortality relationship by adding quadratic terms for the pollution measures. None of the quadratic term coefficients are significant (the point estimates suggest that mortality is concave in PM10 and ozone levels and convex in CO), and their inclusion has no effect on the unemployment rate-mortality relationship. Model 6 age adjusts the total mortality rate using CDC definitions of the age-standardized population (e.g., see Murphy, Kochanek, and Xu 2012). In contrast to Stevens et al. (2011), we obtain very similar results to those found when using unadjusted mortality rates. A likely reason for the difference is that we include more complete controls for age in all of our models.

Model 7 includes controls for state-specific linear and quadratic time trends. This specification is less general than our preferred model, which includes a full set of state-by-year fixed effects, but is presented because state trends have frequently been included in related previous research (e.g., Dehejia and Lleras-Muney 2004; Stevens et al. 2011; Hoyes, Miller, and Schaller 2012; Schaller 2013). The results are robust to this specification, although with slightly smaller coefficient estimates and standard errors. But as shown in appendix tables A1 and A2, the inclusion of state or county-level linear trends reduces the attenuation of the unemployment coefficient when controlling for pollution.

The dependent variable in model 8 is the level rather than log of total mortality. The unemployment rate coefficients are reduced, as expected since mortality rates are per 1,000 individuals, but the addition of pollution controls continues to attenuate the predicted macroeconomic effect, although by a modestly smaller amount than before. Finally, in column 9 we include only those counties that include a monitor, rather than those that have a monitor within 20 miles (4,082 counties rather than 8,876 counties). This smaller sample includes more populous counties on average, and the relationship between mortality and unemployment is 60% larger than in the main model. Including pollution reduces the coefficient magnitude by about 10%.

We also estimated a number of additional models. As an alternative to the standardized pollution variables, we kept pollution in native units (i.e., in μg/m³ for PM10 and ppm CO and O₃). This had no impact on the estimated effects of macroeconomic conditions, nor did it change the direction or relative significance of the pollution coefficients. Second, we estimated specifications using trend deviations in Gross State Product (GSP) as the macroeconomic proxy. The GSP coefficient was

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53. When age standardizing, mortality in each age category is weighted by a fixed age distribution for each location, rather than the actual age distribution.

54. Our regressions include seven age categories (plus a reference group) versus just two in Stevens et al. (2011).

55. Data were from the Bureau of Economic Analysis: http://www.bea.gov/iTable /iTable.cfm?ReqID=70&step=1. We detrended each state’s annual real GSP (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).
positive but not quite significant (the $p$-value was 0.19), weakly suggesting the procyclicality of mortality. However, the attenuation occurring when adding the pollution controls was only around 2%.\(^{56}\) Third, we replicated the regressions after first-differencing the pollution measures, unemployment, and mortality rates. The relationship between unemployment and pollution was consistent with the base case results though somewhat smaller in magnitude, but with no evidence of a relationship between pollution and mortality. We attribute this to a misspecification from first-differencing when the data are actually stationary.\(^{57}\) Fourth, we restricted the sample to those county years for which we have a measure for CO, but not necessarily for the other pollutants. We do this since CO was most consistently linked to deaths in our and previous studies. This increases the sample size from 8,876 to 12,498 county years. The results are similar to those using the base specification, though the magnitude of the relationship between unemployment and mortality is somewhat reduced. Finally, we estimated models for the subperiods 1982–94 and 1995–2009.\(^{58}\) Consistent with the results of two recent studies (Stevens et al. 2011; Ruhm 2015), we found that mortality was highly procyclical during the earlier period but not at all in the later one, with the addition of pollution controls modestly attenuating the predicted macroeconomic effects in both periods. However, as Ruhm (2015) has emphasized, estimates obtained using relatively short time periods are likely to be sensitive to the precise choice of starting and ending dates, and so we do not have much confidence in these results.

4.7. 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

---

56. Differences in results when using GSP versus those for unemployment rates are not surprising, since previous studies (e.g., Gerdtham and Johannesson 2005) provide evidence of stronger procyclical fluctuations in mortality when proxied by unemployment rates than when using other measures, such as deviations from GDP trends.

57. Unit root tests suggested that most series were stationary. Specifically, we conducted the Levin-Lin-Chu (2002) and the Harris-Tzavalis (1999) tests for stationarity for all three pollution variables, the unemployment rate, and total mortality. Since both tests require balanced panels, we used the subset of counties that are present in all years from 1986 to 2009 (185 counties) in this analysis. We reject a unit root for both PM10 and O\(_3\) using both tests but for CO only with the Levin-Lin-Chu test. We reject a unit root for unemployment and for total mortality only with the Harris-Tzavalis test (which is more appropriate in our data set with large $N$ but moderate $T$).

58. 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.
of the potential for uncontrolled confounding factors. We can, however, examine whether our predicted pollution effects are plausible when compared to results from previous research providing microlevel estimates of the relationship between ambient concentrations and mortality. This exercise is summarized in table 9, with a detailed description of the underlying methods and calculations provided in appendix C.

