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CHANGES IN U.S. HOSPITALIZATION AND MORTALITY RATES FOLLOWING SMOKING BANS

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ABSTRACT

U.S. state and local governments are increasingly restricting 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 workplace 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 workplace ban are as common as the large decreases reported in the published literature.

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1 Introduction

State and local governments have increasingly banned smoking in public places (including workplaces, restaurants and bars) as a means of limiting non-smoker exposure and of discouraging smoking (Centers for Disease Control and Prevention 2007). 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% decrease in the annual incidence of acute myocardial infarction (AMI) (Sargent, Shepard et al. 2004; Bartecchi, Alsever et al. 2006; Cesaroni, Forastiere et al. 2008). 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 implementation of these bans relative to communities that did not implement bans. We use the variation in implementation dates across the country and fixed effects models to control for unobservable factors including improving prevention and treatment of cardiovascular disease, decreasing smoking rates, and smoking restrictions enacted by private

businesses. We find that smoking restrictions are unlikely to substantially affect short-term mortality and hospitalization rates in both the elderly, working-age, and child populations. We find some evidence that smoking bans could reduce mortality in the elderly but the results are not statistically significant (-1.4%, 95% confidence interval: -3.0 to 0.2%).

All previous published studies on the health effects of smoking bans share a common methodology: they compare the outcomes in a single community that has passed a smoking ban with outcomes in a small set of nearby communities that have not passed bans. 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.

2 Background

In this section, we discuss how environmental tobacco smoke (also known as second-hand smoke) is related to health outcomes, the history and effects of smoking bans in the U.S. and internationally, and the implication of previous studies for U.S. smoking policy.

2.1 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 (Glantz and Parmley 1991; He, Vupputuri et al. 1999; Barnoya and Glantz 2005; U.S. Department of Health and Human Services. 2006). Laboratory studies support the notion that small quantities of inhaled cigarette smoke can induce similar biochemical responses in non-smokers as in chronic smokers. Such effects could predispose non-smokers to greatly elevated risk of AMI and stroke. Epidemiologic studies typically compared outcomes non-smoking spouses of smokers and non-smokers. Although some meta-analyses disputed these findings, most agreed that chronic ETS exposure increased risk of AMI by 20 to 30%. 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 (Ong and Glantz 2004; Sargent, Shepard et al. 2004; Bartecchi, Alsever et al. 2006). There is some biological justification for this in the medical literature (U.S. Department of Health and Human Services. 2006). The full

effects of eliminating tobacco smoke may take years to occur because some aspects of coronary artery disease (such as narrowing of the coronary arteries) develop slowly over time. However, a heart attack occurs due to 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 year after a ban if it eliminates even relatively minor exposure. As a result many prior studies examined AMI rates in single regions in the 6 to 18 months following a smoking ban. In addition to increased risk of AMI, those exposed to secondhand tobacco smoke may suffer higher rates of asthma, chronic obstructive pulmonary disease, infections, cancer and other diseases.

2.2 Public bans on smoking in U.S. public places

As evidence on ETS accumulated, many U.S. employers began restricting smoking in the workplace; the proportion of covered workers increased from 25% to 70% between 1986 and 1993 (Farkas, Gilpin et al. 1999; Farrelly, Evans et al. 1999; Centers for Disease Control and Prevention 2000). Following these private restrictions, several communities in California 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 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% in Helena, Montana and 27% in Pueblo, Colorado (relative to surrounding communities) following the imposition of broad restrictions (Sargent, Shepard et al. 2004; Bartecchi, Alsever et al. 2006). 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 2003 (Juster, Loomis et al. 2007). This study estimated that the laws reduced AMI admissions by 8%, 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 smoke more in vehicles, which could be a distraction while driving. Adams and Cotti use national data and hence measures 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.

