Changes in U.S. Hospitalization and Mortality Rates Following Smoking Bans

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Abstract

U.S. state and local governments have increasingly adopted restrictions on smoking in public places. This paper analyzes nationally representative databases, including the Nationwide Inpatient Sample, to compare short-term changes in mortality and hospitalization rates in smoking-restricted regions with control regions. In contrast with smaller regional studies, we find that smoking bans are not associated with statistically significant short-term declines in mortality or hospital admissions for myocardial infarction or other diseases. An analysis simulating smaller studies using subsamples reveals that large short-term increases in myocardial infarction incidence following a smoking ban are as common as the large decreases reported in the published literature. © 2010 by the Association for Public Policy Analysis and Management.

INTRODUCTION

State and local governments have increasingly banned smoking in public places (including workplaces, restaurants, and bars) as a means of limiting nonsmoker exposure and of discouraging smoking. Several recent studies in the medical literature using a small number of regions suggest that smoking bans lead to a short-term 8 to 40 percent decrease in the annual incidence of acute myocardial infarction (AMI) (Bartecchi et al., 2006; Cesaroni et al., 2008; Sargent, Shepard, & Glantz, 2004). Despite these findings, it is unclear how well the results would translate to typical U.S. communities. We examine whether governmental smoking restrictions affect hospitalization and mortality rates in a large sample of U.S. communities.

We calculate death and hospitalization rates for AMI and other diseases using Medicare Provider Analysis and Review (MEDPAR) files, national death records (otherwise known as the multiple cause of death files, hereafter MCD), and hospitalization data from the Healthcare Cost and Utilization Project's Nationwide Inpatient Sample (NIS). We compare rates before and after passage of these bans relative to communities that did not pass bans. We use the variation in implementation dates across the country and fixed effects models to control for unobservable factors, which include the nationwide improvement in medical care and general health over time, as well as the decrease in the smoking rate and private factors over time. We use variation in implementation dates across the country and fixed effects models to identify the effect of government smoking bans. We find that smoking restrictions do not substantially affect short-term mortality and hospitalization rates in elderly, working-age, and child populations.

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Most previous published studies on the health effects of smoking bans share a common methodology: They compare the change in outcomes in a single community that has passed a smoking ban with the change in outcomes in a small set of nearby communities that have not passed bans (a few studies do not employ a set of control communities). A major contribution of this paper is that we simulate the results from all possible small-scale studies using subsamples from the national data. We find that large short-term increases in AMI incidence following a smoking ban are as common as the large decreases reported in the published literature.

BACKGROUND

Environmental Tobacco Smoke and Health Outcomes

In a recent review, the U.S. Surgeon General reports that numerous epidemiologic and laboratory studies have linked environmental tobacco smoke (ETS) exposure to increased rates of cardiovascular disease, respiratory illness, and lung cancer (Barnoya & Glantz, 2005; Glantz & Parmley, 1991; He et al., 1999). Laboratory studies support the notion that small quantities of inhaled cigarette smoke can induce biochemical responses in nonsmokers similar to those in chronic smokers. Such effects could predispose nonsmokers to greatly elevated risk of AMI and stroke. Epidemiologic studies typically compared outcomes of nonsmoking spouses of smokers and nonsmokers. Although some meta-analyses disputed these findings, most agreed that chronic ETS exposure increased risk of AMI by 20 to 30 percent. Although no amount of secondhand smoke is likely to be beneficial, the above studies do not address the potential effects of intermittent exposure to secondhand smoke, as might be caused by exposure to cigarette smoke in public places.

Secondhand smoke in public places has been most strongly linked to AMI among all potential adverse health outcomes (Bartecchi et al., 2006; Ong & Glantz, 2004; Sargent, Shepard, & Glantz, 2004; U.S. Department of Health and Human Services, 2006). There is some theoretical justification for this in the medical literature. The full effects of eliminating tobacco smoke may take years to develop because some aspects of coronary artery disease (such as narrowing of the coronary arteries) develop slowly over time. However, heart attacks often follow sudden clot formation in diseased arteries; in laboratory settings, exposure to even small quantities of tobacco smoke can induce biochemical states that predispose to heart attacks. Therefore, a smoking ban could plausibly reduce AMI incidence and mortality as early as the first month after a ban if it eliminates even relatively minor exposure in nonsmokers. In addition, smoking bans presumably raise social and other costs of smoking, which may cause current smokers to quit, thus improving health outcomes in smokers. As a result, many prior studies examined AMI rates in single regions in the 6 to 18 months following a smoking ban. In addition, those exposed to secondhand tobacco smoke may suffer higher rates of asthma and chronic obstructive pulmonary disease (COPD). Reducing ETS exposure could plausibly improve these outcomes as well.

Public Bans on Smoking in U.S. Public Places

As evidence on ETS accumulated, many U.S. employers began restricting smoking in the workplace, and the proportion of workers covered by a smoking ban increased from 25 percent to 70 percent between 1986 and 1993 (Centers for Disease Control and Prevention [CDC], 2000; Farkas et al., 1999; Farrelly, Evans, & Sfekas, 1999). Following these private restrictions and a few isolated smoking bans enacted in the 1980s, communities in California increasingly banned smoking in workplaces, restaurants, and bars in the early 1990s. Many other states and municipalities followed (American Nonsmokers' Rights Foundation, 2007). In addition to these local policies, several prominent politicians have advocated a national policy banning smoking in public places (OnTheIssues.org, 2007).

Although these bans have proven popular, the scientific literature to date has only examined the imposition of a smoking ban in a few specific U.S. regions and several European countries. AMI rates decreased approximately 40 percent in Helena, Montana, and 27 percent in Pueblo, Colorado (relative to surrounding communities), following the imposition of broad restrictions (Bartecchi et al., 2006; Sargent, Shepard, & Glantz, 2004). A larger study compared rates of AMI and acute stroke admissions in New York state before and after comprehensive smoking bans, which were largely implemented in March and July of 2003 (Juster et al., 2007). This study estimated that the laws reduced AMI admissions by 8 percent, although it did not compare these changes in AMI and acute stroke admissions to changes that may have occurred in nearby states that did not implement smoking bans over this period.

Examining a different mechanism, Adams and Cotti (2008) find increased rates of vehicular deaths following the enactment of smoking bans. They attribute this increase to smokers driving out of their native area to find a place to smoke in public. Another possibility is that these bans lead smokers to a smoke more in vehicles, which could be a distraction while driving. They use national data, and hence measure the average effect of a ban across all communities in the U.S.; unlike the previously cited studies, they do not measure the effect in individual communities. In another study using national data, Adda and Cornaglia (2010) found that the imposition of smoking bans did not reduce ETS exposure among nonsmokers.

International Smoking Bans

Two relatively large studies of the effect of smoking bans on AMI incidence in Rome and the Piedmont region of Italy concluded that smoking bans reduced AMI incidence by 7 to 11 percent in younger populations (Barone-Adesi et al., 2006; Cesaroni et al., 2008). The authors demonstrated that the prevalence of smoking, cigarette consumption per smoker, and nonsmoker exposure dropped in Italy after the national ban (Barone-Adesi et al., 2006; Cesaroni et al., 2008; Gallus et al., 2007).[1]

A study of the Scottish public smoking ban found large reductions in AMI rates as well (Pell et al., 2008). The Scottish government implemented a comprehensive ban in March 2006. The authors measured admissions to 9 hospitals (which treat over 3 million people) as well as death records for the general population. They measured actual exposure to cigarette smoke among nonsmokers (former smokers and never-smokers) and current smokers. In prior studies of the general population, serum cotinine levels declined—from 0.43 to 0.25 nanograms/dL in nonsmokers and from 167 to 103 ng/dL in current smokers. The authors then noted statistically significant declines in admissions for acute coronary syndromes in all groups—14 percent in smokers, 19 percent in former smokers, and 21 percent in those who never smoked. The decline in the AMI rate was noticeably larger than the 4 percent decline in neighboring England during the same time period (which lacked a comprehensive ban).

