Journal of the American College of Cardiology
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Published by Elsevier Inc.

Vol. 54, No. 14, 2009
ISSN 0735-1097/09/$36.00
doi:10.1016/j.jacc.2009.08.006

Public Smoking Bans Are Good for the Heart*

Steven A. Schroeder, MD

San Francisco, California

In this time of health care reform debate in the U.S., it is fashionable to focus on what is wrong. Unmentioned is arguably the most important health triumph in the past 50 years: the steep decrease in deaths from cardiovascular disease (1–3). Between 1950 and 2005, the age-adjusted, all-age death rate from heart disease decreased from 587 per 100,000 persons to 211 per 100,000, a spectacular improvement (4). As often happens with such remarkable successes, the credit gets shared. One factor with a major claim to causality is the decrease in smoking prevalence as the result of strong tobacco-control programs (5,6).

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In that regard, Goldman (2) estimates that 25% of the decrease in deaths from cardiovascular disease is from decreased disease incidence and 75% is from reducing deaths in persons with known disease. Of that 75% decrease, approximately one-half is due to risk factor reduction. Obviously, smoking figures in both primary prevention (decreased disease incidence) and secondary prevention (risk factor reduction). Indeed, the decrease in smoking prevalence is another modern health triumph. The prevalence of smoking among men in the U.S. has decreased from 57% in 1955 to 21% in 2007, whereas smoking among women declined from a high of 34% in 1965 to 18% in 2007 (5). The 2007 overall smoking prevalence rate of 19.8% marked a modern low (7).

There are 2 routes whereby tobacco smoke can hurt a patient's heart—by smoking directly or by inhaling someone else's smoke. When smoking rates were high, it was hard to avoid secondhand smoke, and it was also unclear that secondhand smoke was damaging because the assumption was that—as with smoking and lung cancer—sustained exposure was required for harm. But now that smoking is less common and smokers are increasingly marginalized (8,9), evidence has emerged that secondhand smoke exposure is nearly as harmful to the heart as is chronic active smoking. Barnoya and Glantz (10) recently reviewed the many ways in which this harm occurs. Secondhand smoke exposure alters platelet function, causes endothelial dysfunction, increases arterial stiffness, decreases levels of high-density lipoprotein, increases markers of inflammation, increases arterial intima-media thickening, increases infarct size, causes oxidative stress and mitochondrial damage, decreases heart rate variability (thus increasing the risk of malignant arrhythmias), and increases insulin resistance (10). It is hard to imagine substances that would be more cardiotoxic. Furthermore, these adverse effects are observed at very low exposure doses (11).

So, what can cardiologists do to keep their patients from being exposed to tobacco smoke? Obviously, they should counsel patients who smoke to quit by serving as a resource to help that happen; directing them to affiliated cessation systems such as those of Kaiser Permanente, Group Health Cooperative, the Mayo Clinic, or the Veterans Administration system; or referring them to toll-free telephone counseling available through 1-800-QUIT NOW (12,13). However, cardiologists now must also attend to the risks of secondhand smoke exposure.

This issue of the *Journal* contains a meta-analysis by Myers et al. (14) of published studies that examine changes in rates of acute myocardial infarction (AMI) after the institution of second-hand smoke bans in specific locales. The review takes advantage of natural experiments in which public smoking was banned, and rates of AMI were compared before and after that ban, sometimes including comparison sites without a ban. The inclusion criteria restricted the analysis to only 11 peer-reviewed articles representing 10 sites: 5 from the U.S., 1 from Canada, 3 from Italy, and 1 from Scotland. Observation times for measuring the effect ranged from as short as 2 months to as long as 3 years. The populations covered in the studies varied from a high of 19 million people (New York State) to a low of 29,000 (Bowling Green, Ohio). In the meta-analysis, overall results were weighted by the population size included in each study, as well as the duration of observation after the ban was imposed.

The overall mean decrease in AMI incidence after ban imposition was 17%. All studies showed at least 1 subgroup with decreases, and 9 had overall substantial declines, ranging from −9% to −50%. Two sites, both in Italy, had essentially no change. Differences were greater in sites from the U.S. and in those with longer observation periods, with AMI incidence decreasing by 26% with each year of post-ban observation. As shown in the authors' Figure 3, the longer the observation period after the ban, the lower the incidence rates.

Why is this article important? First, by adding published reports outside of the U.S., it builds on evidence of a previous meta-analysis that showed a decrease in post-ban AMI incidence of 19% (15). When the first observation of the effect of smoking bans on community disease incidence in Helena, Montana, was reported, it was unclear whether this

*Editorials published in the *Journal of the American College of Cardiology* reflect the views of the authors and do not necessarily represent the views of *JACC* or the American College of Cardiology.

From the Department of Medicine, University of California San Francisco, San Francisco, California. Dr. Schroeder is supported by grants from the Robert Wood Johnson and American Legacy Foundations.

relationship was an artifact or real, especially because the comparison site had a large (46%) increase in AMI frequency (16). To some, those results seemed too good to be true. By combining all of the published studies on the effects of such bans, Myers et al. (14) add to the evidence that these bans do protect the hearts of those prone to coronary disease. Further, in the 5 reports that used nonban comparison sites, the nonban sites all had much smaller decreases in AMI prevalence. Finally, the findings that the effect is stronger the longer the period of observation and that the largest declines occurred in the U.S.—where smoking prevalence is lower and thus the proportion of nonsmokers who could be exposed to secondhand smoke is greater—support the hypothesis that smoking bans are beneficial. As the authors note, the smaller effect sizes all occurred in studies outside the U.S. plus the New York State study. All these sites had short observation periods.

What are the limitations of this study? Publication bias is always a potential problem, although the authors were unable to surface any reports that went unpublished. More problematic is the reality that in the >3,000 communities and 33 states that restrict public smoking in the U.S. (17), most have not tracked changes in AMI incidence. The same holds for other countries. Another potential limitation is the vigor with which such bans are enforced. Given the lower smoking prevalence rates in the U.S. and the stigma attached to smoking in that country, it is likely that such bans are more easily accepted there and better enforced than in Italy and Scotland, where the published reports showed either a lower (Italy) or average (Scotland) effect of smoking bans. A related question is whether these decreases merely reflect the secular trends of decreasing AMI frequency. However, the rate of decrease is much greater than those secular trends, and those studies with comparison sites all showed lower rates of decline, or in the case of Helena, a 46% increase.

