ONLINE FIRST | HEALTH CARE REFORM

Myocardial Infarction and Sudden Cardiac Death in Olmsted County, Minnesota, Before and After Smoke-Free Workplace Laws

Richard D. Hurt, MD; Susan A. Weston, MS; Jon O. Ebbert, MD; Sheila M. McNallan, MPH; Ivana T. Croghan, PhD; Darrell R. Schroeder, MS; Véronique L. Roger, MD, MPH

Background: Reductions in admissions for myocardial infarction (MI) have been reported in locales where smoke-free workplace laws have been implemented, but no study has assessed sudden cardiac death in that setting. In 2002, a smoke-free restaurant ordinance was implemented in Olmsted County, Minnesota, and in 2007, all workplaces, including bars, became smoke free.

Methods: To evaluate the population impact of smoke-free laws, we measured, through the Rochester Epidemiology Project, the incidence of MI and sudden cardiac death in Olmsted County during the 18-month period before and after implementation of each smoke-free ordinance. All MIs were continuously abstracted and validated, using rigorous standardized criteria relying on biomarkers, cardiac pain, and Minnesota coding of the electrocardiogram. Sudden cardiac death was defined as out-of-hospital deaths associated with coronary disease.

Results: Comparing the 18 months before implementation of the smoke-free restaurant ordinance with the 18 months after implementation of the smoke-free workplace law, the incidence of MI declined by 33% ($P < .001$), from 150.8 to 100.7 per 100 000 population, and the incidence of sudden cardiac death declined by 17% ($P = .13$), from 109.1 to 92.0 per 100 000 population. During the same period, the prevalence of smoking declined and that of hypertension, diabetes mellitus, hypercholesterolemia, and obesity either remained constant or increased.

Conclusions: A substantial decline in the incidence of MI was observed after smoke-free laws were implemented, the magnitude of which is not explained by community cointerventions or changes in cardiovascular risk factors with the exception of smoking prevalence. As trends in other risk factors do not appear explanatory, smoke-free workplace laws seem to be ecologically related to these favorable trends. Secondhand smoke exposure should be considered a modifiable risk factor for MI. All people should avoid secondhand smoke to the extent possible, and people with coronary heart disease should have no exposure to secondhand smoke.

Arch Intern Med.
Published online October 29, 2012.
doi:10.1001/2013.jamainternmed.46

Author Affiliations: Research Program, Nicotine Dependence Center and Department of Internal Medicine (Drs Hurt, Ebbert, and Croghan), Division of Cardiovascular Diseases (Dr Roger), and Department of Health Sciences Research (Mss Weston and McNallan, Mr Schroeder, and Dr Roger), Mayo Clinic, Rochester, Minnesota.

SECONDHAND SMOKE (SHS) EXposure is associated with coronary heart disease (CHD) in nonsmokers, and a nonlinear dose relationship exists such that SHS exposure has a larger than expected adverse effect at low levels of exposure.[1] Research suggests that the cardiovascular effects of SHS are nearly as large as those with active smoking.[1] Indeed, the magnitude of endothelial dysfunction in nonsmokers approaches that noted in active smokers[2] and may be only partially reversible.[3] The 2006 US Surgeon General's Report[4] underscored the negative impact of SHS, stating that "exposure to SHS has immediate effects on the cardiovascular system." An estimated 46 000 nonsmoking Americans die annually from cardiovascular events related to SHS.[5] Eliminating smoking in public places holds potential for improving public health and reducing cardiovascular events beyond the expected impact of the reduction in active smoking.[6] Finding additional scientific data in support of smoke-free policies will help to ensure their continuation and encourage locales without such laws to consider them.

See also Invited Commentary

Several studies[7-14] described a decline in hospital admissions for myocardial infarction (MI) after implementation of smoke-free legislation, and a meta-analysis[15] of 17 published studies reported a 10% reduction in admissions for MI after the implementation of such legislation. An expert committee from the Institute of Medicine[16] concluded that "there is a causal relationship between smoking bans and decreases in acute coronary events," but the committee did not determine the magnitude of the decrease in relative risk. The 10-member committee concluded that none of the studies to date was of ideal design. To evaluate the population effect of the implementation of smoke-free laws on cardiovascular events, we examined data from the Rochester Epidemiology Project, including individual patient data for MI cases validated using rigorous epidemiologic criteria and sudden cardiac death (SCD) in Olmsted County, Minnesota, before and after implementation of smoke-free workplace laws.

METHODS

STUDY SETTING

Olmsted County, located in southeastern Minnesota, has a population of 144 248 (86% white, 51% female).[17] Only a few providers (chiefly Mayo Clinic and Olmsted Medical Center) deliver nearly all medical care to county residents. Medical records used by each provider capture information for all encounters and can be retrieved from indices based on all diagnoses and procedures maintained by Mayo Clinic.[18] This results in the linkage of medical records from all sources of care, providing a unique infrastructure to analyze disease occurrence and outcomes at the population level. Potential cases identified through the Rochester Epidemiology Project can then be validated by applying standardized methods appropriate for each disease entity. This process has been implemented for extensive cardiovascular disease epidemiologic research.[19-22]

In Olmsted County, a smoke-free restaurant law that did not include bars or other workplaces (Ordinance 1) was implemented on January 1, 2002, and on October 1, 2007, all workplaces (including bars) became smoke-free (Ordinance 2).