Implications of Prior Work for U.S. Smoking Policy

The aforementioned studies link smoking bans to impressive health improvements and suggest that widespread public smoking bans could demonstrably improve U.S.

[1] The authors used cotinine levels, obtained from blood or salivary samples, to measure cigarette smoke exposure in nonsmokers reliably.

public health. However, the extant literature may not be relevant for U.S. policymakers. First, the international experience may not translate to the U.S. setting. Nonsmokers' exposure to secondhand smoke and smoking prevalence in Italy and in Scotland were much higher and private smoking restrictions were weaker than in the U.S. For example, Juster et al. (2007) cited the 47 percent decline in salivary cotinine levels in New York (which was based on a response rate of 26 percent) to lend biological plausibility to the large decline in the AMI rate.[2] However, the cotinine level declined from 0.078 ng/mL to 0.041 ng/mL, which is an order of magnitude smaller decline than the drop from 0.68 to 0.56 ng/mL in Pell et al. (2008). The pre-ban New York state level is below the limit of detection for a study by Pell et al. (2009) that examined outcomes in nonsmokers following acute coronary syndrome, which further illustrates the baseline differences between U.S. and European studies.

Second, restricting the analysis to U.S. studies does not eliminate questions about generalizability. Prior U.S. studies were small in scale, having examined only a few regions; it is possible that those regions are not representative of typical U.S. communities. Although difference-in-difference analyses can control for unobserved factors, a simple pair-wise comparison using an atypical pair of communities will yield results that may not be representative. In contrast to the aforementioned studies, a simulation study found that extending smoking restrictions from 70 percent to 100 percent of U.S. workplaces would prevent roughly 1,500 myocardial infarctions in the first year (Ong & Glantz, 2004). Although this may be a clinically relevant improvement, it represents a much smaller reduction (<1 percent) than those reported in the small-scale studies based in Helena, Pueblo, or New York state.

Third, publication bias may have prevented null effect studies from being published, thus biasing overall impressions from the literature. Two recent reviews by Meyers, Neuberger, and He (2009) and the Institute of Medicine (2009) concluded that smoking bans substantially decreased AMI risk. Meyers, Neuberger, and He plotted the effect size against the standard error for 11 major studies from the literature. Meyers et al. noted that smoking ban studies with smaller standard errors (and generally larger sample sizes) tended toward a null effect and that smoking ban studies with larger standard errors (and generally smaller sample sizes) tended toward large beneficial effects. Although they acknowledged that the skewed distribution suggests the possibility of publication bias, Meyers et al. argued that the smaller studies were fundamentally different because they analyzed longer periods of time, which would have permitted the smoking bans under consideration to take effect. Although this could be true, publication bias could also have caused the skew toward very beneficial short-run studies.

Fourth, the mechanism for the tremendous declines in AMI rates reported in the small-scale studies is unclear, which makes their results less certain. It is unknown whether U.S. public bans effectively reduce exposure to secondhand smoke and whether the reduced exposure causes clinically significant cardiovascular risk reduction. There is conflicting evidence as to whether typical U.S. government smoking restrictions dramatically reduce exposure to ETS or induce smokers to quit, as restrictions initiated by employers have been shown to do (Adda & Cornaglia, 2010; Evans, Farrelly, & Montgomery, 1999; Longo et al., 1996; Metzger, Mostashari, & Kerker, 2005). If strong private restrictions were prevalent in the U.S. and avoidance of public bans was relatively easy, public bans might have no effect at all. In support of this possibility, Bitler, Carpenter, and Zavodny (2009) find that the imposition of governmental bans on smoking in workplaces or restaurants did not change whether smoking was, in fact, banned at those locations—either because smoking had previously been voluntarily banned or because the governmental ban was flouted.

[2] The original survey was reported in the Centers of Disease Control's *Morbidity and Mortality Weekly Report* (CDC, 2007).

It is therefore unclear whether governmental restrictions substantially reduce ETS exposure or simply codify existing workplace practice.

Furthermore, the estimates of risk due to ETS exposure due to public smoking from these small-scale studies are similar in magnitude to those from studies of intensive household exposure to secondhand smoke (Thun, Henley, & Apicella, 1999). The equivalent effect sizes for public and private ETS exposure implies that secondhand smoke presents large heath risks at low levels and minimal additional health risks at higher levels, which seems unlikely.[3] In addition, U.S. national AMI hospitalization rates have remained stable over time, despite the substantial rise in the incidence of smoking bans (Lucas et al., 2006).

We address these issues by analyzing the impact of U.S. public smoking restrictions on health outcomes in a large, heterogeneous group of U.S. communities. By analyzing much larger populations (all elderly persons in the U.S. and a diverse group of U.S. hospitals and counties) and by accounting for underlying secular trends and region-specific characteristics, we mitigate the possibility of selection bias.

DATA

Data on Smoking Bans

We use ordinance data from the American Nonsmokers' Rights Foundation to identify states and municipalities that implemented restrictions on smoking between 1990 and 2004. We adapt the classification scheme from the American Nonsmokers' Rights Foundation (ANRF) to identify those bans that restrict smoking in all workplaces except bars and restaurants as "workplace" bans. In the ANRF data, we identified all "100 percent smoke-free" and "qualified" bans. Furthermore, we also code several regions not included in the ANRF list as having "qualified" bans. For example, California adopted a nearly complete ban on workplace smoking in 1995 (with modest exemptions). We classified California as smoking-restricted starting in 1995. We also created a data set of bans of any site—workplaces, bars, or restaurants. We classify each three-digit zip code, city, and county in the U.S. by smoking ban status and date of implementation.

Data Sources for Health Outcomes

We analyze health outcomes using the Multiple Cause of Death (MCD) database (1989 to 2004), Medicare claims (1997 to 2004), and the Nationwide Inpatient Survey (NIS), collected 1993 to 2004 by the Healthcare Cost and Utilization Project, which is sponsored by the Agency for Healthcare Research and Quality (Agency for Healthcare Research and Quality, 2006). The MCD database identifies the underlying cause for each death in the U.S. Our source for Medicare claims is the Medicare Provider Analysis and Review (MEDPAR) files, which include all fee-for-service Medicare beneficiaries in the U.S. The NIS is a nationally representative 20 percent sample of all discharges from U.S. community hospitals (which includes all nonfederal acute care hospitals). Excluded hospitals include Veterans Affairs hospitals and long-term rehabilitation hospitals.

We identified mortality and hospitalizations due to AMI and all-cause deaths and hospitalizations because both might plausibly improve in the short run. Broad disease measures like AMI and all-cause events are less likely to be miscoded in administrative data, reducing measurement error (Petersen et al., 1999; Tirschwell & Longstreth, 2002). Mortality has not been used frequently as an outcome variable in studies specifically addressing smoking bans. However, at least one recent study

[3] Similar risk reductions were noted in European and U.S. studies, though baseline smoking prevalence, secondhand smoke exposure, and efficacy varied, which argues against a plateau in risk.

of ETS (Pell et al., 2009) and numerous clinical studies (Berger et al., 1999; Gan et al., 2000; Rogers et al., 2008) use early mortality following AMI as an outcome variable.[4] Accordingly, we use this outcome to measure an important component of cardiovascular diseases. In addition, using mortality allows us to take advantage of comprehensive national death records, which are far more demographically and geographically diverse than any data on hospitalizations. We also consider cases of asthma and COPD, which are chronic diseases, though secondhand smoke possibly triggers acute exacerbations. We also use hip fracture hospitalizations as a negative control because their incidence would be unlikely to decrease quickly after a smoking ban (Hoidrup et al., 2000).

We assemble each data set in a similar fashion: We first classify deaths and hospital admissions according to their primary diagnoses (such as AMI and asthma) as identified using codes from the International Classification of Diseases, 9th and 10th editions (ICD-9 and ICD-10).[5] We then sum all outcomes in each region (hospital catchment area, county, or zip code). Finally, we add information on smoking ordinances for that region.