Could there have been a coding effect, whereby the diagnosis of AMI somehow changed in communities with bans? That seems unlikely. More troubling is the fact that the effects are much smaller in the reports from large population sites, with the exception of Scotland. The authors tested whether geographic region (U.S. vs. non-U.S.), population size, or length of post-ban observation period affected the incidence reduction rate and concluded that region and observation period did but size did not. How robust that conclusion is remains to be seen. It is likely that the larger the population encompassed by a ban, the less uniform enforcement of the ban might be.

The take-away messages for cardiologists are clear. A 17% risk reduction for AMI is not trivial. It is prudent to assume that exposure to secondhand smoke is almost as dangerous to persons with diagnosed or latent coronary disease as active smoking (10). Therefore, cardiologists should expand their clinical repertoire to include screening and counseling for secondhand smoke exposure, just as they screen for lipid disorders. In their roles as health advocates, they should also support bans on public smoking, as well as other tobacco control measures such as tax increases on cigarettes, countermarketing campaigns, and expanded cessation services such as telephone quitlines (6,18,19). These initiatives lower the probability of young people initiating smoking, increase the rate at which smokers quit, and lower the frequency of smoking among those not yet willing or able to quit. Cardiologists not only have much to celebrate about the spectacular decreases in cardiovascular disease, they also have the opportunity to do much more. Further decreased exposure to tobacco smoke, such as occurs with public smoking bans, is a keystone to such progress.

Reprint requests and correspondence: Dr. Steven A. Schroeder, Department of Medicine, University of California San Francisco, 3333 California Street, San Francisco, California 94118. E-mail: schroeder@medicine.ucsf.edu.

REFERENCES

1. Ford ES, Ajani UA, Croft JB, et al. Explaining the decrease in U.S. deaths from coronary disease, 1980–2000. N Engl J Med 2007;356:2238–98.
2. Goldman L. The decline of coronary heart disease: determining the paternity of success. Am J Med 2004;117:274–6.
3. Rodriquez T, Malvezzi M, Chatenoud L, et al. Trends in mortality from coronary heart and cerebrovascular diseases in the Americas: 1970–2000. Heart 2006;92:453–60.
4. Centers for Disease Control and Prevention. Health United States, 2008. Table 35. Death rates for diseases of heart, by sex, race, Hispanic origin, and age: United States, selected years 1950–2005. Available at: http://www.cdc.gov/nchs/data/hus/hus08.pdf#035. Accessed August 6, 2009.
5. Schroeder SA. Shattuck Lecture: we can do better—improving the health of the American people. N Engl J Med 2007;357:1221–8.
6. Schroeder SA. Tobacco control in the wake of the 1994 Master settlement agreement. N Engl J Med 2004;350:293–301.
7. Centers for Disease Control and Prevention. Cigarette smoking among adults—United States, 2007. MMWR Morb Mortal Wkly Rep 2008;57:1221–6.
8. Christakis NA, Fowler JH. The collective dynamics of smoking in a large social network. N Engl J Med 2008;358:2249–58.
9. Schroeder SA. Stranded in the periphery: the increasing marginalization of smokers. N Engl J Med 2008;358:2284–5.
10. Barnoya J, Glantz SA. Cardiovascular effects of secondhand smoke: nearly as large as smoking. Circulation 2005;111:2684–98.
11. Benowitz NL. Cigarette smoking and cardiovascular disease: pathophysiology and implications for treatment. Prog Cardiovascular Dis 2003;46:91–111.
12. Fiore MC, Jaen CR, Baker TB, et al. Treating Tobacco Use and Dependency: 2008 Update Practice Guideline. Rockville, MD: Public Health Service; 2008.
13. Schroeder SA. What to do with a patient who smokes. JAMA 2005;294:482–7.
14. Myers DG, Neuberger JS, He J. Cardiovascular effect of bans on smoking in public places: A systematic review and meta-analysis. J Am Coll Cardiol 2009;54:1249–55.
15. Glantz SA. Meta-analysis of the effects of smoke-free laws on acute myocardial infarction: an update. Prev Med 2008;47:452–543.
16. Sargent RP, Shepard RM, Glantz SA. Reduced incidence of admissions for myocardial infarction associated with public smoking ban: before and after study. BMJ 2004;328:977–80.
17. Americans for Nonsmokers Rights Foundation. Available at: http://www.no-smoke.org. Accessed August 27, 2009.
18. Warner KE. Tobacco policy research: insights and contributions to public health policy. In: Warner KE, editor. Tobacco Control Policy. San Francisco, CA: Jossey-Bass; 2007:3–86.
19. Barnoya J, Bialous SA, Glantz SA. Effective interventions to reduce smoking-induced heart disease around the world: time to act. Circulation 2005;112:456–8.

Key Words: smoking ▪ acute myocardial infarction ▪ public health ▪ secondhand smoke ▪ incidence.

Journal of the American College of Cardiology
© 2009 by the American College of Cardiology Foundation
Published by Elsevier Inc.

Vol. 54, No. 14, 2009
ISSN 0735-1097/09/$36.00
doi:10.1016/j.jacc.2009.07.022

QUARTERLY FOCUS ISSUE: PREVENTION/OUTCOMES

Cardiovascular Effect of Bans on Smoking in Public Places

A Systematic Review and Meta-Analysis

David G. Meyers, MD, MPH,*† John S. Neuberger, DrPH, MPH, MBA,† Jianghua He, PhD‡

Kansas City, Kansas

Objectives A systematic review and a meta-analysis were performed to determine the association between public smoking bans and risk for hospital admission for acute myocardial infarction (AMI).

Background Secondhand smoke (SHS) is associated with a 30% increase in risk of AMI, which might be reduced by prohibiting smoking in work and public places.

Methods PubMed, EMBASE, and Google Scholar databases plus bibliographies of relevant studies and reviews were searched for peer-reviewed original articles published from January 1, 2004, through April 30, 2009, using the search terms "smoking ban" and "heart" or "myocardial infarct." Investigators supplied additional data. All published peer-reviewed original studies identified were included. Incidence rates of AMI per 100,000 person-years before and after implementation of the smoking bans and incidence rate ratios (IRRs) with 95% confidence intervals (CIs) were calculated. Random effects meta-analyses estimated the overall effect of the smoking bans. Funnel plot and meta-regression assessed heterogeneity among studies.