ASCERTAINMENT OF MI

Potential cases of MI included patients admitted to Olmsted County hospitals whose diagnosis was assigned the *International Classification of Disease, Ninth Revision* code 410 (acute MI). Infarctions were validated after manual data collection of relevant information and using algorithms integrating cardiac pain as well as electrocardiographic (ECG) and biomarker data (creatine kinase and MB fraction of creatine kinase).[23,24] Biomarker values were recorded for up to 3 measurements on each of the first 3 days after admission or MI onset, if the patient was already hospitalized.[25,26] All biomarkers were measured with a sandwich electrochemiluminescence immunoassay (Elecsys 2010; Roche Diagnostics Corporation) in the certified laboratories of the Department of Laboratory Medicine and Pathology at Mayo Clinic in Rochester, Minnesota, with robust quality control in place. Three ECGs per episode were coded using the Minnesota Code Modular ECG Analysis System.[27] Only first-ever MIs were considered as incident. All MIs were continuously abstracted for the duration of this study. These methods have been used in the Rochester Epidemiology Project coronary disease surveillance work for more than a decade, with excellent reliability.[23,25,28]

ASCERTAINMENT OF SCD

Our methods of ascertaining SCD have been published.[22,29] In brief, SCD was defined as out-of-hospital deaths with the primary cause of death classified as CHD on the death certificate (*International Classification of Diseases, Ninth Revision* codes 410-414).[29]

CLINICAL DATA

Baseline demographic and clinical characteristics were collected from the medical records by nurse abstractors at the time of MI diagnosis or SCD. Clinicians' diagnoses were used to define hypertension, hyperlipidemia, diabetes mellitus, familial CHD, and smoking status. Body mass index was calculated as weight in kilograms divided by height in meters squared.

SMOKING PREVALENCE AND OTHER CARDIOVASCULAR RISK FACTORS

We used Behavioral Risk Factor Surveillance System (BRFSS) data from Minnesota adults for self-reported current smoking prevalence, hypercholesterolemia, diabetes mellitus, hypertension, and obesity (body mass index, ≥30).[30] The BRFSS is a state-based system of health surveys collecting information on health risk behaviors, preventive health practices, and health care access primarily related to chronic disease and injury. Telephone interviews are conducted with more than 350 000 adults in all 50 states, the District of Columbia, and US territories.[31] No Olmsted County–specific data for these risk factors exist.

STATISTICAL ANALYSIS

Unadjusted and age- and sex-adjusted incidence rates of MI and SCD were calculated for 18 months before and 18 months after implementation of each smoking law. The counts of events were used as the numerators, and the denominators were the Olmsted County population as determined by census data for the years 1990, 2000, and 2010, with linear interpolation for intercensal years. Adjusted rates were directly standardized to the age and sex distribution of the 2000 US population. Calculation of standard errors and 95% CIs was based on the Poisson error distribution. Differences in incidence in the 18 months before and after implementation of each law were assessed with Poisson regression, adjusting for age and sex and using an indicator variable with a value of 0 or 1 for the 18 months before and after the law, respectively. Specific counts for each period, age, and sex were used as the unit of observation, with the period-, age-, and sex-specific Olmsted County population as the offset. Interactions between age and period were assessed to determine whether changes in incidence rates were dependent on age. In all cases, no significant interactions were detected.

Cause of death was missing for 128 (3.7%) of the 3480 persons who died out-of-hospital during the 18 months before and after each smoke-free ordinance. To account for these missing data, multiple imputation methods were used to create 5 complete imputed data sets.[32] Analyses were performed for each imputed data set, and the results were combined using Rubin rules.[33] Nonlinear trends were explored using data from all years from 1995 to 2009. For these analyses, Poisson regression models were generated with the use of generalized additive models[34] with calendar year as a smoothed term.

Analyses were performed using commercial software (SAS, version 9.2; SAS Institute Inc) and R (http://www.R-project.org). All aspects of the study were approved by the Mayo Clinic institutional review board.

RESULTS

PATIENT CHARACTERISTICS

During the 18 months before and after implementation of each smoke-free ordinance, there was a total of 717 incident cases of MI and 514 people who experienced

SCD. Demographic and clinical characteristics of the patients are reported in **Table 1**. No characteristics were found to differ significantly between the before and after ordinance periods, except for hyperlipidemia in persons experiencing SCD before and after Ordinance 1 (36.4% vs 54.0%, respectively; *P*=.004).

INCIDENCE OF MI

The age- and sex-adjusted rate of MI was 150.8 per 100 000 (95% CI, 129.0-172.6) for the 18 months before Ordinance 1 and 144.6 per 100 000 (95% CI, 123.6-165.5) for the 18 months after Ordinance 1 (**Table 2**). The incidence of MI did not significantly decline during this period (age- and sex-adjusted relative risk [RR], 0.96; 95% CI, 0.78-1.18; *P* = .71).

Conversely, for the period surrounding Ordinance 2, the incidence of MI adjusted for age and sex declined from 152.3 per 100 000 (95% CI, 131.4-173.3) to 100.7 per 100 000 (95% CI, 83.8-117.5). This equated to a 34% decline over the 18 months before and after implementation of Ordinance 2 (adjusted RR, 0.66; 95% CI, 0.53-0.82; *P* < .001). Over the entire study period comparing 18 months before Ordinance 1 and 18 months after Ordinance 2, we observed a 33% decline in the incidence of MI (adjusted RR, 0.67; 95% CI, 0.53-0.83; *P* < .001).