In addition, workplace smoking bans could have differential effects by age. The elderly and children may be more vulnerable to the diseases exacerbated by ETS and could stand to gain more benefit than a typical working adult. On the other hand, children and the elderly are primarily exposed to workplace ETS as customers, which would reduce their benefit from a smoking ban. To account for these differences, we further stratify outcomes into three age groups per region: children (0 to 17 years), working age adults (18 to 64 years), and the elderly (65+ years).

From each data set, we exclude deaths and hospitalizations where we are unable to determine whether the person lived in an area where a smoking restriction was implemented (35 to 40 percent of the NIS and MCD data). In addition, we exclude the following from our analysis of NIS hospitals: transfer patients, hospitals included in a single survey year, hospitals that merged during 1993 to 2004, hospitals devoted to acute rehabilitation (because few AMIs are admitted), and small hospitals (<1,000 admissions/year). The final samples consist of approximately 60 percent of all deaths and 4 percent of all hospital admissions in the U.S. for the period under study. Table 1 summarizes the characteristics of the samples used.

EMPIRICAL STRATEGY

We estimate region-level fixed effects models using our three national samples. In addition, we perform a number of sensitivity analyses on these models. Finally, we develop an approach to simulate the potential distribution of effects one might observe from pair-wise difference-in-difference models, using the national data.

[4] Notably, IOM (2009) considered the impact of smoking bans on both mortality and morbidity due to AMI.

[5] Pell et al. (2008) noted that the Scottish administrative data had an error rate of nearly 50 percent for AMI (using the ICD-10 code for AMI, I21) and suggested that retrospective analyses using routine administrative data might be biased. However, cross-national differences make generalizations about discharge abstract coding difficult. ICD-9 coding has been the subject of several U.S. studies, which have revealed better accuracy rates for myocardial infarction (AMI) coding. These include Kiyota et al. (2004) and Peterson et al. (1999), which showed 94.1 percent and 96.9 percent accuracy rates for medical records flagged in administrative data. Pladevall et al. (1996) showed that 88 percent of clinical diagnoses of AMI could be detected using the ICD-9 code of 410. Hammar et al. (2001) and Austin et al. (2002) show similar findings using a Swedish registry and Canadian data, respectively. We speculate that U.S. discharge abstracts might record AMI diagnoses more accurately because of the strong tie between accurate coding and reimbursement. As a result, we believe it is unlikely that differences in coding between the two studies could explain the results.

Table 1. Data source characteristics.

Nationwide Inpatient Sample:	
Hospitals	858
States	26
Years	1993–2004
All admissions	36,798,572
Admissions by disease	
Acute myocardial infarction	673,631
Combined asthma and COPD[a]	845,587
Multiple cause of death:	
Counties	467
States	50
Years	1989–2004
All deaths	24,341,772
Acute myocardial infarction	2,018,548
Medicare patients:	
Regions (three-digit zip codes)	868
States	51
Years	1997–2004
Included Medicare population (person-years)	275,303,008
All deaths	13,106,175
All admissions	72,542,544
Admissions by disease	
Acute myocardial infarction	2,382,387
Combined asthma and COPD[a]	2,984,382
Hip fracture	3,381,690

Source: Nationwide Inpatient Sample, multiple cause of death files, and 100 percent Medicare provider analysis and review files.

[a] Chronic obstructive pulmonary disease.

Fixed Effects Regression Model

We use a multivariate linear regression model to analyze changes in mortality and hospitalization following smoking bans. Analyses comparing outcomes before and after bans are implemented may be subject to bias due to unobserved trends. For example, many regions experienced unobserved increases in private smoking restrictions, reductions in smoking prevalence, or improved medical treatment that could have caused changes in outcomes. To mitigate these potential confounding factors, we compare trends in regions where smoking bans were implemented to those in control regions where smoking restrictions were not imposed. In particular, we estimate region fixed effects models. For each outcome (for example, AMI deaths or hospitalizations), we use the following regression model:

$$Outcome_{it} = \alpha_i + \gamma_t + \beta_s\, Smoking\ Ban_{it} + \delta tax_{it} + \varepsilon_{it} \qquad (1)$$

Here $Outcome_{it}$ represents the number of deaths or hospital admissions in region $t = 1 \ldots N$ and year $t = 1 \ldots T$, γ_t is an indicator for each year, tax_{it} is the log of state cigarette taxes[6] in year t, and ε_{it} is the error term. We include α_i, an indicator for each region (county, three-digit zip code, or hospital), to control for idiosyncratic

[6] Data on state cigarette taxes were obtained from the CDC (2010).

differences between regions. Our coefficient of interest is β_s, which represents the break in the time trend induced by a smoking ban, after controlling for secular trends. In presenting final results, we present the mean percentage change in outcomes $\left(\frac{\beta_s}{\mu} \times 100\%\right)$, where μ is $\frac{1}{NT}\sum_{t=1}^{T}\sum_{t=1}^{N} Outcome_{it}$. We use block bootstrap clustered at the regional level to calculate all standard errors. Of note, the effects of private restrictions enacted prior to a government ban are included in pre-ban trends and are excluded from the final estimate. As a result, this strategy plausibly identifies the short-term effects of government restrictions alone. Due to data limitations, we alter our approach in analyzing hospital admissions in NIS hospitals; we compare changes in admissions in the first year following a smoking restriction to changes in control hospitals without smoking restrictions.[7]

Sensitivity Analyses

In our main regression model (Equation 1) we do not include covariates other than time and region-specific indicators. However, it is possible that factors might change differentially over time between regions. If so, the fixed effects model would yield biased estimates. We therefore test model specifications of the following form:

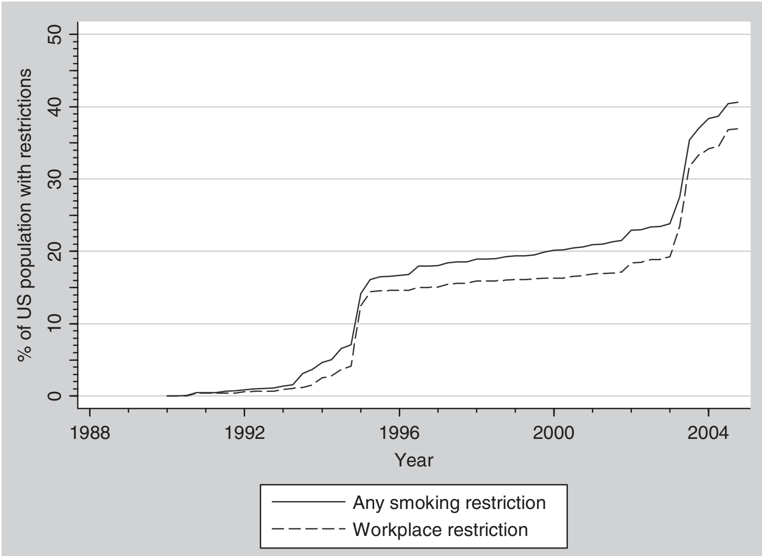
$$Outcome_{it} = \alpha_i + \gamma_t + \beta_s \; Smoking \; Ban_{it} + \beta X_t + \varepsilon_{it} \qquad (2)$$

The terms and results are the same as in Equation (1) except for the addition of βX_i, a vector of county-level characteristics taken from the 2005 Area Resource File. The variables include population size, number of physicians and hospital beds per county, household income, and percent of population in labor force. These data were linked by county and year, where available, to counties from the MCD and hospitals in the NIS data. We do not include these variables (which could potentially add explanatory power) in all model specifications because data for βX_i is often missing for several years, which substantially limits their sample sizes. We perform sensitivity analyses by comparing the results using model specification (2) to the results using model specification (1) for the same set of included regions.