Results Using 11 reports from 10 study locations, AMI risk decreased by 17% overall (IRR: 0.83, 95% CI: 0.75 to 0.92), with the greatest effect among younger individuals and nonsmokers. The IRR incrementally decreased 26% for each year of observation after ban implementation.

Conclusions Smoking bans in public places and workplaces are significantly associated with a reduction in AMI incidence, particularly if enforced over several years. (J Am Coll Cardiol 2009;54:1249–55) © 2009 by the American College of Cardiology Foundation

Secondhand smoke (SHS) increases the risk of acute myocardial infarction (AMI) by 25% to 31% (1–5). In countries where smoking prevalence is high, for example, Britain 50% (6), Europe 62% (7), and Greece 156% (8), versus 22% in the U.S. (2,9), AMI in nonsmokers is particularly increased. The dose-response relationship between SHS and AMI is nonlinear, increasing rapidly even at low concentrations (10–12). Bans on smoking in public places and workplaces have been instituted in several countries, 32 U.S. states, and many cities and counties in the U.S. We performed a systematic literature review and meta-analysis to estimate the overall effect of public (workplace and public place) smoking bans on the risk of AMI in the general population.

Methods

We searched PubMed, EMBASE, and Google Scholar from January 1, 2004, through April 30, 2009, using the search terms "smoking ban" and "heart" or "myocardial infarct" and reviewed pertinent bibliographies. One unpublished abstract and 1 nonpeer-reviewed report were excluded, leaving 11 peer-reviewed published studies concerning 10 geographic locations. Duplicate data abstracting was performed by 2 authors (D.G.M. and J.S.N.). Only AMI cases were included (some investigators supplied additional data), except where the case definition was acute coronary syndrome (ACS), which required an elevated serum troponin.

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To avoid duplicate cases (Piedmont and Latium [Rome] were separately reported), only the regions of Fruili Venezia Giulia (Trieste) and Campania (Naples) were analyzed from the Italian study of 4 regions. Results were converted to incidence rates (new cases/100,000 person-years) using the most recent official census and including all age groups.

Meta-analysis used the random-effects model in the *metan* statistical package in STATA version 10 (Stata Corp., College Station, Texas) (13) because heterogeneity was significant in the fixed effects model ($p < 0.001$). Unlike previously pub-

From the Departments of *Internal Medicine, †Preventive Medicine and Public Health, and ‡Biostatistics, University of Kansas School of Medicine, Kansas City, Kansas.

Manuscript received May 13, 2009; revised manuscript received July 20, 2009, accepted July 28, 2009.

Abbreviations and Acronyms

ACS = acute coronary syndrome

AMI = acute myocardial infarction

CI = confidence interval

ICD = International Classification of Diseases

IRR = incidence rate ratio

SHS = secondhand smoke

lished meta-analyses, which used average yearly incidence rates (14,15), we weighted studies by person-years, thus considering both population size and duration of observation, and assumed that the incidence of AMI satisfied a Poisson process (16). Because the funnel plot showed systematic heterogeneity among the study results, we performed a meta-regression analysis using the *metareg* package of STATA 10 to examine whether the estimate of incidence rate ratio (IRR) depends on such factors as post-ban duration, population size, or region (U.S. or non-U.S.).

Results of the Systematic Review

Abstractor agreement was 100%. Results are summarized in Table 1. All studies reported decreases in incidence (at least in a subgroup), with the largest decreases observed in the U.S. Table 2 lists important study parameters based on the Newcastle-Ottawa scale (17). No study had all parameters, although the Scotland study had nearly all. All studies excluded transients and matched observation periods by season.

Helena, Montana. This community of 47,154 persons passed a ban on public smoking (all but 2 businesses complied) in June 2002, which was judicially suspended in December 2002 (18). Investigators screened cases of AMI by International Classification of Diseases (ICD) 9th Edition codes (410xx) and confirmed cases by chart review (criteria not published). Incidence decreased from 170 to 102 cases/100,000 person-years, then returned to baseline, a 40% temporary decline. In the surrounding area, incidence increased from 118 to 172 cases/100,000 person-years, an increase of 46%. This was the first study of a public smoking ban and the only study to include data from after a ban was suspended.

Pueblo, Colorado. Pueblo (population 103,648) banned smoking in bars, restaurants, bowling alleys, and business establishments, whereas Colorado Springs (population 370,448), 45 miles distant in El Paso County, did not (19). Cases included a primary diagnosis of AMI by ICD-9 code 410xx (with no confirmation by biomarkers) for 18 months before and the initial 18 months during ban enforcement. During the ban, AMI incidence in the city of Pueblo decreased by 27% (257 to 187 cases/100,000 person-years, IRR: 0.73, 95% confidence interval [CI]: 0.64 to 0.82). The surrounding Pueblo County (noncity population 44,103) decreased 15% (135 to 115 cases/100,000 person-years, IRR: 0.85, 95% CI: 0.56 to 1.14). Adjacent El Paso County (population 550,478) experienced a 4% decrease (157 to 150 cases/100,000 person-years, IRR: 0.96, 95% CI: 0.87 to 1.04). An additional 18 months of observation noted a further 19% reduction in Pueblo City (152 cases/100,000 person-years) for an overall 3-year reduction of 41% (IRR: 0.59, 95% CI: 0.49 to 0.70) with no reduction in either Pueblo County (IRR: 1.03, 95% CI: 0.68 to 1.39) or adjacent El Paso County (IRR: 0.95, 95% CI: 0.87 to 1.03) (20). This study used well-separated communities and shared the longest observation period.