The annual unadjusted MI incidence rates from 1995 to 2009 are presented in **Figure 1**. A smoothing spline with 95% CI is superimposed on the actual rates. The test for nonlinearity was significant (*P* = .009), with an inflection point occurring around 2006.

Table 1. Patient Characteristics

Characteristic	MI (n = 717), No. (%)	SCD (n = 514)[a], No. (%)
Age, mean (SD), y	67.8 (15.2)	77.4 (15.3)
Median (IQR)	69 (56-80)	82 (66-89)
Female sex	274 (38.2)	221 (43.0)
Hypertension	461 (67.0)	339 (73.5)
Missing data	29	53
Hyperlipidemia	419 (60.9)	243 (52.6)
Missing data	29	52
Current smoking	173 (25.1)	72 (15.7)
Missing data	29	56
Diabetes mellitus	154 (22.4)	111 (24.2)
Missing data	29	56
BMI		
Normal, <25	196 (27.6)	188 (41.4)
Overweight, 25-29	268 (37.8)	140 (30.8)
Obese, ≥30	245 (34.6)	126 (27.8)
Missing data	8	60
Familial coronary heart disease	142 (20.9)	56 (12.2)
Missing data/unknown	38	57

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); IQR, interquartile range; MI, myocardial infarction; SCD, sudden cardiac death.

[a] Data are presented for individuals who died out-of-hospital and had data available on the cause of death. Age and sex were obtained from the death certificates. For all other characteristics, patients who had not provided consent for the use of their medical records for research purposes are not included.

INCIDENCE OF SCD

No decline in SCD was observed for the 18 months before and after Ordinance 1 (adjusted RR, 1.01; 95% CI, 0.80-1.27; *P* = .96), with the age- and sex-adjusted rates of SCD being 109.1 per 100 000 (95% CI, 91.0-127.2) and 112.7 per 100 000 (95% CI, 94.3-131.0), respectively (Table 2). Similarly, there was not a significant change in rates of SCD 18 months before Ordinance 2 compared with 18 months after Ordinance 2 (adjusted RR, 1.17; 95% CI, 0.91-1.51; *P* = .22). The age- and sex-adjusted rates of SCD were 78.8 per 100 000 (95% CI, 64.0-93.5) 18 months before the ordinance and 92.0 per 100 000 (95% CI, 75.7-108.3) 18 months after the ordinance. There was a 17% decline in the incidence of SCD for the overall study period comparing the 18 months before Ordinance 1 with the 18 months after Ordinance 2 (adjusted RR, 0.83; 95% CI, 0.65-1.06; *P* = .13).

The annual SCD rates from 1995 to 2009 are presented in Figure 1. A smoothing spline with 95% CI is superimposed on the unadjusted rates. No significant difference was found in the test for nonlinearity (*P* = .09).

Table 2. Incidence Rates and Relative Risks of MI and SCD 18 Months Before and After Implementation of Smoke-Free Laws

Characteristic	Before, No.	Before, Rate per 100 000 (95% CI)[a]	After, No.	After, Rate per 100 000 (95% CI)[a]	Adjusted RR, (95% CI)[a]	*P* Value
MI						
Ordinance 1	187	150.8 (129.0-172.6)	185	144.6 (123.6-165.5)	0.96 (0.78-1.18)	.71
Ordinance 2	206	152.3 (131.4-173.3)	139	100.7 (83.8-117.5)	0.66 (0.53-0.82)	<.001
Before Ordinance 1 vs after Ordinance 2	187	150.8 (129.0-172.6)	139	100.7 (83.8-117.5)	0.67 (0.53-0.83)	<.001
SCD						
Ordinance 1	143	109.1 (91.0-127.2)	148	112.7 (94.3-131.0)	1.01 (0.80-1.27)	.96
Ordinance 2	111	78.8 (64.0-93.5)	133[b]	92.0 (75.7-108.3)	1.17 (0.91-1.51)	.22
Before Ordinance 1 vs after Ordinance 2	143	109.1 (91.0-127.2)	133	92.0 (75.7-108.3)	0.83 (0.65-1.06)	.13

Abbreviations: MI, myocardial infarction; RR, relative risk; SCD, sudden cardiac death.

[a] Adjusted for age and sex.

[b] Cause of death was missing for 3.7% of out-of-hospital deaths. The number reported herein represents the estimated number of SCDs obtained via multiple imputation for missing data.