Simulating Small-Sample Results

We complete our analysis by using subsamples of the national data to simulate a complete set of pair-wise comparison studies of the sort available in the published literature, including Bartecchi et al. (2006) and Sargent, Shepard, and Glantz (2004). These studies compared a treatment unit where a ban was passed against a control unit with no ban on the basis of the change in an outcome variable (heart attack admission rates, for instance) in a short period (6 to 18 months) after the ban was passed in the treatment region. We simulate the range of such effects by first calculating the percent change in admissions in each hospital located in a region with workplace smoking restrictions between the year before and the year following a workplace smoking ban; we also calculate the same statistics for all control hospitals from the same time period (Rosenbaum, 2001). We then subtract the change in outcomes in each contemporaneous control hospital from each intervention hospital.

[7] In our analysis of the NIS data, our primary unit of analysis is the hospital's catchment area, which includes the hospital's home city but whose full extent is unobservable. We use the number of admissions to a hospital in a particular month as a measure of the admission rate within its catchment area. Catchment areas tend to remain stable over time, so the approximation error is likely to be small. It is possible that we misclassified some admissions. However, many bans were enforced in the county or state of origin, which would include the entire catchment area. In addition, we only consider patients with serious illnesses, who tend to be taken to the nearest hospital, which further reduces bias.



Source: American Nonsmokers Rights Foundation and California Department of Industrial Relations.

Figure 1. Prevalence of Smoking Bans over Time.

The resulting data set consists of the universe of possible (30,143) pair-wise comparisons of one smoking-restricted hospital with one control hospital. We conduct a similar analysis of heart attack mortality, except in this case, we use MCD mortality data and our unit of analysis is the county. The final data set consists of 15,824 pair-wise comparisons and represents the universe of such comparisons in the MCD data between counties with 100 percent of the population covered by a smoking ban and all control counties (without smoking bans). We also perform several sensitivity analyses using MCD data that compare control counties to counties in which the percentage of the population covered increased by at least 50 percent. We allow such bans when there are too few bans covering entire counties to allow robust analysis. We then adjust these comparisons to account for the fact that the observed effects can be attributed to effectively smaller bans.

RESULTS

Smoking Restrictions over Time

The percentage of U.S. regions that imposed smoking restrictions increased dramatically between 1988 and 2004; the prevalence of smoking restrictions rose sharply in 1995 and 2003 to 2004 and more gradually in other years (see Figure 1). Notably, this national trend follows the actions of private employers. By 1993, most workplaces isolated cigarette smoke, which would diminish the impact of legislative bans (CDC, 2000; Farrelly, Evans, & Sfekas, 1999).

Main Estimates

Workplace smoking restrictions are not associated with reductions in all-cause mortality or mortality due to AMI in any age group (see Table 2). We find no statistically significant reduction in admissions due to AMI among working-age adults (1.8 percent, 95 percent CI: −4.5 to 8 percent, $p = 0.58$) or among the elderly

Table 2. Mortality and smoking restrictions.

Disease	% Change in Mortality (95% CI)	*p*-Value
Workplace smoking restrictions:		
All deaths (0–17 years old)	–0.7 (–5.4 to 4.1)	0.779
AMI[a] (18–64 years old)	–3.6 (–9.6 to 2.5)	0.249
All deaths (18–64 years old)	–1.1 (–2.6 to 0.4)	0.161
AMI (all ages)	1.9 (–0.9 to 4.7)	0.186
All deaths (all ages)	0.3 (–0.7 to 1.3)	0.514
All deaths (age 65+)	2.1 (–0.2 to 4.3)	0.142
Any smoking restrictions:		
All deaths (0–17 years old)	–0.7 (–4.9 to 3.6)	0.755
AMI (18–64 years old)	–4.1 (–9.4 to 1.3)	0.135
All deaths (18–64 years old)	–0.7 (–2 to 0.6)	0.297
AMI (all ages)	1.3 (–1.1 to 3.6)	0.294
All deaths (all ages)	1.0 (0.2 to 1.8)	0.019
All deaths (age 65+)	1.5 (–0.4 to 3.5)	0.220

Source: Multiple cause of death files, years 1993 to 2004 except in age 65+, which are from 100 percent Medicare provider analysis and review files, 1997 to 2004.

[a] AMI indicates deaths from acute myocardial infarction.

(5.9 percent, 95 percent CI: 0.3 to 11.1 percent, $p = 0.09$) following the enactment of a workplace smoking restriction (see Table 3). We similarly find no evidence of a net reduction in admissions across age groups for other diseases. Furthermore, workplace restrictions are not associated with decreased all-cause admissions in children (1.0, 95 percent CI: −3.1 to 5.0 percent, $p = 0.635$). In the elderly, we find no statistically significant reduction of smoking bans on hip fracture admissions, our negative control. The increase in hip fracture admissions seen in the working-age population is not statistically significant after adjusting for multiple comparisons using Hochberg's method (Hochberg, 1988); furthermore, the all-ages hip fracture rate does not significantly change (0.5 percent, 95 percent CI: −3.0 to 3.9 percent). This supports the hypothesis that unobserved characteristics of regions do not confound our results.[8]

We do find some suggestive evidence (similar to that reported in Adda & Cornaglia, 2010) that smoking bans may lead smokers to smoke more at home. While asthma admissions show a statistically significant decline of 7.2 percent among individuals aged 18 to 64 following the imposition of smoking individuals, they also show a statistically significant increase of 14.6 percent among children. We do not want to overinterpret these findings, however, as the statistical significance disappears if we adjust for multiple comparisons using Hochberg's method. In addition, workplace bans appear to have a null effect on asthma admissions in all age groups (−0.1 percent, $p = 0.986$).

[8] We calculate the probability, given our sample, of finding a 17 percent reduction in heart attack admission (which is the reduction reported by the IOM, 2009, and by Meyers, Neuberger, & He, 2009). The standard error associated with our estimate of the effect of a workplace ban on AMI admission is 2.561. Thus, the probability of accepting the null of no effect given a true effect of –17 percent is <0.001. In other words, we are well powered to detect effects in the range reported in the literature. Because a 17 percent reduction in heart attack admissions would roughly translate into a 17 percent reduction in heart attack mortality, we can similarly use the standard error associated with our estimate of the effect of a workplace ban on AMI mortality, which is 1.436. Thus, the probability of accepting the null of no effect given a true effect of −17 percent is once again <0.001.

Table 3. Hospital admissions and smoking restrictions.

Disease	% Change in Admissions (95% CI)	*p*-Value
Workplace smoking restrictions:		
All admissions (age 0–17)	1.0 (–3.1 to 5)	0.635
Asthma (age 0–17)	14.6 (3.7 to 25.5)	0.008
AMI[a] (age 18–64)	1.8 (–4.5 to 8)	0.581
All admissions (age 18–64)	–0.4 (–2.2 to 1.5)	0.703
Asthma (age 18–64)	–7.2 (–13.8 to 0.5)	0.036
COPD[b] (age 18–64)	–6.5 (–15.6 to 2.5)	0.158
Hip fracture (age 18–64)	9.6 (0.1 to 18.2)	0.029
AMI (age 65+)	5.1 (–1.6 to 11.8)	0.209
All admissions (age 65+)	3.4 (–0.1 to 7.0)	0.110
Asthma (age 65+)	5.4 (–0.2 to 11.4)	0.132
COPD (age 65+)	1.4 (–2.7 to 5.8)	0.581
Hip fracture (age 65+)	–0.1 (–5.9 to 5.7)	0.982
AMI (all ages)	–2.0 (–7 to 3)	0.434
All admissions (all ages)	–0.6 (–2.2 to 0.1)	0.438
Asthma (all ages)	–0.1 (–7.5 to 7.3)	0.986
COPD (all ages)	–5.0 (–11.1 to 1.2)	0.111
Hip fracture (all ages)	0.5 (–3 to 3.9)	0.8
Any smoking restrictions:		
All admissions (age 0–17)	0.2 (–3.6 to 4.1)	0.899
Asthma (age 0–17)	9.0 (–1 to 19.1)	0.078
AMI (age 18–64)	–0.3 (–5.5 to 5)	0.925
All admissions (age 18–64)	–0.3 (–1.9 to 1.4)	0.741
Asthma (age 18–64)	–7.6 (–13.4 to –1.8)	0.011
COPD (age 18–64)	–4.9 (–13 to 3.2)	0.232
Hip fracture (age 18–64)	11.1 (2.6 to 19.6)	0.01
AMI (age 65+)	5.9 (0.3 to 11.1)	0.087
All admissions (age 65+)	4.9 (2.0 to 7.5)	0.004
Asthma (age 65+)	5.1 (–0.6 to 11.1)	0.140
COPD (age 65+)	4.9 (0.8 to 8.5)	0.032
Hip fracture (age 65+)	3.4 (–1.1 to 8.0)	0.222
AMI (all ages)	–1.8 (–6.7 to 3.1)	0.467
All admissions (all ages)	–0.5 (–1.9 to 0.9)	0.485
Asthma (all ages)	–1.3 (–6.5 to 4)	0.636
COPD (all ages)	–3.5 (–9.2 to 2.1)	0.218
Hip fracture (all ages)	0.2 (–3.6 to 4.1)	0.899