New York State. Many communities in New York State (population 18,976,457) had banned public smoking, and the state had increased taxation on tobacco before the July 2003 implementation of a statewide ban on work and public smoking (bars, restaurants, and hospitality venues) (21). A statewide database (252 hospitals) was searched for the primary diagnosis of AMI cases (ICD-9 codes 410.0 to 410.99 with no biomarker confirmation) for 1995 through 2004. The AMI incidence decreased 8%, from 483 (46,332 cases) to 445 cases/100,000 person-years (45,412 cases). Compliance with the ban was 93%. Had there been no local laws, the comprehensive state law would have been associated with a 19% decline in admissions. From 2002 to 2004, New York City smoking prevalence decreased from 21.5%

Table 1 Summary Results of Smoking Bans

Ban Location	Population Exposed to Ban	Post-Ban Observation Period (Yrs)	Pre-Ban Rate*	Post-Ban Rate*	Incidence Rate Change in Ban Area	Incidence Rate Change in Non-Ban Area
U.S.						
Helena	68,140	0.5	170	102	−40%	+46%
Pueblo	698,229	3.0	257	152	−41%	−5%
New York	18,976,457	1.0	483	445	−8%	None
Indiana	239,332	1.5	14	7	−50%	−20%
Ohio	29,636	3.0	277	223	−20%	−5%
Canada						
Saskatoon	202,340	1.0	176	152	−13%	None
Europe						
Piedmont	~4,300,000	0.5	200	204	+2%†	None
Rome	2,663,182	1.0	252	253	0%‡	None
Italy	7,033,451	0.2	159	149	−6%	None
Scotland§	~3,000,000	0.8	129	107	−17%	−4%

*Cases per 100,000 person-years. †The acute myocardial infarction incidence decreased 9.8% in those age <65 years and increased 6.2% in those age >65 years. ‡The acute myocardial infarction incidence decreased 11% in those age <65 years and decreased 8% in those age 75 to 84 years, particularly among men. §The end point was acute coronary syndrome.

Table 2 Important Study Parameters

Location	Prospective Design	Population >1 Million	Follow-Up >1 Yr	AMI Primary Diagnosis	Case Smoking Status Ascertained	SHS Measured	Compliance With the Ban	Smoking Prevalence	Contemporaneous Controls
U.S.									
Helena							×		×
Pueblo			×	×					×
New York		×	×	×		×	×	×	
Indiana			×		×				×
Ohio			×	×					×
Canada									
Saskatoon			×	×			×	×	
Europe									
Piedmont		×		×		×	×	×	
Rome		×	×			×		×	
Italy		×		×					
Scotland	×	×		×	×	×	×		×

AMI = acute myocardial infarction; SHS = secondhand smoke.

to 18.5% (22). Exposure, as measured by salivary cotinine, decreased 47% (23). This is the largest population studied.

Monroe County, Indiana. Monroe County (population 120,563) banned smoking in all restaurants, retail outlets, and workplaces in 2003, but excluded bars until 2005 (24). Delaware County (population 118,769), 90 miles distant, did not restrict smoking. Cases during 18 months before and during the ban were ascertained by ICD-9 codes for primary and secondary diagnosis of AMI (410.0x to 410.9x) and confirmed with biomarkers. Patients with a "...past cardiac procedure..." or who had "...comorbidity such as hypertension and high cholesterol that could have precipitated acute myocardial infarction..." were excluded (24). Case smoking status was ascertained. The AMI incidence decreased 50%, from 14 to 7 cases/100,000 person-years. The Delaware County rate of 15 cases/100,000 person-years declined by 20% to 12 cases/100,000 person-years. The reduction was primarily among nonsmokers, whose admissions decreased 70% (−12 cases, 95% CI: −21.2 to −2.8). The exclusion of subjects with prior cardiac procedures or risk factors likely reduced AMI incidence.

Bowling Green, Ohio. Investigators compared admissions for coronary heart disease (ICD-9 codes 410 to 414 and 428 including angina, heart failure, atherosclerosis, and AMI with no biomarker confirmation) in Bowling Green, Ohio (population 29,636), which banned smoking in workplaces and public places except bars, to admissions in Kent, Ohio (population 27,906), 150 miles distant with no ban (25). Hospital discharge data for 3 years before and after the ban (6 months immediately after ban implementation were excluded) for cases in the 2 cities were obtained from a state database. The AMI incidence in Bowling Green decreased by 19%, from 277 to 223 cases/100,000 person-years ($p = 0.015$). Incidence in Kent did not significantly change (440 cases/100,000 person-years in 1999 to 2002 and 417 cases/100,000 person-years in 2003 to 2005, $p = 0.22$). This study shared the longest observation period. Only AMI data supplied by the investigators were used.

Saskatoon, Canada. Saskatoon (population 202,340) implemented a smoking ban in all enclosed public places and outdoor seating areas in July 2004. The AMI cases (ICD-9 410.00 to 410.92 and ICD-10 121.1 to 121.9) in the database of the Strategic Health Information Planning Service were identified (no biomarker confirmation) for 1 year during the ban and for the previous 4 years (26). Compliance was 99%. Incidence of AMI decreased 14%, from 176 (95% CI: 165 to 187 cases/100,000 person-years) to 152 cases/100,000 person-years (95% CI: 135 to 169 cases/100,000 person-years). Smoking prevalence was 24% in 2003 and 18% in 2005.

Piedmont, Italy. Italy banned smoking in cafes, restaurants, bars, and discos in 2005. Investigators used ICD-9 codes (410.xx) in the Hospital Discharge Registry of the Piedmont region (population 4,300,000) to identify AMI cases (no confirmation) before and during 5 months after ban initiation (27). Before the ban, an average of 3,581 AMIs were reported between February and June (200 cases/100,000 person-years). During the comparable 5 months of ban enforcement, 3,655 cases were reported (204 cases/100,000 person-years). A decrease was observed only among women <60 years of age (women IRR: 0.75, 95% CI: 0.58 to 0.96; men IRR: 0.91, 95% CI: 0.83 to 1.01). Cases in persons >60 years of age were unchanged (IRR: 1.05, 95% CI: 1.00 to 1.11). The ban was almost universally observed (28), nicotine vapor in public places decreased 90% to 95% (29), cigarette sales declined 8.9% (28), and cigarette consumption decreased 7.6% (28). The investigators suggested a greater effect of the ban on younger people and a lower attributable risk of AMI from smoking among older people (27). This study provided age- and sex-specific data.