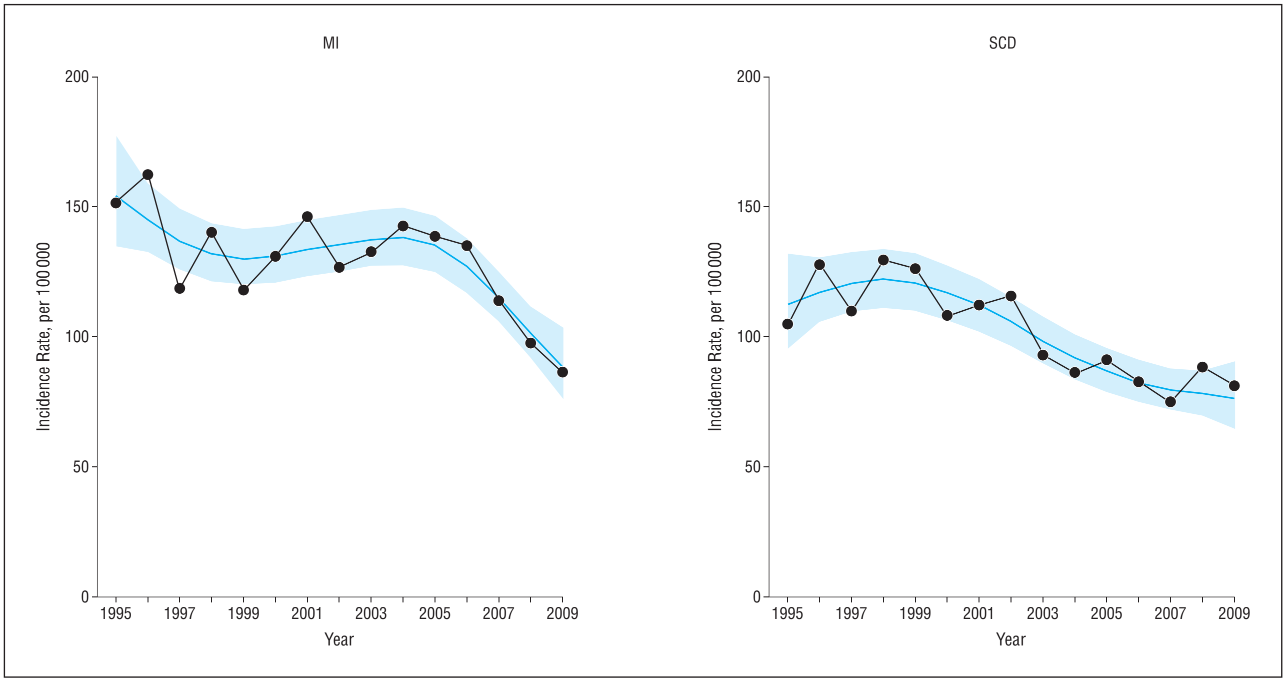

Figure 1. Incidence (data points) of myocardial infarction (MI) and sudden cardiac death (SCD) in Olmsted County, Minnesota, 1995-2009, with smoothing spline (solid lines) and 95% CIs (shaded areas).

SMOKING PREVALENCE AND OTHER CARDIOVASCULAR RISK FACTORS

Based on the Minnesota BRFSS data,[30] the self-reported smoking prevalence among adults decreased from 19.8% in 2000 to 14.9% in 2010. During the same period, the prevalence of diabetes mellitus and obesity increased, and the prevalence of hypertension and hypercholesterolemia remained flat (**Figure 2** and **Figure 3**).

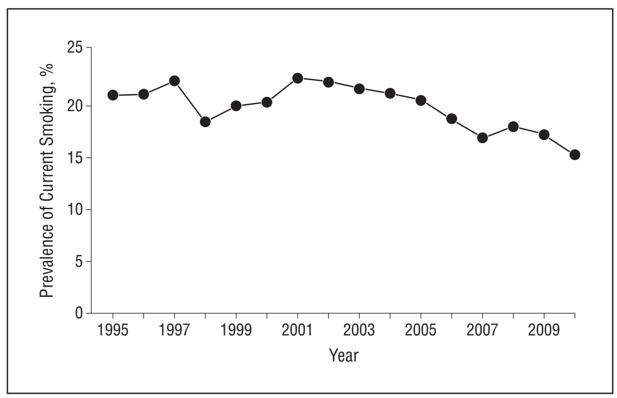

Figure 2. Prevalence of self-reported current smoking in Minnesota, 1995-2010, from Behavioral Risk Factor Surveillance System Survey data.[30]

COMMENT

We report a substantial decline in the incidence of MI from 18 months before the smoke-free restaurant law was implemented to 18 months after the comprehensive smoke-free workplace law was implemented 5 years later. This decrease in the incidence of MI is similar to the 40% decrease in the first report in Helena, Montana.[7] In a large study[14] that included biomarker confirmation, the rate of admission to Scottish hospitals decreased by 19% after a smoke-free workplace law was implemented. This study was limited by its inclusion of only 9 hospitals, which accounted for 64% of the country's hospital admissions; thus, the rates were not true incidence rates, and the time frame was much shorter than ours.

The mechanisms of the deleterious effect of SHS are diverse. As few as 5 minutes of exposure to SHS in nonsmokers reduces aortic distensibility,[35] and abdominal aortic stiffness is increased with SHS exposure in children.[36] In nonsmokers, 30 minutes of SHS exposure produces an abrupt and dramatic reduction in coronary artery flow velocity reserve.[37] Thirty minutes of SHS exposure leads to vascular injury characterized by mobilization of dysfunctional endothelial progenitor cells with blocked nitric oxide production, which is essential to endothelial function because its release mediates vasodilatation.[38] Exposure to SHS in nonsmokers has been associated with lower high-density lipoprotein cholesterol levels, increased markers of inflammation, increased serum fibrinogen and homocysteine, decreased antioxidant levels, and increased insulin resistance.[39-43]

CAUSAL INFERENCE

Although it does not embody the "ideal design" as described by the Institute of Medicine report,[16] our study addresses some key limitations by reporting data from a defined community with a large sample size and a prolonged period of observation. Because our data constitute before vs after comparisons in the absence of a comparison geographic area in which no smoke-free law has been implemented, its interpretation should also explicitly consider alternative causes of changes in MI incidence. To this end, the new diagnostic criteria for MI were deployed in 2000;