2.3 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% in younger populations (Barone-Adesi, Vizzini et al. 2006; Cesaroni, Forastiere et al. 2008). The authors demonstrated that the prevalence of smoking, cigarette consumption per smoker, and non-smoker exposure dropped in Italy after the national ban (Barone-Adesi, Vizzini et al. 2006; Gallus, Zuccaro et al. 2007; Cesaroni, Forastiere et al. 2008).[1]

A study of the Scottish public smoking ban found large reductions in AMI rates as well (Pell, Haw 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 non-smokers (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 nanograms/dL in current smokers. The authors then noted statistically significant declines in admissions for acute coronary syndromes in all groups - 14% in smokers, 19% in former smokers and 21% in those who never smoked. The decline in the AMI

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

rate was noticeably larger than the 4% decline in neighboring England during the same time period (which lacked a comprehensive ban).

2.5 Implications of prior work for U.S. smoking policy

The aforementioned studies linking smoking bans to impressive public health gains suggest that widespread public smoking bans would demonstrably improve U.S. public health. However, the international experience also may not translate because 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.

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. By contrast, a simulation study found that extending smoking restrictions from 70% to 100% of U.S. workplaces would prevent roughly 1,500 myocardial infarctions in the first year (Ong and Glantz 2004). Although this may be a clinically relevant improvement, it represents a much smaller reduction

(<1%) than those reported in the small-scale studies based in Helena or Pueblo or the pre-post study of New York State.

The mechanism for these tremendous declines in AMI rates reported in the small-scale studies is unclear as well, which makes their results less certain. It is unknown whether public bans effectively reduce exposure to secondhand smoke and whether the reduced exposure leads to clinically significant cardiovascular risk reduction. There is conflicting evidence as to whether typical government smoking restrictions dramatically reduce exposure to ETS or induce smokers to quit, as restrictions initiated by employers have been shown to do (Longo, Brownson et al. 1996; Evans, Farrelly et al. 1999; Metzger, Mostashari et al. 2005; Adda and Cornaglia 2006). Although Pell et al. (2008) showed that bans were associated with large relative reductions in ETS among non-smokers, the absolute reductions were very small. In addition, 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. It is therefore unclear whether governmental restrictions affect public health substantially, 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 et al. 1999). The

similarity implies exposure to secondhand smoke presents large heath risks at low levels and no additional health risks at higher levels, which seems unlikely.[2]

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 in 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.

3. Data

To construct the data sets used for our analysis, we merge data on the timing and location of smoking bans to three large nationwide data sources on health outcomes.

3.1 Data on smoking bans

We use ordinance data from the American Nonsmokers' Rights Foundation to identify states, counties, and municipalities that implemented restrictions on smoking between 1990 and 2004. We adapt the classification scheme from the American Nonsmokers' Rights Foundation to identify those bans that restrict smoking in all workplaces except bars and restaurants as "workplace" bans. Although not included in

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

the former list, California adopted a nearly complete ban on workplace smoking in 1995. We classified California as smoking-restricted starting in 1995. We also create a dataset of bans of any site - workplaces, bars or restaurants. We classify each 3-digit zip code, city, and county in the U.S. by smoking ban status and date of implementation.

3.2 Data sources for health outcomes

We analyze health outcomes using the Multiple Cause of Death (MCD) database (1989-2004), Medicare claims (1997-2004), and the Nationwide Inpatient Survey (NIS), collected 1993-2004 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 are 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% sample of all discharges from U.S. community hospitals (which includes all non-federal 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. In addition, broad disease measures like AMI and all-cause events are less likely miscoded in administrative data, reducing measurement error (Petersen, Wright et al. 1999;

Tirschwell and Longstreth 2002). We also consider cases of asthma and chronic obstructive pulmonary disease; while these are chronic diseases, secondhand smoke possibly triggers acute exacerbations (U.S. Department of Health and Human Services. 2006). We also identify hip fracture hospitalizations because these can act as a negative control because their incidence would be unlikely to change quickly after a smoking ban (Hoidrup, Prescott et al. 2000). We don't expect the incidence of hip fracture to be affected by smoking bans. If we were to find an association between smoking bans and the incidence of hip fracture, we would question the validity of the empirical strategy.