Rome, Italy. Investigators in Rome (population 2,663,182), using 2 databases, identified all hospital admissions with a primary or secondary diagnosis of ACS, including AMI (ICD-9-CM code 410.xx without biomarker confirmation)

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or other forms of ischemic heart disease (ICD-9-CM code 411) and all out-of-hospital deaths caused by ischemic heart disease, among persons >34 years of age before and after enactment of the smoking ban (30). Pre-ban incidence was 252 cases/100,000 person-years. During the ban, incidence was 253 cases/100,000 person-years. Incidence decreased significantly in 35- to 64-year-old men (IRR: 0.89, 95% CI: 0.85 to 0.93) and in 65- to 74-year-old men (IRR: 0.92, 95% CI: 0.88 to 0.97), but not in 75- to 84-year-old men (IRR: 1.02, 95% CI: 0.98 to 1.07). Among women (30% of AMI cases), the decrease was confined to the young (IRR: 0.90, 95% CI: 0.81 to 1.00 in 35- to 64-year-old women and IRR: 0.95, 95% CI: 0.88 to 1.04 in 65- to 74-year-old women). Decreases occurred in indoor particle and urinary cotinine concentrations and per capita cigarette sales, whereas nicotine replacement product sales increased (31,32). This study adjusted for several confounders, including weather and temporal trends. The meta-analysis used AMI data supplied by the investigators.

Four regions, Italy. The Italian Health Ministry used regional databases to identify cases (age 40 to 64 years) discharged with AMI (ICD-9 code 410.xx, no biomarker confirmation) in 4 regions of Italy with 16,995,734 people (Piedmont, Friuli Venezia Giulia, Latium, and Campania), representing 28% of the Italian population, during corresponding 2-month periods before and during the national ban (33). The AMI incidence decreased 6%, from 159 to 149 cases/100,000 person-years. The preceding years had increasing rates. Reduction in incidence was limited to 45- to 54-year-old men, an 8% decrease. No sex or age group experienced an increase in AMIs. This study had the shortest observation period. The meta-analysis excluded the regions of Piedmont and Latium (Rome), reported separately (27,30).

Scotland. Since March 2006, smoking has been prohibited in all enclosed public places in Scotland (population 5.1 million). Investigators identified all patients admitted to 9 hospitals (catchment area included approximately 3 million persons, accounting for 64% of the country's hospital admissions) for a diagnosis of ACS from June 2005 through March 2006 and for the corresponding 10 months after ban institution (34). An ACS was defined as chest pain with "a detectable level of cardiac troponin," routinely measured in all cases of chest pain. Case smoking status was ascertained by self-report and serum cotinine levels, which allowed estimation of passive exposure to SHS. Results were compared with admissions in England, which did not have a ban. In the 10 months before the ban, 3,235 patients were admitted. After the ban, 2,684 patients were admitted, a 17% decrease (95% CI: 16% to 18%). England experienced a 4% decrease. In Scotland, admissions decreased by 14% in smokers, 19% in former smokers, and 21% in never smokers. The investigators estimated that 67% of the admissions prevented involved nonsmokers. Nonsmokers reporting exposure to SHS decreased from 43% to 22%. Serum cotinine levels decreased by 18%. Compliance was 98%: SHS in bars decreased by 86% within 2 weeks of ban implementation (35). This is the only prospective study and used both direct and indirect measurement of exposure.

Results of the Meta-Analysis

As shown in Figure 1, the overall IRR comparing AMI before and after smoking bans is 0.83 (95% CI: 0.75 to 0.92), indicating that smoking bans on average reduced AMI incidence by 17%.

To further examine the adequacy of a random-effect meta-analysis, a funnel plot of the estimated IRR versus the standard error of natural log of IRR was obtained (Fig. 2).

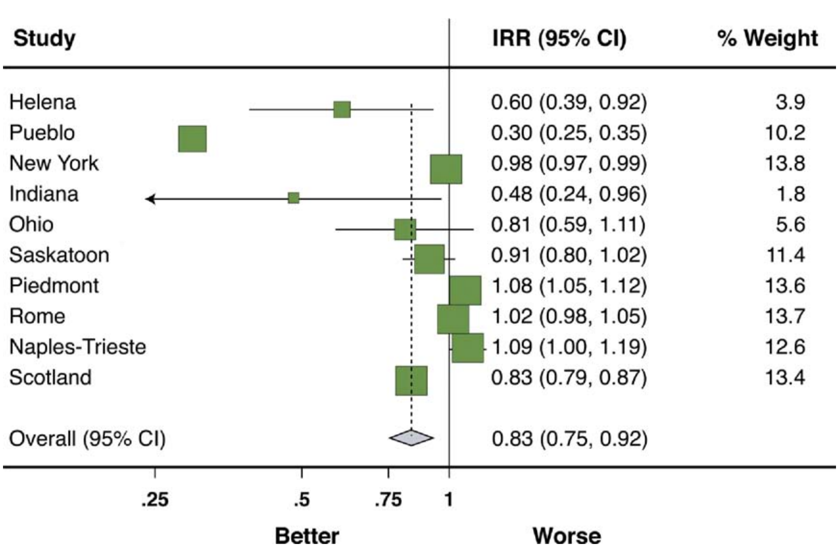

Figure 1 Effects of Community Smoking Bans on Incident Acute Myocardial Infarction (Person-Year Approach)

Meta-analysis results for 11 studies in 10 geographic locations. CI = confidence interval; IRR = incidence rate ratio.

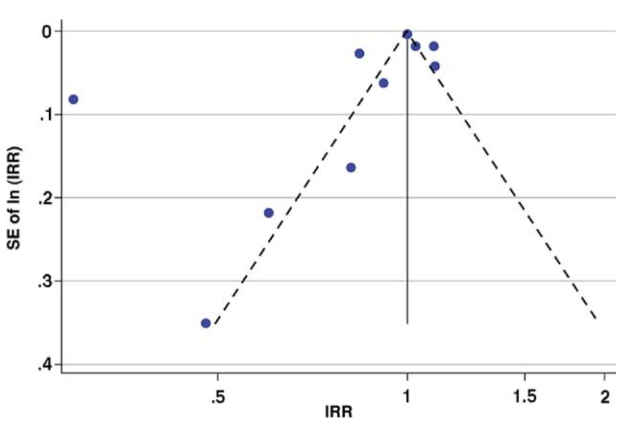

Figure 2 **Funnel Plot of Estimated IRRs**

A funnel plot with all points evenly distributed on both sides of the **solid vertical line** indicates no publication bias. In this plot there are more points to the **left of the vertical line**, suggesting heterogeneity caused by either variation in length of observation or publication bias. IRR = incidence rate ratio.

The funnel plot shows asymmetry, indicating either publication bias or heterogeneity that cannot be explained by a random-effect meta-analysis. Contact with other investigators has not indicated the presence of unpublished peer-reviewed studies. Meta-regression modeling showed that heterogeneity is at least partially explained by variation in population size and observation duration.