Although the basic procedure is straightforward, several issues need to be addressed. First, the 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 $\mu g/m^3$) on different 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 prior 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 also emphasize that the prior estimates are often imprecise, so that deviations between our results and those of earlier work do not necessarily indicate biases or errors in our predictions. For this analysis we use our estimates from the state-level analysis (see app. B), because these provide more consistent estimates of the relationship between mortality and unemployment.\footnote{59. In particular, the relationship between unemployment and infant mortality, one of the main outcomes examined in previous studies, is positive (but insignificant) in the county-level models.}

Table 9 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 mortality effects of pollution, combined with our results showing how pollution varies with macroeconomic conditions, pollution is estimated to account for between 5% and 40% of the procyclicality of mortality in most models, although with smaller or larger estimates in three cases (cols. 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 8%–12% of the cyclical fluctuations, in cases where we estimate that it has any effect.\footnote{60. We did not find a significantly positive effect of PM10 on deaths from strokes, heart attacks, or cardiovascular diseases, so table 9 reports a zero impact in these cases.}
<table>
<thead>
<tr>
<th></th>
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<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Pollutant</td>
<td>CO</td>
<td>TSP/PM10</td>
<td>CO</td>
<td>PM10</td>
<td>CO</td>
<td>PM10</td>
<td>PM10</td>
<td>PM10</td>
<td>PM2.5/PM10</td>
</tr>
<tr>
<td>Outcome</td>
<td>Infant mortality</td>
<td>Infant mortality</td>
<td>Infant mortality</td>
<td>Infant mortality</td>
<td>Heart attacks</td>
<td>Heart attacks</td>
<td>Respiratory deaths</td>
<td>Cardiovascular deaths</td>
<td>Stroke</td>
</tr>
<tr>
<td>Estimated effect of a one-unit pollution increase on mortality(^1) based on:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Previous research</td>
<td>18.1</td>
<td>1.00</td>
<td>13.1</td>
<td>10.1</td>
<td>0.0550</td>
<td>0.00600</td>
<td>0.00443</td>
<td>0.00294</td>
<td>0.00103</td>
</tr>
<tr>
<td>Current research</td>
<td>30.3</td>
<td>1.39</td>
<td>30.3</td>
<td>1.39</td>
<td>0.0642</td>
<td>0</td>
<td>0.00342</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Change in cyclical deaths explained by pollution based on:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Previous research</td>
<td>5.56%</td>
<td>8.46%</td>
<td>4.02%</td>
<td>85.5%</td>
<td>8.19%</td>
<td>2.47%</td>
<td>110%</td>
<td>36.3%</td>
<td>6.81%</td>
</tr>
<tr>
<td>Current research</td>
<td>9.29%</td>
<td>11.8%</td>
<td>9.29%</td>
<td>11.8%</td>
<td>9.58%</td>
<td>0%</td>
<td>8.47%</td>
<td>0%</td>
<td>0%</td>
</tr>
</tbody>
</table>

\(^1\) Units are deaths per 100,000 live births (change in death rate), in response to a one-unit change in pollution, in columns 1–4 (5–9). CO units are parts per million (ppm); PM10 units are μg/m\(^3\).
5. 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 than for persons of prime working age, suggesting that the direct effects of changes in labor market conditions are unlikely to fully explain these patterns. A plausible alternative is that variations in other risks explain some of the macroeconomic fluctuations. One such risk, traffic fatalities, has been widely studied and universally found to increase when the economy strengthens. However, a different potential health threat—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 investigating how three types of pollutants, carbon monoxide (CO), particulate matter (PM10), and ozone (O3), 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 concentrations. Consistent with previous research, we uncovered a negative correlation between county unemployment and mortality rates, after controlling for county demographic characteristics and state-by-year fixed effects. Adding the three air pollutants to the model attenuated the predicted unemployment rate effect by about 17%, consistent with a substantial role for air pollution. This attenuation is significant at the 10% level or better but is insubstantial in models that include linear time trends. CO concentrations were estimated to be more important than PM10 or O3 concentrations, but we do not know whether this represents differences in true health effects, the accuracy of pollution measurement, or correlations with other types of pollution or uncontrolled confounding factors.

The results for specific causes of death were also largely consistent with a 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 and their inclusion substantially and significantly attenuated the unem-

61. 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.

62. 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 (US EPA 2010). Consistent with this, some recent research (Currie and Neidell 2005; Beatty and Shimshack 2014; Arceo et al., forthcoming) 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., forthcoming). We also link PM10 to mortality in some of our estimates. Thus, disentangling between CO and PM10 mortality effects remains an area with unanswered questions.
ployment rate coefficients in these models. However, the macroeconomic estimates were often imprecise, and the attenuation resulting from including the pollution controls was modest for cardiovascular mortality, the leading cause of death. Pollution levels were also positively associated with nonvehicle 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 examining pollution effects on mortality. The results of this analysis corroborate the possibility that changes in pollution levels explain a portion of the observed procyclical variation in deaths.

This research should be considered preliminary because of some unexplained results and since many extensions would be desirable. At the most basic level, we cannot be sure that we are accurately estimating the true effects of pollution. Our pollution measures are crude, being limited to just three of many types of pollutants, and 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 county-level data is also potentially problematic since unemployment rates are measured quite noisily. We addressed this by conducting a state-level investigation, which provided results that were largely consistent with county-level findings.

Second, we only examine the contemporaneous relationship between health, pollution, and the business cycle; however, both pollution and recessions could have uncaptured longer-term effects.63 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 pollution 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.64

Our findings should certainly not be taken to imply that recessions are beneficial (although they may be 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

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63. See Coile, Levine, and McKnight (2014) for evidence that recessions have long-run effects on health.
64. On this front, Hicks et al. (2015) conduct a similar analysis using a meteorological phenomenon known as thermal inversions as an instrument for pollution.
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equalized) do not vary over the business cycle. Conversely, it may be useful to mod-erate 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 since the costs of polluting would rise in periods of economic strength, when the demand for permits is high.

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