We assemble each dataset in a similar fashion: we first classify deaths and hospital admissions according to their primary diagnoses (AMI, asthma, etc.); we then sum all outcomes in each region (hospital catchment area, county, or zip code); finally, we merge information on smoking ordinances for that region.

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 (0-17 years), working age adults (18-64 years), and the elderly (65+ years).

From each dataset, we exclude deaths and hospitalizations where we are unable to determine whether the person lived in an area where a smoking restriction was implemented (35-40% 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 1993-2004, hospitals devoted to acute rehabilitation (because few AMIs are admitted) and small hospitals (<1000 admissions/year). The final NIS and MCD samples consist of approximately 60% of all deaths and 4% of all hospital admissions in the U.S. for the period under study. Table 1 summarizes the characteristics of the samples used.

4. Empirical strategy

We estimate region-level fixed effects models using our three national samples. In addition, we perform a number of sensitivity analysis 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.

4.1 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 (e.g, AMI death or hospitalizations), we use the following regression model:

(1) $Outcome_{it} = \alpha_i + \gamma_t + \beta_s SmokingBan_{it} + \varepsilon_{it}$

Here $Outcome_{it}$ represents the number of deaths or hospital admissions in region i (1…N) and time t (1…T), $Outcome_{it}$ is an indicator for each year, and ε_{it} is the error term. We include α_i, an indicator for each region (county, 3 digit zip code, or hospital), to control for idiosyncratic differences between regions. β_s is the coefficient of interest, representing 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 $(\frac{\beta_s}{\mu} \times 100\%)$ where μ is $\frac{1}{N \times T} \sum_{i=1}^{N} \sum_{t=1}^{T} Outcome_{it}$. 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.[3]

4.2 Sensitivity analyses.

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

$$(2)\ Outcome_{it} = \alpha_i + \gamma_t + \beta_s SmokingBan_{it} + \mathbf{\beta X} + \varepsilon_{it}$$

The terms and results are the same as in (1) except for the addition of $\mathbf{\beta X}$, 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 as well as hospitals in the NIS data. We do not include these variables (which could potentially add explanatory power) in

[3] 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 considered patients with serious illnesses who tend to be taken to the nearest hospital, which further reduces bias.

all model specifications because data for $\boldsymbol{\beta X}$ 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.

4.3 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. (2005) and Sargent et al. (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 (6-18 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. We then subtract the change in outcomes in each contemporaneous control hospital from each intervention hospital. The resulting dataset consists of the universe of possible (19,406) 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 dataset consists of 23,938 pair-wise comparisons and represents the universe of such comparisons in the MCD data.

5. Results

5.1 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 2003-2004 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 (Farrelly, Evans et al. 1999; Centers for Disease Control and Prevention 2000).

5.2 Main estimates

Workplace smoking restrictions are unrelated to changes in all-cause mortality or mortality due to other AMI in all age groups. Restrictions on smoking of any sort are associated with reduced all-cause mortality among the elderly (-1.4%, 95% CI: -3.0 to 0.2%) but the result is only significant at the 10% level (p=0.06) (see Table 2).

We find no statistically significant reduction in admissions due to AMI among working-age adults (-4.2%, 95% CI: -10.2 to 1.7%, p=0.165) or among the elderly (2.0%,

95% CI: -3.7 to 7.7%, p =0.48) following the enactment of a workplace smoking restriction (see Table 3). We similarly find no evidence of reduction in admissions for other diseases in any age group, though smoking restrictions of all sorts are associated with statistically insignificant increases in asthma (11.4%, 95% CI: -2.4 to 25.3%, p=0.11) and total admissions (3.7, 95%CI: -2.1 to 9.5%, p = 0.21) among children. Among both the elderly and working-age adults, we find no statistically significant effect of smoking bans on hip fracture admissions, our negative control. This supports the hypothesis that unobserved characteristics of regions do not confound our results.