We notice that studies with smaller effect sizes (IRR close to 1.0) include all non-U.S. studies and the New York State study, all of which have large populations and short post-ban durations (≤1 year). Meta-regression analysis was used to examine whether region (U.S. or non-U.S.), population size, and post-ban duration affect IRR. When tested separately the effect of population size is not significant ($p = 0.19$), whereas both post-ban duration ($p = 0.002$) and region (U.S. vs. non-U.S., $p = 0.03$) are significant. Post-ban duration becomes borderline significant ($p = 0.096$) and region becomes not significant ($p = 0.399$) when they are included in the same model. Because the effect of bans may not be maximal in ≤1 year, it is likely that post-ban observation time is inversely related to IRR (effect size). This relationship is shown in Figure 3, in which the trend line illustrates the overall relationship between the estimated IRR and post-ban duration estimated by meta-regression analysis. The size of each bubble is proportional to the weight of the study (the inverse of the standard deviation of the natural log of IRR). The coefficient of post-ban duration in the meta-regression model is −0.30 (95% CI: −0.49 to 0.11), meaning that the IRR decreases by 26% (95% CI: 10% to 39%) for each year of post-ban observation (e.g., IRR: 0.74 after 1 year, then 0.55, then 0.41 compared with pre-ban).

Discussion

This meta-analysis of 11 studies in 10 locations suggests that community smoking bans are associated with an overall 17% reduction in risk of AMI. This is consistent with reductions of 5% to 19% predicted by modeling (36,37). A meta-analysis of the 4 earliest studies reported a relative risk (RR) of 0.73 (14). Subsequent meta-analysis by the same author (15) included 8 studies (including an unpublished report noting an 11% decrease in ACS (38), which was not used in the current analysis) and yielded a pooled risk reduction estimate of 19% (95% CI: 14% to 24%). The large studies from the Italian Piedmont (27), 4 regions (33), and Scotland (34), which observed smaller effects than the earlier American studies, had not yet been published.

In New York, local laws were enacted beginning in 1995, before the implementation of a statewide law on July 24, 2003. Our analysis may capture only the additive effect of this state law, in addition to local laws, rather than the cumulative effect of local and state laws together. Because 1995 is the year within 1995 to 2004 that is affected the least by local laws, we reanalyzed the data using only 1995 as the pre-ban period, versus 2004 as the post-ban period. The estimated IRR for New York State is only slightly smaller than the original estimate based on all data within 1995 to 2004 (0.97 vs. 0.98).

The evidence for an association between smoking bans and reduced AMI incidence is strengthened by beneficial changes in several intermediate factors: high levels of compliance with bans (28,35,39), decreased smoking prevalence and sales of tobacco (20,28,32), improved air quality (29,31,35), and reduced environmental exposure to tobacco smoke (28–30,35,40). Noteworthy is the Helena study (18), which documented a return to the pre-ban AMI incidence rate in the 6 months after suspension of the ban.

Two studies determined the smoking status of AMI cases (24,34). The Indiana study (24) observed a 70% decline in nonsmokers compared with no change among smokers,

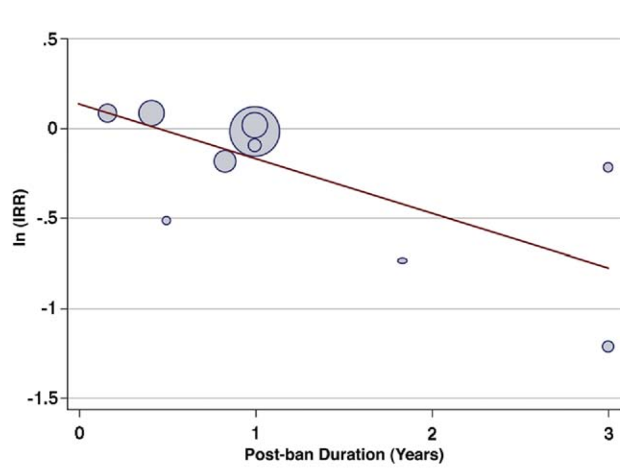

Figure 3 **Bubble Plot of Estimated Effects of Smoking Bans (Log of IRR) and the Post-Ban Duration (Years)**

The **size of the bubbles** indicates the weight of each study in the meta-analysis. The **trend line** indicates the degree to which the incidence rate ratio (IRR) decreases as the duration of the post-ban period increases.

whereas the Scottish study (34) noted a 19% to 21% decrease in admissions among nonsmokers and a 14% decrease among smokers.

The Italian 4 regions study (33) observed a reduced risk in men only, whereas the Piedmont region (27) and Scotland (34) studies experienced a larger decrease in women. A greater benefit in men might be attributed to their higher prevalence of smoking, allowing for a larger percentage to quit. In Italy, men were more likely than women to quit smoking after the ban (41). Because the workforce has proportionately more men, workplace bans might have a greater effect on SHS exposure among men. Yet, a greater post-ban reduction in serum cotinine levels in nonsmoking women (47%) compared with men (37%) in Scotland (34) suggests that exposure decreased more among women. The relative risk associated with smoking is greater in women than men and has a steeper dose response (42).

A greater effect in younger individuals was noted in the studies that evaluated age-specific incidence (27,30,33,34). Smoking bans encourage cessation particularly among young smokers (43–45). Older individuals might benefit less from smoking restrictions in the workplace, bars, and discos. As risk of AMI associated with smoking decreases with age, the largest effect of eliminating SHS would occur in younger persons.

The smaller effect size in the 5 non-U.S. studies (RR: 0.95 vs. 0.75) may be partially explained by their shorter post-ban observation time. Additional causes might include differences in AMI case definition (although a uniform definition has been suggested [46]), lifestyle and diet, smoking prevalence (U.S. 22.1%, Canada 21.3%, Europe 30.0%), and compliance. Yet, studies from Italy (47) and Ireland (48) indicate substantial reductions in SHS exposure.

The beneficial effect of smoking bans seems to be rapid, with declines in AMI incidence within 3 months (33). Among smokers, incident ACS is reduced within days after smoking cessation (11,41). In nonsmokers, even brief exposure to SHS has been associated with changes in platelet activation (49–51), vascular elasticity (52), endothelial function (53), heart rate variability (54), and lipid metabolism (55,56), supporting the biological plausibility of smoking bans' effect on AMI.