Source: Nationwide Inpatient Sample (1993 to 2004) except age 65+, which are from 100 percent Medicare provider analysis and review files, years 1997 to 2004.

[a] Acute myocardial infarction/ischemic heart disease.

[b] Chronic obstructive pulmonary disease.

Sensitivity Analyses

Solely using region-specific indicators to capture trends potentially omits important confounding variables, but comprehensive data is not available for all regions and years. We test for bias by adding variables for demographics and medical resource availability to the regression model. (We obtain these data from the 2005 Area Resource File, which contains information for all counties and some years.) We examine the importance of these omitted variables by comparing results with and without additional variables for the same time periods.

Table 4 shows the effect of any smoking ban on total death rates in all age groups using different sets of control variables. The estimates do not change dramatically

Table 4. Comparison of regression models: All-cause mortality following smoking ban of any sort (MCD[a] counties).

Variables Included	(1)	(2)	(3)	(4)
Any bans	–0.69769	–0.68	–0.09569	0.06183
	(0.66938)	(0.6708)	(1.10035)	(0.69781)
Hospital beds/person			0.01063	
			(0.0024)	
County population	0.000061	7.39E-05	0.000066	0.000163
	(0.0000192)	(0.0000218)	(0.0000213)	(0.0000143)
Physicians/person		–0.00108	–0.00081	0.000367
		(0.00283)	(0.00276)	(0.00197)
Percent population in labor force				15.42519
				(14.85652)
No. years in sample	15	13	9	5
No. of observations	24,884	21,580	15,512	7,308
Log (cigarette taxes)	–0.83342	–0.89405	–0.9993	–0.24762
	(0.36595)	(0.40096)	(0.52737)	(0.51228)
Results from original model	–0.63201	–0.60871	0.07003	0.11158
	(0.66551)	(0.66518)	(1.00374)	(0.68609)

[a] MCD = Multiple cause of death files. Each coefficient represents the percent change in outcomes due to a 1-unit change in the explanatory variable. Standard errors are clustered at the area level and are reported in parentheses.

with inclusion of different variables, but do change as the study sample changes. The sample is reduced from 24,884 county-quarters in column 1 to 7,308 county-quarters in column 4. Regression estimates using the same samples but different regression models are similar. For example, based on a limited sample of 15,512 county-quarters across 9 years in column 3, the estimated association between any smoking ban and total death rates was −0.10 percent (with a standard error of 1.10 percent) with controls for hospital beds per capita and physicians per capita and 0.07 percent (with a standard error of 1.0 percent) without additional controls. We show similar results in Tables 5 and 6. These results suggest that ordinary demographic changes are accounted for using the difference-in-difference identification strategy.

In all of the results reported in the tables, we calculate standard errors that allow for clustering at the area level (county, hospital area, or zip code). If, in addition, we were to correct for multiple comparisons using Hochberg's method, our standard errors increase further. For example, although we find that smoking bans were associated with an increase in asthma admissions in children, after correction for multiple comparisons, the result becomes statistically insignificant at the 5 percent level.

Pair-Wise Comparisons Simulating Small Sample Results

Figures 2 and 3 plot all possible pair-wise comparisons of changes in AMI incidence after a workplace smoking ban to changes in randomly selected control regions. Figure 2 shows that the mean measured effect of workplace smoking bans on heart attack admissions is close to zero, but 10 percent or greater declines and 10 percent or greater increases in AMI admissions are common. Figure 3 shows similar results for a comparison of AMI mortality in smoking-restricted counties from the year after a workplace ban with rates in counties without a ban. The results of this simulation analysis shows that results from prior small sample studies, which found

Table 5. Comparison of regression models: Myocardial infarction admission rates in working-age adults (NIS[a] hospitals) following workplace bans.

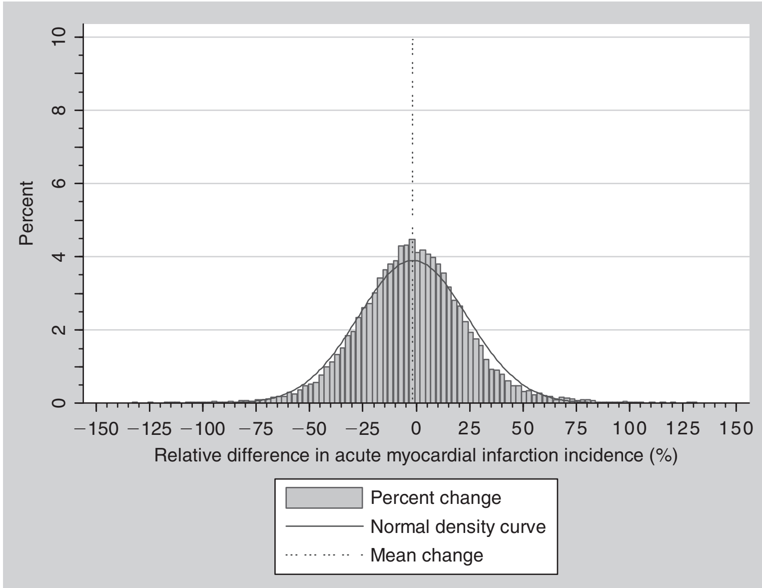
Variables Included	(1)	(2)	(3)	(4)
Workplace bans	1.75424	2.03768	9.39718	–6.58813
	(3.18218)	(3.1853)	(10.42659)	(6.12242)
Hospital beds/person			0.00527	
			(0.0027)	
County population	0.000031	0.000027	0.000020	0.000213
	(0.0000321)	(0.0000322)	(0.0000248)	(0.0000934)
Physicians/person		0.00531	0.00473	0.00468
		(0.00288)	(0.00344)	(0.01993)
Percent population in labor force				97.25818
				(206.0239)
No. years in sample	12	12	9	5
No. of observations	6,899	6,899	4,352	1,340
Log (cigarette taxes)	0.65992	0.55897	0.0992	3.45897
	(3.32944)	(3.33546)	(4.26416)	(5.91273)
Results from original model on this sample	1.71809	1.71809	8.993481	–6.53189
	(3.19176)	(3.19176)	(10.31603)	(5.91816)

[a] NIS = Nationwide Inpatient Sample. Each coefficient represents the percent change in outcomes due to a 1-unit change in the explanatory variable. Standard errors are clustered at the area level and are reported in parentheses.

Table 6. Comparison of regression models: All-cause admission rates in working-age adults (NIS[a] hospitals).