5.3 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 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 +0.330% (with a standard error of 0.794%) without additional controls and +0.328% (with a standard error of 0.804%) with controls for hospital beds per capita and physicians per capita. 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 (Hochberg 1988), our standard errors increase further.

5.5 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% or greater declines and 10% of 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 very large decreased 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.

6. 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 find some evidence that smoking bans are associated with a reduced all-cause mortality rate among the elderly (-1.4%) but only at the 10% significance level.

We also 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. Thus, publication bias could plausibly explain why dramatic short-term public health improvements were seen in prior studies of smoking bans.

Our study focuses only on the health effects of smoking bans. Future research should estimate non-health related benefits of these bans to non-smokers. Prior to a smoking ban, non-smokers at risk for respiratory symptoms or cardiovascular events might have avoided businesses with high ETS levels. After a ban, non-smokers could gain comfortable access to these businesses, but based on our findings in this study, this benefit would not also result in reduced hospitalization or death rates. Our study design plausibly identifies only short-term benefits of smoking bans (as has 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 study of short-term outcomes and would benefit from further study.

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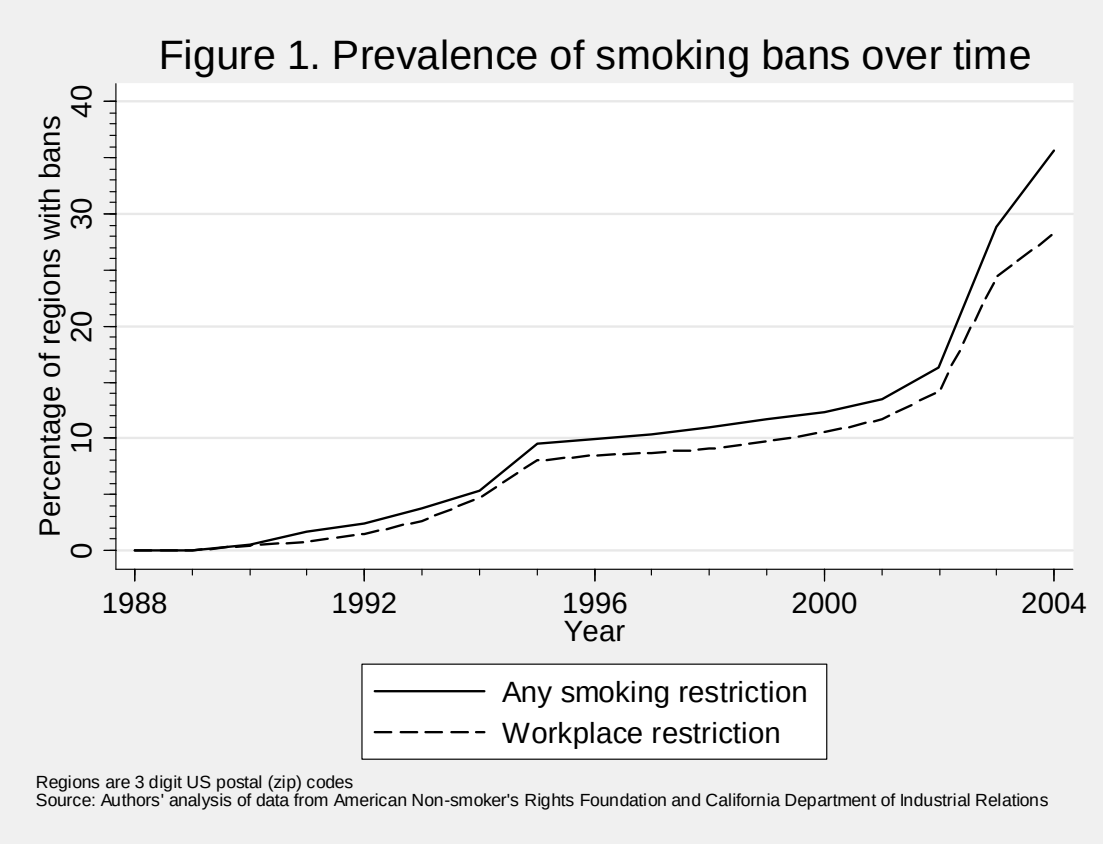
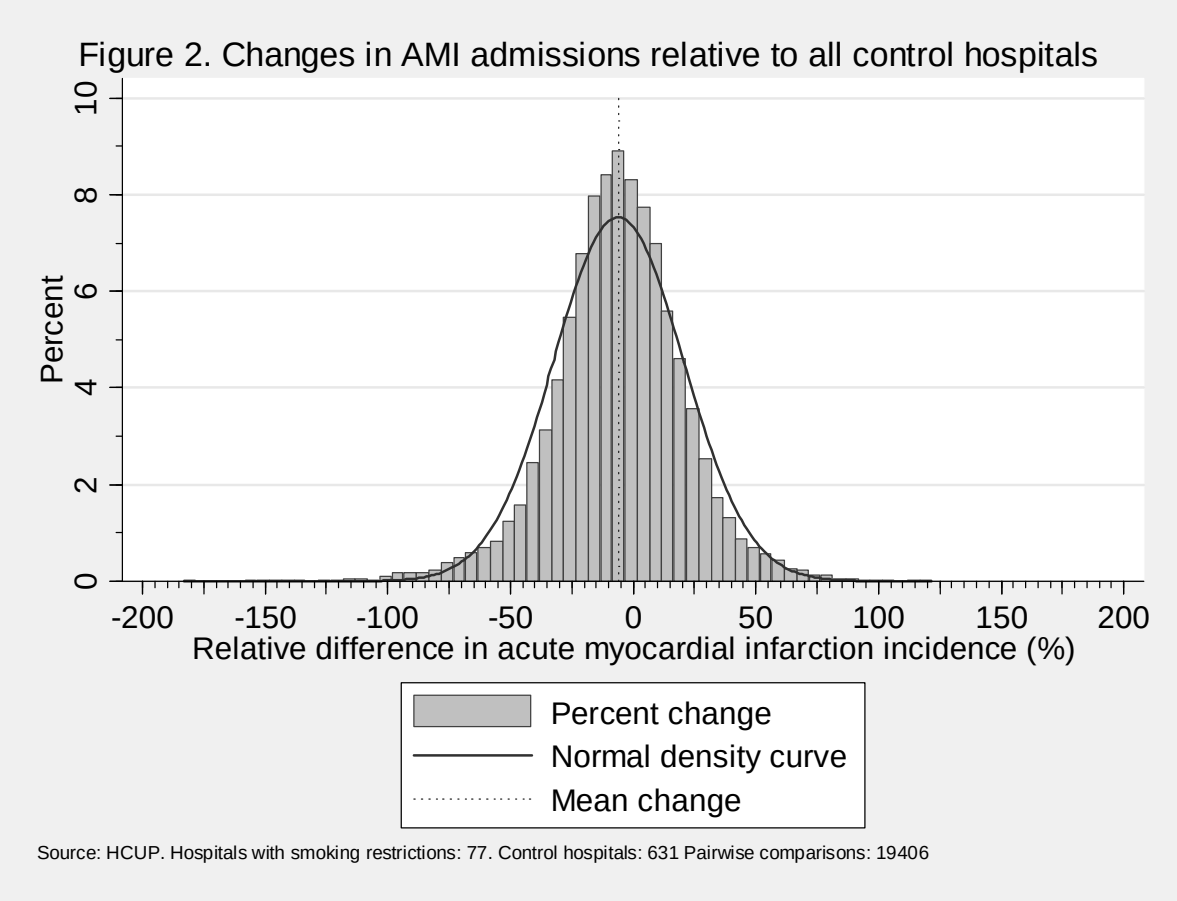
Figure 1