The 11 studies are all ecological in design. Such a design is primarily hypothesis generating. Many of these studies differ in case definition, SHS exposure information, smoking prevalence data, and case confirmation. Many were of short-term duration.

The advantage of person-year analysis in the current study is that analysis is based on the actual total counts of AMI events rather than the estimated average rate. Thus, the total variation of incidence over time is considered. One population estimate for each region was used regardless of the pre- and post-ban observation times. This may explain why our estimates of IRR are sometimes different from the RR based on average age-adjusted rate as published in the original reports. With the limited number and durations of studies, we are unable to ascertain whether the IRR changes associated with post-ban observation time follow a nonlinear pattern, as suggested by the dose-response relationship of SHS to AMI (10–12).

A non–peer-reviewed working paper (57) used U.S. databases and smoking ban implementation dates to model the effect of workplace and public place smoking bans on mortality and AMI admissions. The investigators found no significant effect of these bans in any outcome for any age group. The investigators suggest that the observed benefits in other studies are attributable to the wide variation in incidence related to small samples.

Although additional reports can be expected (e.g., Ireland and France), the 11 reports included represent the current findings. These studies include nearly 24 million people, observed 215,524 cardiac events, and suggest that community smoking bans are associated with a 17% reduction in AMI incidence. If this association represents a cause-and-effect relationship, and assuming approximately 920,000 incident AMIs each year in the U.S., a nationwide ban on public smoking might ultimately prevent as many as 156,400 new AMIs yearly.

Reprint requests and correspondence: Dr. David G. Meyers, Division of Cardiovascular Diseases, Kansas University School of Medicine, 3901 Rainbow Boulevard, Kansas City, Kansas 66160. E-mail: dmeyers@kumc.edu.

REFERENCES

1. Husten CG, Thorne SL. Tobacco: health effects and control. In: Wallace RB, Kohatsu N, editors. Public Health and Preventive Medicine. 15th edition. New York, NY: McGraw Hill, 2008:953–98.
2. U.S. Department of Health and Human Services. Introduction, summary, and conclusions. In: The Health Consequences of Involuntary Exposure to Tobacco Smoke. A Report of the Surgeon General. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, Office of Smoking and Health, 2006:1–15.
3. Law MR, Morris JK, Wald NJ. Environmental tobacco smoke exposure and ischemic heart disease: an evaluation of the evidence. BMJ 1997;315:973–80.
4. He J, Vupputuri S, Allen K, Prerost MR, Hughes J. Passive smoking and the risk of coronary heart disease—a meta-analysis of epidemiologic studies. N Engl J Med 1999;340:920–6.
5. Barnoya J, Glantz SA. Cardiovascular effects of secondhand smoke: nearly as large as smoking. Circulation 2005;111:2684–98.
6. Whincup PH, Gilg JA, Emberson JR, et al. Passive smoking and risk of coronary heart disease and stroke: prospective study with cotinine measurement. BMJ 2004;329:200–5.
7. Teo KK, Ounpuu S, Hawken S, et al., for the INTERHEART Investigators. Tobacco use and risk of myocardial infarction in 52 countries in the INTERHEART study: a case-control study. Lancet 2006;368:647–58.
8. Pitsavos C, Panagiotakos DB, Chrysohoou C, et al. Association between exposure to environmental tobacco smoke and the development of acute coronary syndromes: the CARDIO2000 case-control study. Tob Control 2002;11:220–5.
9. Maurice E, Thorne S, Ajani U, Malarcher A, Merritt R, Husten C. State-specific prevalence of current cigarette smoking among adults and secondhand smoke rules and policies in homes and workplaces—U.S., 2005. MMWR Morb Mortal Wkly Rep 2006;55:1148–51.
10. Howard G, Thun MJ. Why is environmental tobacco smoke more strongly associated with coronary artery disease than expected? A