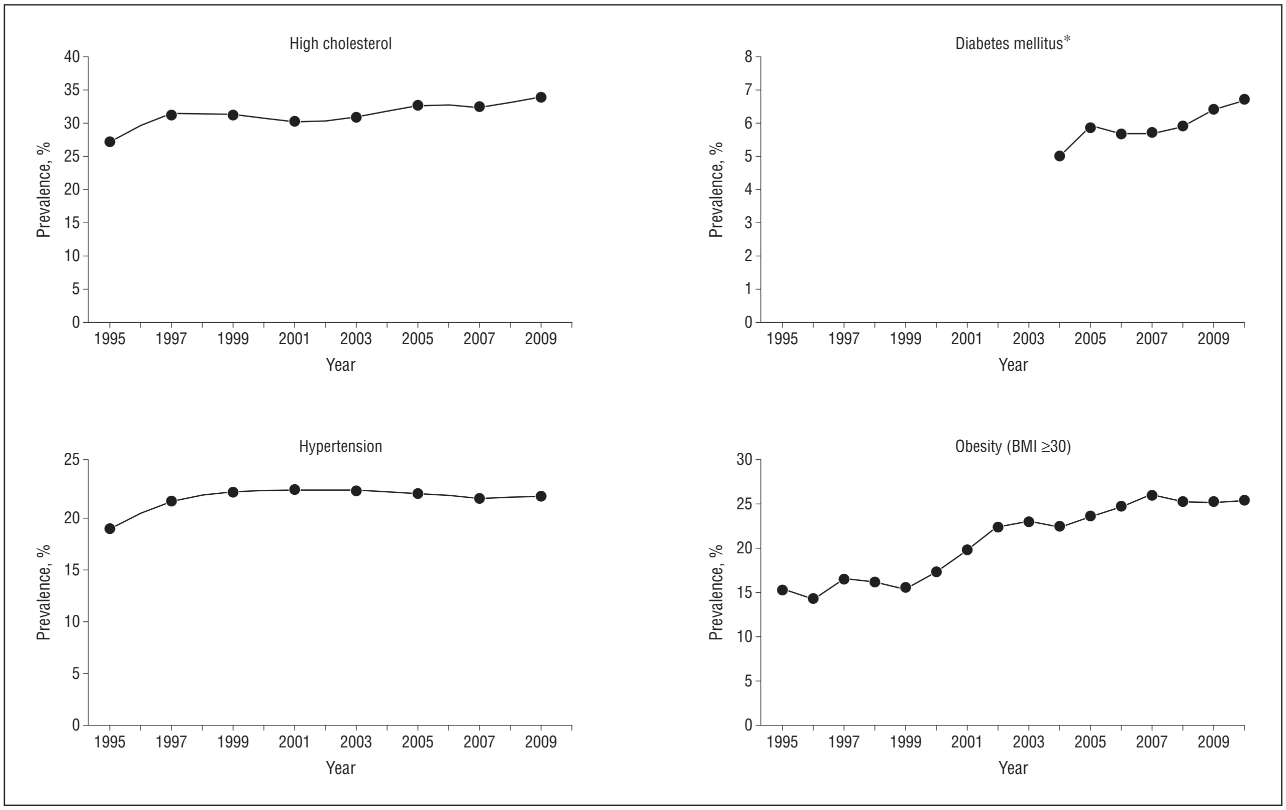

Figure 3. Prevalence of self-reported high cholesterol level, diabetes mellitus, hypertension, and obesity (body mass index [BMI] ≥30; calculated as weight in kilograms divided by height in meters squared) in Minnesota, 1995-2010, from Behavioral Risk Factor Surveillance System survey data.[30] *Data on diabetes were not available for the entire study period.

these new criteria increased the number of MIs included[25,26] and thus could mask a decline in incidence. The results of the present study, however, are not contaminated by the change in biomarkers, since all MIs were confirmed with creatine kinase and the MB band of creatine kinase. No concurrent intervention could explain the trends observed during the period of the study. In particular, although automated external defibrillators have been increasingly used in Rochester, Minnesota, the concurrent incidence of out-of-hospital cardiac arrest associated with ventricular fibrillation had begun to level before implementation of the first smoke-free law.[44] Furthermore, the prevalence of smoking declined (Figure 2) and the rate of hypertension and hypercholesterolemia remained essentially constant, whereas the rate of diabetes mellitus and obesity increased (Figure 3).[30]

Finally, one important consideration is the interpretation of the trends reported herein in light of secular trends. Although the incidence of SCD has been declining over the past 30 years, the decrease in the incidence of SCD and MI accelerated during the period of the implementation of these smoke-free workplace laws. There were other tobacco control activities in Minnesota during the time of this study, including a 2001 mass media campaign focused on helping smokers to stop smoking, using a tobacco quitline or clinic services, and a 2004-2007 mass media campaign focused on the hazards of SHS. A 2005 Health Impact fee of \$0.75 per pack of cigarettes was imposed, followed by a \$0.62 per pack increase in federal excise tax. In addition to a decrease in smoking prevalence, from 1999 to 2010, per capita cigarette sales in Minnesota declined by 40% and smoke-free homes increased from 64.5% (1999) to 74.8% (2003) and from 83.2% (2007) to 87.2% (2010).[45]