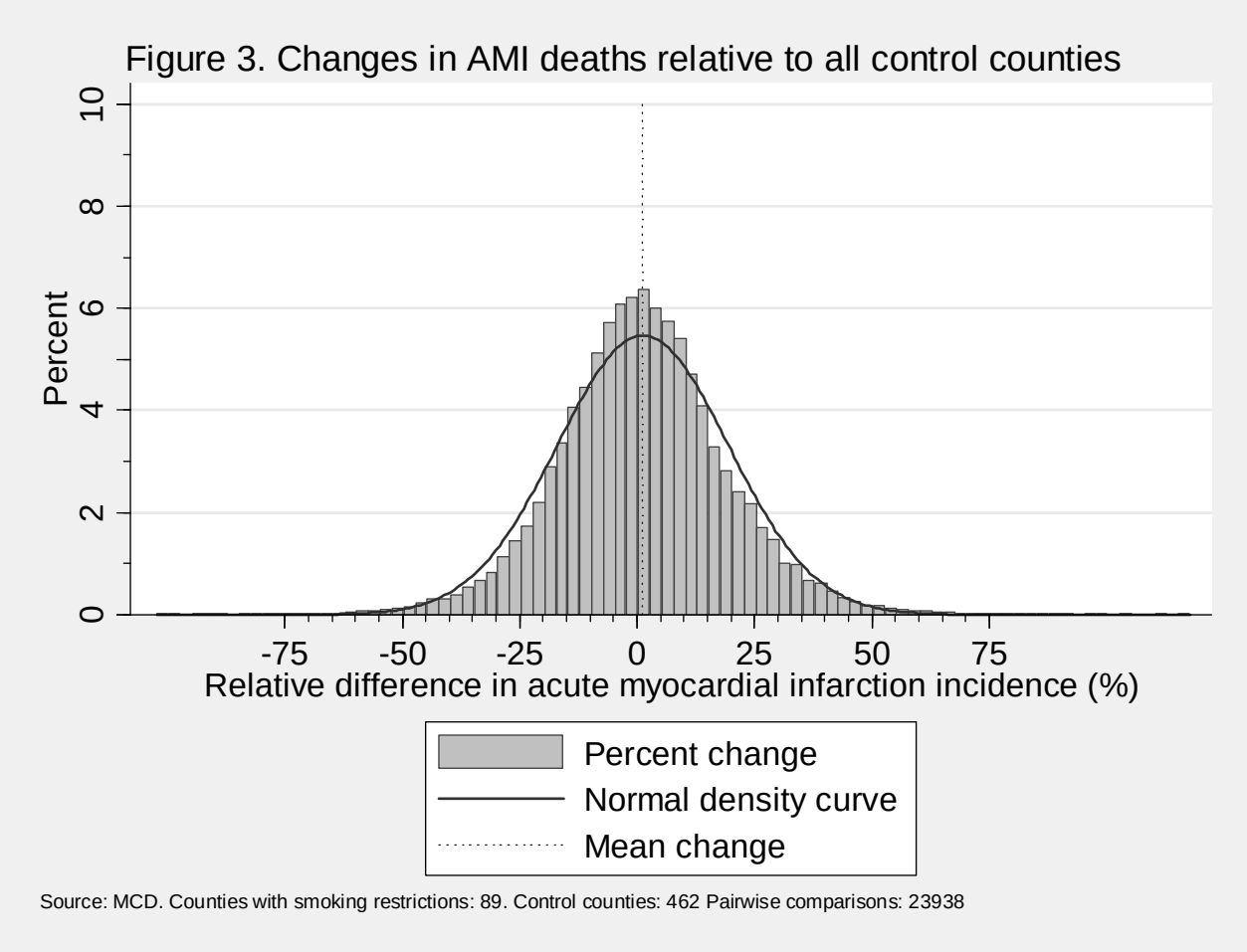
Regions are 3 digit U.S. postal (zip) codes
Source: Authors' analysis of data from American Non-smoker's Rights Foundation and California Department of Industrial Relations

Figure 2.