review of potential biases and experimental data. Environ Health Perspect 1999;107 Suppl 6:853–8.
11. Law MR, Wald NJ. Environmental tobacco smoke and ischemic heart disease. Prog Cardiovasc Dis 2003;46:31–8.
12. Benowitz NL. Cigarette smoking and cardiovascular disease: pathophysiology and implications for treatment. Prog Cardiovasc Dis 2003;46:91–111.
13. Sterne JAC, Bradburn MJ, Egger M. Meta-analysis in STATA. In: Systematic Reviews in Health Care. Egger M, Smith GD, Altman DG, editors. London: BMJ Books, 2001:347–69.
14. Dinno A, Glantz S. Clean indoor air immediately reduces heart attacks. Prev Med 2007;45:9–11.
15. Glantz SA. Meta-analysis of the effects of smoke-free laws on acute myocardial infarction: an update. Prev Med 2008;47:452–543.
16. Rothman K, Greenland S. Modern Epidemiology. 2nd edition. Philadelphia, PA: Lippincott-Raven, 1998:237–9.
17. Newcastle-Ottawa scale for assessing the quality of non-randomized studies in meta-analysis. Ottawa Health Research Institute. Available at: http://www.ohri.ca/programs/clinical_epidemiology/oxford.htm. Accessed February 18, 2009.
18. Sargent RP, Shepard RM, Glantz SA. Reduced incidence of admissions for myocardial infarction associated with public smoking ban: before and after study. BMJ 2004;328:977–80.
19. 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;114:1490–6.
20. Centers for Disease Control and Prevention. Reduced hospitalizations for acute myocardial infarction after implementation of a smoke-free ordinance. City of Pueblo, Colorado, 2002–2006. MMWR Morb Mortal Wkly Rep 2009;57:1373–7.
21. 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;97:2035–9.
22. Centers for Disease Control and Prevention. Decline in smoking prevalence—New York City, 2002–2006. MMWR Morb Mortal Wkly Rep 2007;56:604–8.
23. Centers for Disease Control and Prevention. Reduced secondhand smoke exposure after implementation of a comprehensive statewide smoking ban—New York, June 26, 2003–June 30, 2004. MMWR Morb Mortal Wkly Rep 2007;56:705–8.
24. Seo, DC, Torabi MR. Reduced admissions for acute myocardial infarction associated with a public smoking ban: matched controlled study. J Drug Educ 2007;37:217–26.
25. Khuder SA, Milz S, Jordan T, Price J, Sivestri K, Butler P. The impact of a smoking ban on hospital admissions for coronary artery disease. Prev Med 2007;45:3–8.
26. Lemstra M, Neudorf C, Oponda J. Implications of a public smoking ban. Can J Public Health 2008;99:62–5.
27. 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;10:1093–7.
28. Gallus S, Zuccare P, Colombo P, et al. Smoking in Italy 2005–2006: effects of a comprehensive national tobacco regulation. Prev Med 2007;45:198–201.
29. Gorini G, Gasparrini A, Fondelli MC, et al. Environmental tobacco smoke (ETS) exposure in Florence hospitality venues before and after the smoking ban in Italy. J Occup Environ Med 2005;47:1208–10.
30. Cearoni G, Forastiere F, Agabita N, Valente P, Zuccaro P, Perucci CA. Effect of the Italian smoking ban on population rates of acute coronary events. Circulation 2008;117:1183–8.
31. Valente P, Forastiere R, Bacosi A, et al. Exposure to fine and ultrafine particles from secondhand smoke in public places before and after the smoking ban, Ital 2005. Tob Control 2007;16:312–7.
32. Galeone D, Laurendi G, Vasselli S, Spizzichino L, D'Argenio P, Greco D. Preliminary effects of Italy's ban on smoking in enclosed public places. Tob Control 2006;15:143.
33. Vaselli B, Papini P, Gaelone D, et al. Reduction incidence of myocardial infarction associated with a national legislative ban on smoking. Minerva Cardioangiol 2008;56:197–203.
34. Pell JP, Haw S, Cobbe S, et al. Smoke-free legislation and hospitalizations for acute coronary syndrome. N Engl J Med 2008;359:482–91.
35. Semple S, Creely KS, Naji A, Miller BG, Ayres JG. Secondhand smoke levels in Scottish pubs: the effect of smoke-free legislation. Tob Control 2007;127:127–32.
36. Pechacek TF, Babb S. How acute and reversible are the cardiovascular risks of secondhand smoke? BMJ 2004;328:980–3.
37. Richiardi L, Vizzini L, Merletti F, Barone-Adesi F. Cardiovascular benefits of smoking regulations: the effect of decreased exposure to passive smoking. Prev Med 2009;48:167–72.
38. Cronin E, Kearney P, Kearney P, Sullivan P. Impact of a national smoking ban on the rate of admissions to hospital with acute coronary syndromes. Presented at: European Society of Cardiology 2007 Congress; September 4, 2007; Vienna, Austria.
39. RTI International. First Independent Evaluation of New York's Tobacco Control Program: Final Report. Research Triangle Park, NC: RTI International, 2004.
40. Centers for Disease Control and Prevention. Indoor air quality in hospitality venues before and after implementation of a clean indoor air law. MMWR Morb Mortal Wkly Rep 2004;53:1038–41.
41. Bietta C, Binkin N, Bertozzi N, et al. Abitudine al fumo: I dati della 123 ASI. Dello studio "Passi." BEN-Bollettino Epidemiologico Nazionale. BEN Notiziario ISS 2006;19:1–3.
42. Njolstad I, Arnesen E, Lund-Larsen PG. Smoking, serum lipids, blood pressure, and sex differences in myocardial infarction: a 12-year follow-up of the Finn-mark Study. Circulation 1996;93:450–6.
43. Fichtenberg CM, Glantz SA. Effect of smoke-free workplaces and smoking behaviors: systematic review. BMJ 2002;325:188.
44. Brownson RC, Hopkins DP, Wakefield MA. Effects of smoking restrictions in the workplace. Annu Rev Public Health 2002;23:333–48.
45. Gallus S, Zuccaro P, Columbo P, et al. Effects of new smoking regulations in Italy. Ann Oncol 2006;17:346–7.
46. The Joint European Society of Cardiology/American College of Cardiology Committee. Myocardial infarction redefined—a consensus document of the Joint European Society of Cardiology/American College of Cardiology Committee for the redefinition of myocardial infarction. J Am Coll Cardiol 2000;36:959–69.
47. Valente P, Forastiere F, Bacosi A, et al. Exposure to fine and ultrafine particles from secondhand smoke in public places before and after the smoking ban, Italy 2005. Tob Control 2007;16:312–7.
48. Mulcahy M, Evans DS, Hammond SK, Repace JL, Byrne M. Secondhand smoke exposure and risk following the Irish smoking ban: an assessment of salivary cotinine concentrations in hotel workers and air nicotine levels in bars. Tob Control 2005;14:384–8.
49. Burghuber OC, Punsengruber C, Sinzinger H, Haber P, Silberbauer K. Platelet sensitivity to prostacycline in smokers and nonsmokers. JAMA 1986;90:34–8.
50. Sinzinger H, Kefalides A. Passive smoking severely decreases platelet sensitivity to antiaggregetory prostaglandins. Lancet 1982;2:392–3.
51. Schmid P, Karanikas G, Kritz H, et al. Passive smoking and platelet thromboxane. Thromb Res 1996;81:451–60.
52. Stefandis C, Vlachopoulos C, Tsiamas E, et al. Unfavorable effects of passive smoking on aortic function in man. Ann Intern Med 1998;128:426–34.
53. Osuka R, Watanabe H, Hirata K, et al. Acute effects of passive smoking on the coronary circulation of healthy young adults. JAMA 2001;286:436–41.
54. Pope CA, Eatough DJ, Gold DR, et al. Acute exposure to environmental tobacco smoke and heart rate variability. Environ Health Perspect 2001;109:711–6.
55. Valkonen M, Kuusi T. Passive smoking induces atherogenic changes in low density lipoprotein. Circulation 1998;97:2012–6.
56. Moffatt RL, Chelland SA, Pecott DL, Stamford BA. Acute exposure to environmental tobacco smoke reduces HDL-c and HDL2-C. Prev Med 1978;299:21–4.
57. Shetty KD, DeLeire T, White C, Bhattacharya J. Changes in U.S. Hospitalization and Mortality Rates Following Smoking Bans. NBER Working Paper No. 14790. Cambridge, Massachusetts: National Bureau of Economic Research, 2009. Available at: http://www.nber.org/papers/w14790. Accessed July 9, 2009.

Key Words: smoking ▪ myocardial infarct ▪ public health ▪ secondhand smoke ▪ incidence.