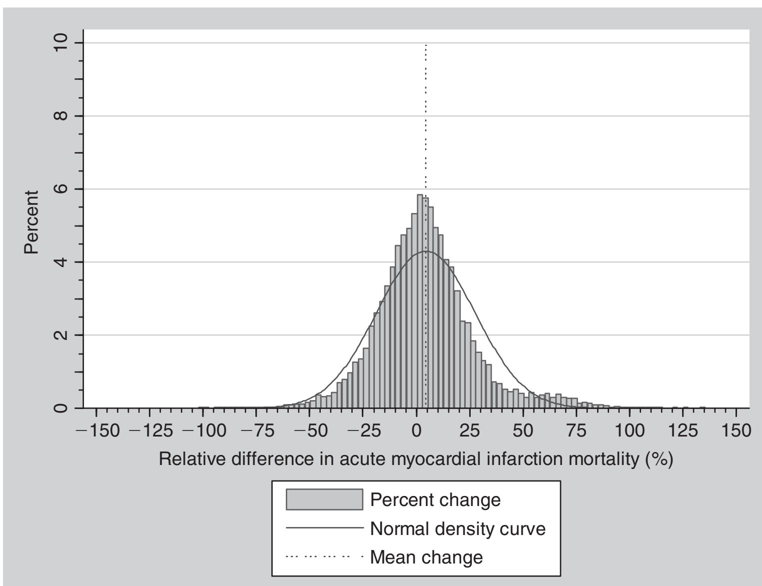
Variables Included	(1)	(2)	(3)	(4)
Workplace bans	–0.35931	–0.41509	–2.60612	1.78185
	(0.94125)	(0.93646)	(1.25905)	(2.47137)
Hospital beds/person			0.000517	
			(0.00105)	
County population	0.000043	0.000044	0.000036	0.000131
	(0.000016)	(0.000016)	(0.000021)	(0.000037)
Physicians/person		–0.00104	–0.00086	–0.00417
		(0.00143)	(0.00155)	(0.00454)
Percent population in labor force				1.82087
				(69.67497)
No. years in sample	12	12	9	5
No. of observations	6,899	6,899	4,352	1,340
Log (cigarette taxes)	–0.43008	–0.41022	0.4733	2.4302
	(1.40284)	(1.4078)	(2.0412)	(2.11001)
Results from original model on this sample	–0.33575	–0.33575	–2.63309	1.33431
	(0.94483)	(0.94483)	(1.27942)	(2.43288)

[a] NIS = Nationwide Inpatient Sample. Each coefficient represents the percent change in outcomes due to a 1-unit change in the explanatory variable. Standard errors are clustered at the area level and are reported in parentheses.



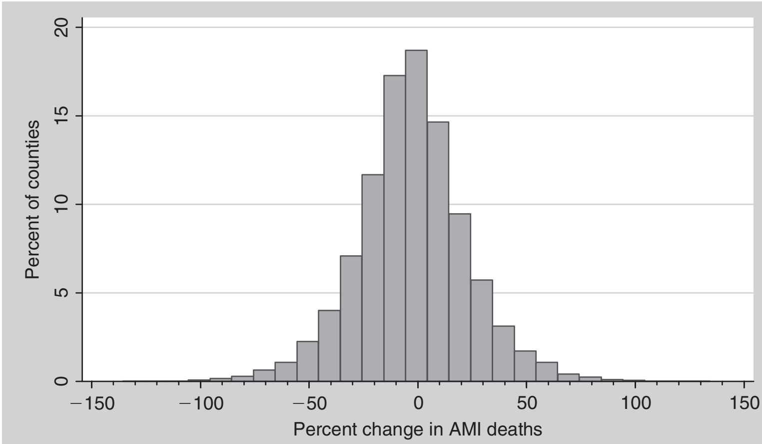
Source: HCUP Nationwide Inpatient Sample. Hospitals with smoking restrictions: 119. Control hospitals: 727. Pair-wise comparisons: 30,143. Relative difference may be less than –100 percent if a large percentage increase in a control hospital is subtracted from a large decrease in a smoking-restricted hospital.

Figure 2. Changes in AMI Admissions Relative to All Control Hospitals.



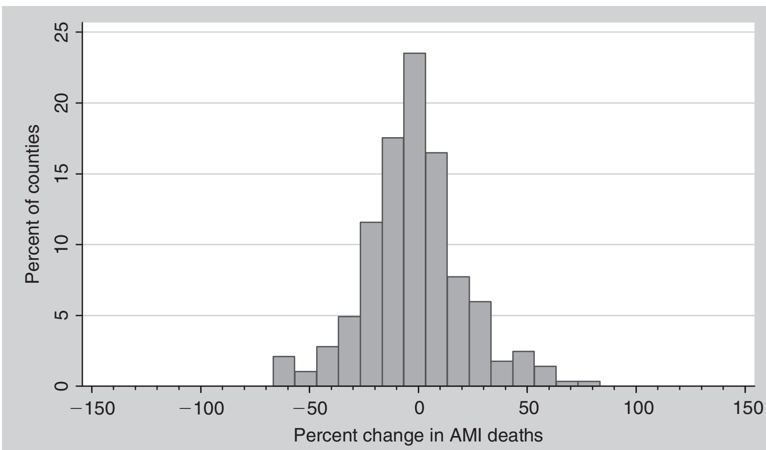
Source: MCD. Counties with smoking restrictions: 70. Control counties: 413. Pair-wise comparisons: 15,824. Relative difference may be less than −100 percent if a large percentage increase in a control county is subtracted from a large decrease in a smoking-restricted county. Counties with smoking restrictions include only those counties that cover 100 percent of the county with workplace restrictions. Control counties have no workplace restrictions.

Figure 3. Changes in AMI Deaths Relative to All Control Counties (Excludes Smoking Ban Counties with <100% Population Covered).



Source: National death records, all ages, 1989 to 2004. Control counties: 460.

Figure 4a. Year-to-Year Change in AMI Deaths in Counties without Smoking Bans.

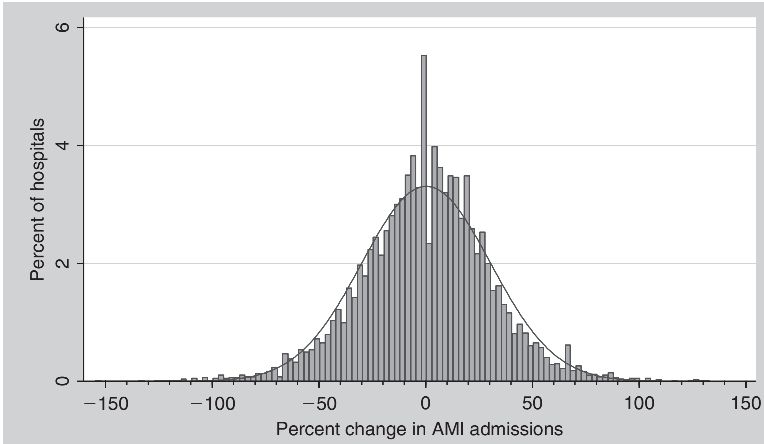


Source: National death records, all ages, 1989–2004. Counties with smoking restrictions: 76.

Figure 4b. Year-to-Year Change in AMI Deaths in Counties with Smoking Bans in First Year after Enactment of Workplace Bans Covering Entire County.

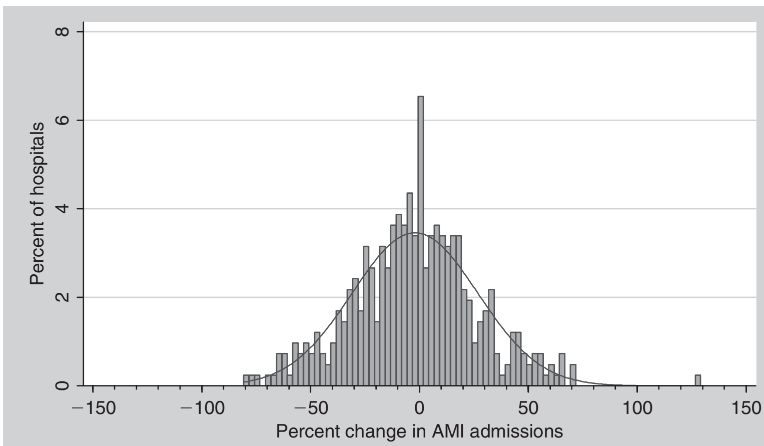
very large decreases in AMI admissions and mortality following the enactment of smoking bans, are feasible. However, results with the opposite sign and of similar magnitude are also feasible and should be equally common.