Source: Authors' analysis of data from Nationwide Inpatient Sample. Hospitals with smoking restrictions: 52. Control hospitals: 336. Pair-wise comparisons: 8,529. Relative difference may be less than -100% if a large percentage increase in a control hospital is subtracted from a large decrease in a smoking-restricted hospital.

Figure 3.



Source: Authors' analysis of data from Multiple cause of death database. Counties with smoking restrictions: 89. Control counties: 462. Pair-wise comparisons: 23,905. Relative difference may be less than -100% if a large percentage increase in a control county is subtracted from a large decrease in a smoking-restricted county.

Table 1. Data source characteristics

Data source	
Nationwide Inpatient Sample:	
Hospitals	874
States	27
Years	1993-2004
All admissions	21,820,484
Admissions by disease	
Acute myocardial infarction	217023
Combined asthma and COPD§	433674
Multiple Cause of Death:	
Counties	468
States	50
Years	1989-2004
All deaths	24,610,532
Acute myocardial infarction	2,042,812
Medicare patients:	
Regions (3 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	2,382,386
Combined asthma and COPD§	2,984,382
Hip fracture	3,381,690

§Chronic obstructive pulmonary disease

Source: Authors' analysis of data from Nationwide Inpatient Sample, Multiple Cause of Death files, and 100% Medicare Provider Analysis and Review files.

§Chronic obstructive pulmonary disease

Source: Authors' analysis of data from Nationwide Inpatient Sample, national death statistics, and 100% Medicare Provider Analysis and Review files.

Table 2. Mortality and smoking restrictions*

Disease	% change in mortality (95% CI)	P value
Workplace smoking restrictions:		
All deaths (0-17 years old)	-4 (-10.2 to 2.2)	0.204
AMI□ (18-64 years old)	-4.4 (-11.6 to 2.7)	0.224
All deaths (18-64 years old)	-1.1 (-2.9 to 0.8)	0.247
All deaths (age 65+)	0.8 (-1.2 to 2.8)	0.413
AMI (all ages)	-1.5 (-4.8 to 1.8)	0.374
All deaths (all ages)	-0.3 (-1.6 to 0.9)	0.624
Any smoking restrictions:		
All deaths (0-17 years old)	-2 (-6.5 to 2.6)	0.400
AMI (18-64 years old)	-3.5 (-10 to 3.1)	0.299
All deaths (18-64 years old)	-0.4 (-2.1 to 1.2)	0.601
All deaths (age 65+)	-1.4 (-3.0 to 0.2)	0.062
AMI (all ages)	-1.1 (-4.1 to 1.9)	0.470
All deaths (all ages)	0.5 (-0.6 to 1.6)	0.396

*Authors analysis of data from Multiple Cause of Death files, years 1993-2004 except in age 65+, which are from 100% Medicare Provider Analysis and Review files, 1997-2004.
□ AMI indicates deaths from acute myocardial infarction.