Although the determinants of the secular trend in the incidence of MI and SCD cannot be determined with certainty in ecologic analysis, particularly with concomitant tobacco control activities, the acceleration of the trends (nonlinearity of the smoothing spline analysis for MI), while all other cardiovascular risk factors (except smoking prevalence) were either stable or increasing, supports the critical role of smoke-free workplace laws in tobacco control efforts. The impact of smoke-free legislation is multifold: reducing the intensity of smoking among smokers, increasing quit rates, reducing smoking startup by teenagers, and reducing exposure to SHS. Thus, the impact can be expected to occur over a period before and after implementation. Previous trends in the incidence of MI in Olmsted County were stable between 1987 and 2006.[26] Herein, we report that MI incidence did not change significantly during the 18 months before and the 18 months after Ordinance 1 (July 2000 to June 2003) was implemented, which is consistent with these previously published data. Thereafter, there was a substantial decline in MI incidence from the 18 months before compared with the 18 months after Ordinance 2 (April 2006 to March 2009) was implemented, pointing to a notable discontinuity of the MI trends, within a period during which no other obvious factor could have plausibly played a large role.

LIMITATIONS AND STRENGTHS

Some limitations should be discussed to aid in the interpretation of the data. We examined temporal trends in MI and SCD concomitant to the implementation of smoke-free laws. This design is consistent with a natural experiment with its well-known limitation regarding causal inference. We recognize that misclassification of deaths occurs in death certificates; however, the study focuses on temporal trends that are unlikely to be confounded by the classification of deaths. We do not have county-specific prevalence data for smoking or other risk factors; however, the trends in Olmsted County are usually consistent with those observed in the Minnesota data (Figures 2 and 3).[30] The population of Olmsted County is primarily white, and further studies are needed in communities of more diverse racial and ethnic composition. Furthermore, we do not have self-reported exposure to SHS or biochemical markers of SHS exposure, although we expect results similar to those of the Scottish study,[14] in which self-reported SHS exposure, confirmed by salivary cotinine, decreased after the smoke-free law was implemented.

Our study has several major strengths. It was conducted under the auspices of the Rochester Epidemiology Project, which has a long track record (>50 years) of robust epidemiologic studies. Our results reflect a complete enumeration of the cases of MI and SCD in a well-defined community; in particular, all MI and SCD cases were validated using rigorous epidemiologic criteria.[25,29] The period during which this study was conducted is longer than that reported in most published studies, thereby affording greater power to detect significant trends.

CLINICAL AND PUBLIC HEALTH IMPLICATIONS

We believe that SHS could be considered a major risk factor for MI, joining family history, hypertension, hyperlipidemia, diabetes mellitus, and low physical activity. Hence, all clinicians should ascertain SHS exposure and promote the elimination of SHS exposure as part of their lifestyle recommendations.[46] Furthermore, all clinicians should be encouraged to become advocates for effective tobacco control policies, such as increased taxes, graphic labeling, smoke-free workplaces, and marketing and advertising restrictions, since smoking and SHS exposure are responsible for 10% of all cardiovascular deaths globally.[47]

Sudden cardiac death represents 60% of all deaths from CHD, and most occur in people without a CHD diagnosis who do not meet high-risk criteria as defined by clinical trials and cohort studies.[48] Thus, the prevention of SCD hinges on public health interventions focused on the primary prevention of CHD or the wider availability of automated external defibrillators and implantable cardioverter-defibrillators. We observed a statistically nonsignificant decline in the incidence of SCD, which may reflect the relatively smaller number of events in the SCD group. These findings suggest that SHS exposure could be a risk factor for SCD. Because this risk factor is highly modifiable, the expansion of smoke-free workplace policies could have a major public health impact by reducing the incidence of SCD.

In conclusion, the implementation of smoke-free workplace ordinances was associated with a substantial decrease in MI, the magnitude of which is not explained by concomitant community interventions or changes in cardiovascular risk factors, with the exception of smoking prevalence. Exposure to SHS should be considered a modifiable risk factor for MI. All people should avoid SHS exposure as much as possible, and those with CHD should have no exposure to SHS.

Accepted for Publication: July 21, 2012.
Published Online: October 29, 2012. doi:10.1001/2013.jamainternmed.46
Correspondence: Richard D. Hurt, MD, Nicotine Dependence Center, Mayo Clinic, 200 First St SW, Colonial 3, Rochester, MN 55905 (rhurt@mayo.edu).
Author Contributions: Dr Hurt had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. *Study concept and design:* Hurt, Weston, Ebbert, Croghan, and Roger. *Acquisition of data:* Weston, McNallan, and Roger. *Analysis and interpretation of data:* Weston, Ebbert, McNallan, Croghan, Schroeder, and Roger. *Drafting of the manuscript:* Hurt, Ebbert, McNallan, and Croghan. *Critical revision of the manuscript for important intellectual content:* Hurt, Weston, Ebbert, Croghan, Schroeder, and Roger. *Statistical analysis:* Weston and Schroeder. *Obtained funding:* Hurt and Roger. *Administrative, technical, and material support:* Hurt, Ebbert, McNallan, and Croghan. *Study supervision:* Roger.
Conflict of Interest Disclosures: None reported.
Funding/Support: This study was supported in part by a grant from ClearWay Minnesota (principal investigator, Dr Hurt); grant R01 HL59205 from the National Heart, Lung, and Blood Institute/National Institutes of Health (principal investigator, Dr Roger); and grant R01 AG034676 from the National Institute on Aging/National Institutes of Health (Rochester Epidemiology Project).
Disclaimer: The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.
Previous Presentation: This study was presented in part at the Annual Meeting of the American Heart Association; November 7, 2011; Orlando, Florida.