We conduct a series of analyses to test the robustness of our results. Difference-in-differences estimates may not be appropriate if the control and intervention regions are excessively dissimilar. To mitigate this potential problem, we show the undifferenced comparisons for deaths and admission in Figures 4 and 5. In regions without workplace smoking bans, AMI death and hospitalization rates change substantially from year to year, but the difference may be positive or negative due to random factors. Outcomes in the first year of the smoking ban mirror this wide distribution. Figures 4 and 5 also demonstrate that there is substantial variation in our primary outcome variables; this variation in outcome variables, along with significant



Source: HCUP, all ages, 1993 to 2004. Control hospitals: 755.

Figure 5a. Year-to-Year Change in AMI Admissions in Hospitals Located in Areas Without Smoking Bans.

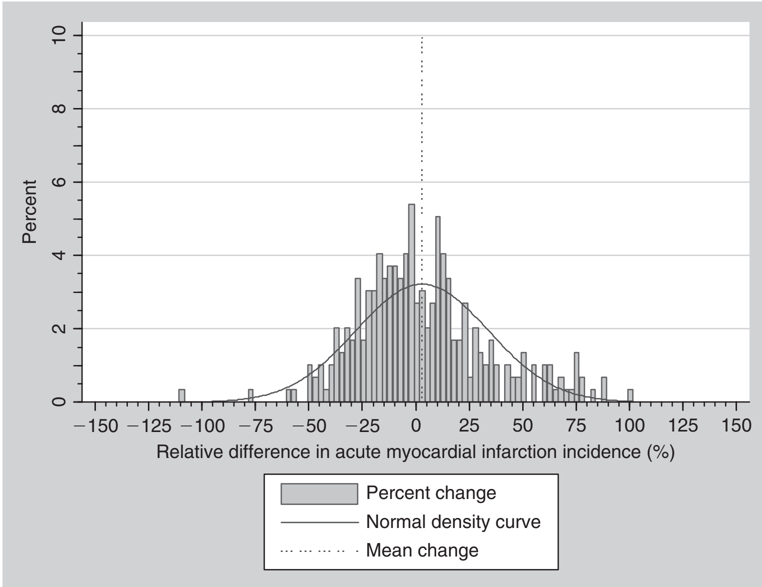


Source: HCUP, all ages, 1993 to 2004. Hospitals with smoking restrictions: 119.

Figure 5b. Year-to-Year Change in AMI Admissions in Hospitals in the First Year after a Smoking Ban.

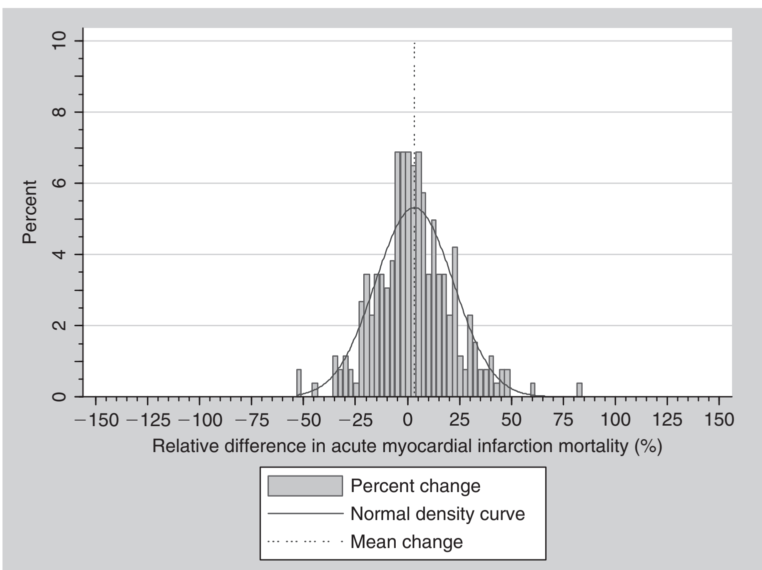
variation in the time and location of smoking ordinances seen in Figure 1, indicates that our empirical strategy has sufficient power for detecting the effect of smoking bans after accounting for the national trends in both AMI incidence and ETS exposure.

We also perform several sensitivity analyses in which we restrict the sample to regions for which we would be more likely to find beneficial effects. As in our primary analyses, the controls are restricted to areas without smoking bans, while the intervention regions are censored one year after the enactment of a ban (which also adjusts for seasonal differences). First, we restrict the sample to the universe of within-state pair-wise comparisons, which potentially offer better difference-in-differences comparisons (Figure 6). However, when analyzing both AMI deaths and admissions, we note the mean effects are centered near zero and widely distributed.



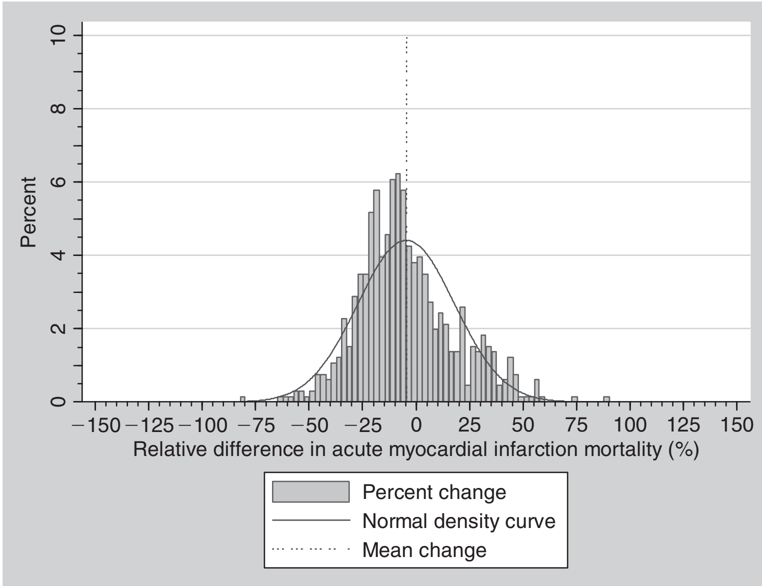
Source: HCUP. Hospitals with smoking restrictions: 15. Control hospitals: 93. Pair-wise comparisons: 296.

Figure 6a. Changes in AMI Admissions Relative to In-State Control Hospitals (Using All States).



Source: MCD. Counties with smoking restrictions: 22. Control counties: 105. Pair-wise comparisons: 262. Counties with smoking restrictions include those counties that have increased the percentage of the population covered by workplace restrictions by at least 50 percent. The change in AMI mortality is then corrected by the change in coverage. Control counties have no workplace restrictions.

Figure 6b. Changes in AMI Deaths Relative to In-State Control Counties (Excluding Smoking Ban Counties with <50 Percent of the Population Covered).



Source: MCD. Counties with smoking restrictions: 9. Control counties: 108. Pair-wise comparisons: 658. Counties with smoking restrictions include those counties that have increased the percentage of the population covered by workplace restrictions by at least 50 percent. The change in AMI mortality is then corrected for the change in coverage. Control counties have no workplace restrictions.