Table 3. Hospital admissions and smoking restrictions

Disease	% change in admissions (95% CI)	P value
Workplace smoking restrictions:		
All admissions (age 0-17)	3.7 (-2.1 to 9.5)	0.211
Asthma (age 0-17)	11.4 (-2.4 to 25.3)	0.106
AMI (age 18-64)	-4.2 (-10.2 to 1.7)	0.165
All admissions (age 18-64)	2.2 (-0.4 to 4.8)	0.101
Asthma (age 18-64)	3.4 (-6.2 to 13.1)	0.485
COPD (age 18-64)	1.7 (-8.6 to 12)	0.746
Hip fracture (age 18-64)	-5.1 (-15.6 to 5.4)	0.340
AMI (age 65+)	2 (-3.7 to 7.7)	0.477
All admissions (age 65+)	1.8 (-3.1 to 6.7)	0.477
Asthma (age 65+)	0.8 (-13.5 to 15.1)	0.909
COPD (age 65+)	3.4 (-3.6 to 10.5)	0.343
Hip fracture (age 65+)	0.1 (-2.4 to 2.6)	0.946
Any smoking restrictions:		
All admissions (age 0-17)	4.6 (-0.9 to 10.1)	0.101
Asthma (age 0-17)	13.7 (-2 to 29.3)	0.087
AMI (age 18-64)	-4.7 (-10.3 to 01)	0.104
All admissions (age 18-64)	2.3 (-0.3 to 4.9)	0.083
Asthma (age 18-64)	3.5 (-5 to 11.9)	0.422
COPD (age 18-64)	-2.1 (-11.8 to 7.7)	0.676
Hip fracture (age 18-64)	-4.7 (-14.6 to 5.1)	0.348
AMI (age 65+)	5.1 (-0.4 to 10.6)	0.068
All admissions (age 65+)	1.7 (-2.0 to 5.4)	0.364
Asthma (age 65+)	6.8 (-4.78 to 18.4)	0.246
COPD (age 65+)	2.7 (-2.4 to 7.8)	0.303
Hip fracture (age 65+)	1.1 (-1.4 to 3.6)	0.385

Source: Authors' analysis of data from Nationwide Inpatient Sample(1993-20004) except age 65+, which are from 100% Medicare Provider Analysis and Review files, years 1997-2004.

 Acute myocardial infarction/Ischemic heart disease

¶ Chronic obstructive pulmonary disease

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

Variables included	(1)	(2)	(3)	(4)
Any bans	0.495	0.324	0.328	1.335
	(0.583)	(0.573)	(0.794)	(0.547)
Hospital beds/person			0.0031	
			(0.0013)	
County population	0.00005	0.00006	0.00005	0.00009
	(0.00002)	(0.00002)	(0.00002)	(0.00003)
Physicians/person		0.0003	0.0002	-0.0007
		(0.0007)	(0.0008)	(0.0010)
Percent population in labor force				4.5
				(6.6)
# Years in sample	15	13	9	5
# observations	24884	21580	15512	7308
Results from original model	0.495	0.322	0.330	1.343
	(0.583)	(0.573)	(0.804)	(0.546)

***Each coefficient represents the % 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 5. Comparison of regression models:
Myocardial infarction admission rates in working-age adults (NIS hospitals) following workplace bans

Variables included	(1)	(2)	(3)	(4)
Workplace bans	-4.2	-4.1	-13.3	-9.2
	(3.1)	(3.0)	(5.8)	(5.9)
Hospital beds/person			0.0054	
			(0.0029)	
County population	0.00004	0.00004	0.00003	0.00023
	(0.00003)	(0.00003)	(0.00002)	(0.00008)
Physicians/person		0.0024	0.0020	-0.0027
		(0.0025)	(0.0028)	(0.0077)
Percent population in labor force				310.3
				(324.9)
# Years in sample	12	12	9	5
# observations	7476	7476	4932	1502
Results from original model on this sample	-4.2	-4.2	-13.3	-9.2
	(3.1)	(3.1)	(5.8)	(5.7)

***Each coefficient represents the % 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 hospitals)

Variables included	(1)	(2)	(3)	(4)
Workplace bans	2.20	2.12	0.35	1.52
	(1.34)	(1.33)	(1.73)	(2.10)
Hospital beds/person			0.000297	
			(0.000998)	
County population	0.00004	0.00004	0.00003	0.00013
	(0.00002)	(0.00002)	(0.00002)	(0.00004)
Physicians/person		-0.00126	-0.00119	-0.00185
		(0.000993)	(0.00109)	(0.00299)
Percent population in labor force				-8.2
				61.8)
# Years in sample	12	12	9	5
# observations	7476	7476	4932	1502
Results from original model	2.20	2.20	0.26	1.40
	(1.34)	(1.34)	(1.73)	(2.08)

***Each coefficient represents the % 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.**