REFERENCES

1. Barnoya J, Glantz SA. Cardiovascular effects of secondhand smoke: nearly as large as smoking. *Circulation.* 2005;111(20):2684-2698.
2. Celermajer DS, Adams MR, Clarkson P, et al. Passive smoking and impaired endothelium-dependent arterial dilatation in healthy young adults. *N Engl J Med.* 1996;334(3):150-154.
3. Raitakari OT, Adams MR, McCredie RJ, Griffiths KA, Celermajer DS. Arterial endothelial dysfunction related to passive smoking is potentially reversible in healthy young adults. *Ann Intern Med.* 1999;130(7):578-581.
4. US Department of Health and Human Services. *The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General.* Atlanta, GA: Office on Smoking and Health, National Center for Chronic Disease Prevention and Health Promotion, Coordinating Center for Health Promotion, Centers for Disease Control and Prevention, US Dept of Health and Human Services; 2006.

5. National Cancer Institute, National Institutes of Health. Smoking and tobacco control monographs, monograph 10: health effects of exposure to environmental tobacco smoke. http://cancercontrol.cancer.gov/tcrb/monographs/10/. Accessed June 20, 2011.
6. Fichtenberg CM, Glantz SA. Effect of smoke-free workplaces on smoking behaviour: systematic review. *BMJ*. 2002;325(7357):188. doi:10.1136/bmj.325.7357.188.
7. Sargent RP, Shepard RM, Glantz SA. Reduced incidence of admissions for myocardial infarction associated with public smoking ban: before and after study. *BMJ*. 2004;328(7446):977-980.
8. Bartecchi C, Alsever RN, Nevin-Woods C, et al. Reduction in the incidence of acute myocardial infarction associated with a citywide smoking ordinance. *Circulation*. 2006;114(14):1490-1496.
9. Khuder SA, Milz S, Jordan T, Price J, Silvestri K, Butler P. The impact of a smoking ban on hospital admissions for coronary heart disease. *Prev Med*. 2007;45(1):3-8.
10. Juster HR, Loomis BR, Hinman TM, et al. Declines in hospital admissions for acute myocardial infarction in New York state after implementation of a comprehensive smoking ban. *Am J Public Health*. 2007;97(11):2035-2039.
11. Barone-Adesi F, Vizzini L, Merletti F, Richiardi L. Short-term effects of Italian smoking regulation on rates of hospital admission for acute myocardial infarction. *Eur Heart J*. 2006;27(20):2468-2472.
12. Cesaroni G, Forastiere F, Agabiti N, Valente P, Zuccaro P, Perucci CA. Effect of the Italian smoking ban on population rates of acute coronary events. *Circulation*. 2008;117(9):1183-1188.
13. Cronin E, Kearney P, Sullivan P. Impact of a national smoking ban on the rate of admissions to hospital with acute coronary syndromes [abstract P3506]. *Eur Heart J*. 2007;28(suppl 1):585. doi:10.1093/eurheartj/ehm419.
14. Pell JP, Haw S, Cobbe S, et al. Smoke-free legislation and hospitalizations for acute coronary syndrome. *N Engl J Med*. 2008;359(5):482-491.
15. Mackay DF, Irfan MO, Haw S, Pell JP. Meta-analysis of the effect of comprehensive smoke-free legislation on acute coronary events. *Heart*. 2010;96(19):1525-1530.
16. Committee on Secondhand Smoke Exposure and Acute Coronary Events; Institute of Medicine. *Secondhand Smoke Exposure and Cardiovascular Effects: Making Sense of the Evidence.* Washington, DC: National Academies Press; 2009.
17. US Census Bureau. State & county quickfacts, Olmsted County, Minnesota. http://quickfacts.census.gov/qfd/states/27/27109.html. Accessed June 1, 2012.
18. Melton LJ III. History of the Rochester Epidemiology Project. *Mayo Clin Proc*. 1996;71(3):266-274.
19. St Sauver JL, Grossardt BR, Yawn BP, Melton LJ III, Rocca WA. Use of a medical records linkage system to enumerate a dynamic population over time: the Rochester epidemiology project. *Am J Epidemiol*. 2011;173(9):1059-1068.
20. Roger VL, Weston SA, Redfield MM, et al. Trends in heart failure incidence and survival in a community-based population. *JAMA*. 2004;292(3):344-350.
21. Bursi F, Weston SA, Redfield MM, et al. Systolic and diastolic heart failure in the community. *JAMA*. 2006;296(18):2209-2216.
22. Adabag AS, Therneau TM, Gersh BJ, Weston SA, Roger VL. Sudden death after myocardial infarction. *JAMA*. 2008;300(17):2022-2029.
23. Roger VL, Killian J, Henkel M, et al. Coronary disease surveillance in Olmsted County objectives and methodology. *J Clin Epidemiol*. 2002;55(6):593-601.
24. White AD, Folsom AR, Chambless LE, et al. Community surveillance of coronary heart disease in the Atherosclerosis Risk in Communities (ARIC) study: methods and initial two years' experience. *J Clin Epidemiol*. 1996;49(2):223-233.