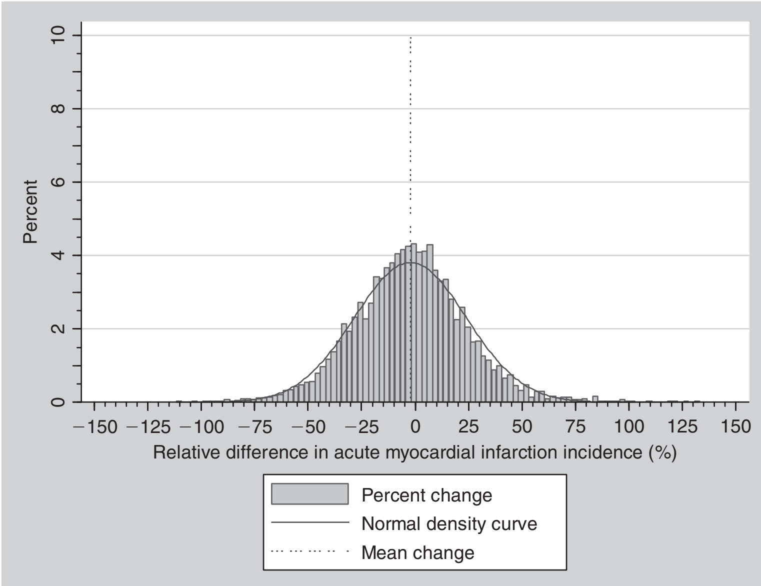
Figure 7. Changes in AMI Deaths Relative to All Control Counties, States with High Smoking Rates (Excludes Smoking Ban Counties with <50 percent of the population Covered).

Second, we restrict the analysis to states with the highest smoking incidence in an effort to discover potential effects in areas where smoking bans might have led to large reductions in ETS exposure. Figure 7 shows that the distribution of year-to-year changes in AMI mortality remains wide and close to zero.[9] We also analyze outcomes following a comprehensive smoking ban (which prohibits smoking in restaurants, bars, and workplaces) by restricting the analysis to control regions (without smoking bans) and those regions with comprehensive smoking bans; we exclude regions with other smoking restrictions. Although these bans would appear more likely to reduce ETS exposure and health outcomes, the results are similar to those with workplace bans (Figure 8).

CONCLUSIONS

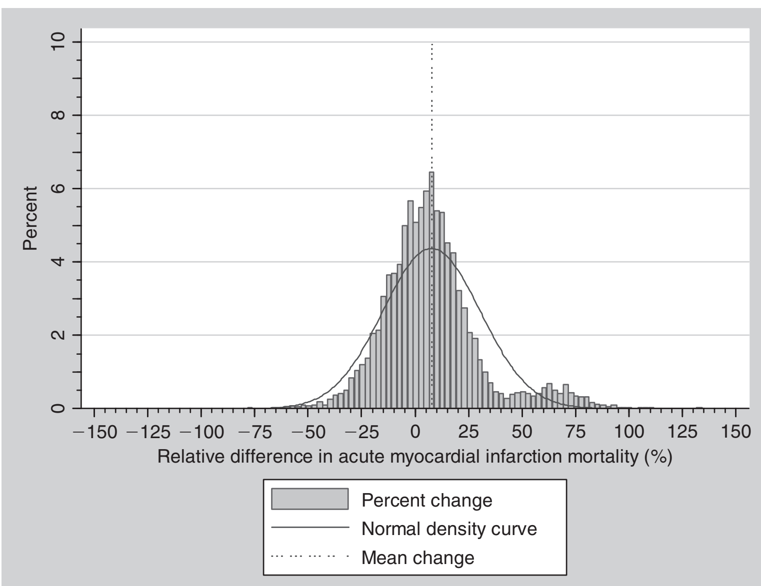
We find no evidence that legislated U.S. smoking bans were associated with short-term reductions in hospital admissions for acute myocardial infarction or other diseases in the elderly, children, or working-age adults. We do not have data on smoking exposure changes for most regions, which makes elucidation of why the aggregate outcomes were null impossible. As such, it remains possible that smoking bans could improve public health outcomes in areas that have limited private restrictions and high baseline smoking rates (such as Scotland); in such cases, public bans might dramatically decrease ETS exposure and adverse health outcomes. However, our results argue against extrapolating from previously published results to typical U.S. cities.

[9] There were too few data to permit a robust analysis of admissions.



Source: HCUP. Hospitals with smoking restrictions: 51. Control hospitals: 481. Pair-wise comparisons: 11,221.

Figure 8a. Changes in AMI admissions relative to all Control Hospitals (Compares Comprehensive Bans with No Bans).



Source: MCD. Counties with smoking restrictions: 25. Control counties: 376. Pair-wise comparisons: 5,680. Comprehensive bans restrict smoking in general workplaces, restaurants, and bars simultaneously

Figure 8b. Changes in AMI Deaths Relative to all Control Counties (Compares Comprehensive Bans with no Bans).

We show that there is wide year-to-year variation in myocardial infarction death and admission rates even in large regions such as counties and hospital catchment areas. Comparisons of small samples (which represent subsamples of our data and are similar to the samples used in the previous published literature) might have led to atypical findings. It is also possible that comparisons showing increases in cardiovascular events after a smoking ban were not submitted for publication because the results were considered implausible. Hence, the true distribution from single regions would include both increases and decreases in events and a mean close to zero, while the published record would show only decreases in events. Publication bias could plausibly explain the fact that dramatic short-term public health improvements were seen in prior studies of smoking bans.

Meyers, Neuberger, and He (2009) considered and discounted publication bias when reviewing the literature. Their dismissal of publication bias rests on the fact that there are some published large-sample studies, mainly from Europe, with ban effect sizes that cluster near zero. The only exception, according to Meyers, Neuberger, and He, is Pell et al. (2008) from Scotland, which is a large study with a large effect size, but also smoking prevalence levels substantially higher than the American setting. All of the published small sample size studies considered by Meyers, Neuberger, and He, all American, found large beneficial short-run effects of bans. In light of the results of our study, contrary to Meyers, Neuberger, and He, our interpretation of the evidence they presented is that it is consistent with publication bias against small studies that find zero effect of bans.[10]

The IOM's (2009) comprehensive review of the literature on bans includes a variety of sources (PubMed, the National Association of City and County Health Officials Web site, the CDC Web site, the American Heart Association Web site, a database of government grants [CRISP], and a clinical trials registry [ClinicalTrials.gov]). They find no published negative studies in the literature; that is, they find no studies where bans failed to have a positive health effect. In light of the evidence that we present in this paper, our reading of the IOM (2009) report is that it too is consistent with publication bias. In both reviews, the evidence for the efficacy of smoking bans appears to rest on a small, heterogeneous group of studies.

We see no evidence of a short-run effect in a large group of short-run studies of bans enacted during 1990 to 2004. Although our results constitute a single publication, they "fill in" the skewed distribution seen in prior reviews by showing the results of a much larger group of short-run studies. We show that positive and negative changes in AMI incidence are equally likely after a smoking ban, which suggests that publication bias, not outcome heterogeneity, explains the skewed results seen in prior reviews. The IOM and other policymakers have relied on the weight of the published literature when making decisions. However, it appears that publication bias did not receive sufficient attention. Our results suggest that only positive studies have been published thus far, and the true short-run effects of governmental workplace smoking bans would be more modest in the U.S. inclusion of such unpublished negative studies might change the conclusions of the IOM and other decision makers on this issue.

Our study focuses only on the short-term health effects of smoking bans. Future research should include non–health related costs and benefits. Prior to a smoking ban, nonsmokers at risk for respiratory symptoms or cardiovascular events might have avoided businesses with high ETS levels. After a ban, nonsmokers could gain comfortable access to these businesses, but this benefit would not result in reduced hospitalization or death rates. In addition, our study design plausibly identifies only

[10] Meyers et al. (2009) justified the skewed study distribution by arguing that longer-run studies were more likely to show an effect; however, even if true, both Meyers et al. (2009) and the IOM (2009) assumed that some smoking bans should have some short-run effect. If so, a small short-run effect should be evident in a sufficiently large sample of short-run studies such as that studied here.

short-term benefits of smoking bans (as have the study designs used by previous studies). We cannot analyze whether smoking bans improve long-term trends for chronic cardiovascular disease or lung cancer. In addition, smoking bans may induce smokers to quit or discourage nonsmokers from starting smoking. These potential long-term benefits will not be apparent in a study of short-term outcomes, and could benefit from further study.

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