25. Roger VL, Killian JM, Weston SA, et al. Redefinition of myocardial infarction: prospective evaluation in the community. *Circulation*. 2006;114(8):790-797.
26. Roger VL, Weston SA, Gerber Y, et al. Trends in incidence, severity, and outcome of hospitalized myocardial infarction. *Circulation*. 2010;121(7):863-869.
27. Kors JA, Crow RS, Hannan PJ, Rautaharju PM, Folsom AR. Comparison of computer-assigned Minnesota Codes with the visual standard method for new coronary heart disease events. *Am J Epidemiol*. 2000;151(8):790-797.
28. Roger VL, Jacobsen SJ, Weston SA, et al. Trends in the incidence and survival of patients with hospitalized myocardial infarction, Olmsted County, Minnesota, 1979 to 1994. *Ann Intern Med*. 2002;136(5):341-348.
29. Goraya TY, Jacobsen SJ, Belau PG, Weston SA, Kottke TE, Roger VL. Validation of death certificate diagnosis of out-of-hospital coronary heart disease deaths in Olmsted County, Minnesota. *Mayo Clin Proc*. 2000;75(7):681-687.
30. Centers for Disease Control and Prevention (CDC). *Behavioral Risk Factor Surveillance System Survey Data.* Atlanta, GA: Centers for Disease Control and Prevention, US Dept of Health and Human Services; 1999-2010. http://www.cdc.gov/brfss/technical_infodata/surveydata.htm. Accessed June 20, 2011.
31. Centers for Disease Control and Prevention. Behavioral Risk Factor Surveillance System: about the BRFSS: turning information into public health. http://www.cdc.gov/brfss/index.htm. Accessed June 4, 2012.
32. Schafer JL, Olsen MK. Multiple imputation for multivariate missing-data problems: a data analyst's perspective. *Multivariate Behav Res*. 1998;33(4):545-571. doi:10.1207/s15327906mbr3304_5.
33. Rubin DB. *Multiple Imputation for Nonresponse in Surveys.* New York, NY: John Wiley & Sons Inc; 1987.
34. Hastie TJ, Tibshirani RJ. *Generalized Additive Models.* London, England: Chapman & Hall; 1990.
35. Stefanadis C, Vlachopoulos C, Tsiamis E, et al. Unfavorable effects of passive smoking on aortic function in men. *Ann Intern Med*. 1998;128(6):426-434.
36. Kallio K, Jokinen E, Hämäläinen M, et al. Decreased aortic elasticity in healthy 11-year-old children exposed to tobacco smoke. *Pediatrics*. 2009;123(2):e267-e273. doi:10.1542/peds.2008-2659.
37. Otsuka R, Watanabe H, Hirata K, et al. Acute effects of passive smoking on the coronary circulation in healthy young adults. *JAMA*. 2001;286(4):436-441.
38. Heiss C, Amabile N, Lee AC, et al. Brief secondhand smoke exposure depresses endothelial progenitor cells activity and endothelial function: sustained vascular injury and blunted nitric oxide production. *J Am Coll Cardiol*. 2008;51(18):1760-1771.
39. Mizoue T, Ueda R, Hino Y, Yoshimura T. Workplace exposure to environmental tobacco smoke and high density lipoprotein cholesterol among nonsmokers. *Am J Epidemiol*. 1999;150(10):1068-1072.
40. Shima M, Adachi M. Effects of environmental tobacco smoke on serum levels of acute phase proteins in schoolchildren. *Prev Med*. 1996;25(5):617-624.
41. Tribble DL, Giuliano LJ, Fortmann SP. Reduced plasma ascorbic acid concentrations in nonsmokers regularly exposed to environmental tobacco smoke. *Am J Clin Nutr*. 1993;58(6):886-890.
42. Henkin L, Zaccaro D, Haffner S, et al. Cigarette smoking, environmental tobacco smoke exposure and insulin sensitivity: the Insulin Resistance Atherosclerosis Study. *Ann Epidemiol*. 1999;9(5):290-296.
43. Venn A, Britton J. Exposure to secondhand smoke and biomarkers of cardiovascular disease risk in never-smoking adults. *Circulation*. 2007;115(8):990-995.
44. Agarwal DA, Hess EP, Atkinson EJ, White RD. Ventricular fibrillation in Rochester, Minnesota: experience over 18 years. *Resuscitation*. 2009;80(11):1253-1258.
45. Centers for Disease Control and Prevention. Decrease in smoking prevalence—Minnesota, 1999–2010. *MMWR Morb Mortal Wkly Rep*. 2011;60(5):138-141 http://www.cdc.gov/mmwr/preview/mmwrhtml/mm6005a2.htm. Accessed June 22, 2012.
46. Chiuve SE, Fung TT, Rexrode KM, et al. Adherence to a low-risk, healthy lifestyle and risk of sudden cardiac death among women. *JAMA*. 2011;306(1):62-69.
47. *Cardiovascular Harms From Tobacco Use and Secondhand Smoke: Global Gaps in Awareness and Implications for Action.* Waterloo, ON, and Geneva, Switzerland: ITC Project, World Health Organization, and World Heart Federation; April 2012.
48. Adabag AS, Luepker RV, Roger VL, Gersh BJ. Sudden cardiac death: epidemiology and risk factors. *Nat Rev Cardiol*. 2010;7(4):216-225.