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Smoking status and life expectancy after acute myocardial infarction in the elderly

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Abstract

Objective—Smokers have lower short-term mortality after acute myocardial infarction than non-smokers; however, little is known about the long-term effects of smoking on life expectancy after AMI. The study aimed to quantify the burden of smoking after AMI using life expectancy and years of life lost.

Methods—We analyzed data from the Cooperative Cardiovascular Project, a medical record study of 158,349 elderly Medicare patients with AMI and over 17 years of follow-up, to evaluate the age-specific association of smoking with life expectancy and years of life lost after AMI.

Results—Our sample included 23,447 (14.8%) current smokers. Current smokers had lower crude mortality up to 5 years, which was largely explained by their younger age at AMI. After adjustment other patient characteristics, smoking was associated with lower 30-day (HR 0.91, 95% confidence interval (CI) 0.87–0.94) but higher long-term mortality (17-year HR 1.19, 95% CI 1.17–1.20) after AMI. Overall, crude life expectancy estimates were lower for current smokers than non-smokers at all ages, which translated into sizeable numbers of life-years lost attributable to smoking. As age at AMI increased, the magnitude of life-years lost due to smoking decreased. After full risk adjustment, the differences in life expectancy between current smokers and non-smokers persisted at all ages.

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Conclusions—Current smoking is associated with lower life expectancy and large numbers of life-years lost after AMI. Our findings lend additional support to smoking cessation efforts after AMI.

Keywords

acute myocardial infarction; smoking; mortality; life expectancy

Smoking increases the risk of coronary heart disease and cardiovascular-related mortality in the general population,[1–3] but studies of patients with acute myocardial infarction (AMI) have consistently reported lower crude mortality in smokers compared with nonsmokers.[4–8] This “smoker’s paradox” has been investigated in numerous short-term studies but relatively few long-term studies. In general, short-term studies have reported lower or similar mortality after adjustment in smokers,[4–13] whereas long-term studies have found similar or higher adjusted mortality in smokers.[14–17] These discrepancies in study findings are likely due to differences in study characteristics and covariate selection. Additionally, short and long-term studies available have been limited by small numbers of current smokers,[5, 8, 11, 13–15] minimal covariate adjustment,[8, 10–12, 14, 15] and specific inclusion criteria such as hospital survivors,[14] patients undergoing revascularization,[5, 8, 9, 13] or non-ST-elevation AMIs,[16] which may limit their generalizability to all patients. As a result, it is unclear whether the “smoker’s paradox” can be explained by other patient characteristics and whether this short-term phenomenon persists over the long-term.

Clarifying these issues requires a comprehensive assessment of the relationship between smoking with early and late mortality in post-AMI patients. However, such assessments have been difficult to conduct because most datasets lack the size, follow-up, or clinical detail required. Using data from the Cooperative Cardiovascular Project (CCP), we took advantage of a unique opportunity to characterize the association between smoking with short and long-term survival after AMI. This large dataset contains over 17 years of follow-up and detailed clinical information. The long follow-up afforded by CCP allows us to estimate the years of life lost or gained after AMI attributable to smoking. This metric quantifies the absolute burden of smoking in patients with AMI by comparing life expectancy in smokers and non-smokers. Although years of life lost has been used extensively in population-based studies to quantify premature mortality and the burden of behavioral risk factors such as smoking, it is virtually absent from the cardiovascular literature.[18] The aims of this study were to determine the association of current smoking with short and long-term mortality after AMI and to quantify the burden of smoking after AMI using years of life lost. We hypothesized that the short-term survival advantage observed in current smokers can be explained, in part, by differences in patient characteristics between current smokers and non-smokers, and that over time, this survival advantage reverses and current smokers have higher mortality compared with non-smokers.

METHODS

Cooperative Cardiovascular Project

Conducted by the Health Care Financing Administration (now Center for Medicare and Medicaid Services (CMS)), the CCP was a prospective, quality-improvement project designed to improve the care of AMI patients in the United States.[19, 20] Between January 1994 and February 1996, all fee-for-service Medicare beneficiaries hospitalized with a principal discharge diagnosis of AMI (*International Classification of Diseases, 9th Revision, Clinical Modification (ICD-9-CM) code 410*) were sampled from nonfederal acute-care hospitals in the U.S. for approximately 8 consecutive months (n=234,769). When patients had repeat admissions for the same AMI episode (*ICD-9-CM code 410.x2*), only the initial admission was included. Medical charts were abstracted for patient demographics, cardiovascular risk factors, medical history, clinical presentation, guideline-based admission therapies, in-hospital events, and discharge disposition. This study was approved by the Yale University institutional review board.

Study Sample

For this analysis, we included all patients aged ≥ 65 years with AMI confirmed by medical record, defined as an elevation of creatine kinase-MB level (>5% of total creatine kinase), or an elevation of lactate dehydrogenase enzyme (LDH) level with isoenzyme reversal (LDH₁>LDH₂), or the presence of at least 2 of the following: chest pain, 2-fold elevation in total creatine kinase, and diagnostic ECG changes (ST-segment elevation or new pathological Q-waves). Patients who were <65 years of age (n=17,593) or without clinically confirmed AMI (n=31,186) were excluded. We limited our sample to elderly patients with AMI in order to obtain a nationally representative sample of AMI patients. Whereas most US citizens are eligible for Medicare upon turning 65 years of age, younger patients are eligible for Medicare only if they qualify for disability or dialysis and thus are not representative of the younger AMI population. For patients with multiple admissions during the study period, we excluded all admissions except the first (n=27,500). We also excluded patients transferred from post-ambulatory or surgical settings (n=19,379) and patients whose records could not be linked to the Medicare Denominator Files to ascertain vital status (n=2,195). Of the 234,769 patients in the CCP, 158,349 met eligibility criteria for this analysis.

Study Variables

Information on current smoking status at the time of AMI was abstracted from the medical record and recorded as a dichotomous variable. The non-smoker group included never smokers and former smokers. The primary outcome was 17-year survival from admission, which was obtained from the 1994–2012 Medicare Denominator files. These files provide complete follow-up on all patients enrolled in Medicare and thus no patients were lost to follow-up except those that could not be linked to the Medicare Denominator files.

Statistical Analyses

Baseline characteristics were compared between current smokers and non-smokers using chi-squared tests for categorical variables and t-tests for continuous variables. Cox proportional hazards models were constructed to evaluate the hazard ratios for death, with current smoking as the independent variable of interest, at 30-days, 1-year, 5-years, and 17-years. For the 30-day, 1-year, and 5-year models, patients surviving these time frames were censored at 30 days, 1 year, and 5 years, respectively. Proportional hazards assumptions were checked for all models using Schoenfeld residuals, examined graphically and tested formally. We used sequential adjustment to determine which covariates, if any, mediated the relationship between current smoking and mortality. In model 1, we adjusted for patient age only. Model 2 controlled for patient demographics (sex and race), traditional cardiovascular risk factors (diabetes, hypertension, body mass index, history of coronary artery disease) and medical history (congestive heart failure (CHF), chronic obstructive pulmonary disease (COPD), cerebrovascular accident, peripheral vascular disease). Model 3 further controlled for clinical presentation (Killip class, AMI location, ST-segment elevation AMI, pulse, systolic blood pressure), and therapies received (percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG) within 30 days of admission, fibrinolytic therapy, and among those eligible, aspirin and beta-blockers on admission). Covariates were selected based on prior reports,[5, 6, 8, 14–16] face validity, and clinical judgment. Patients with missing data on continuous covariates were assigned the median value in the cohort and a binary dummy variable to denote missing. Patients with missing data on categorical covariates were included in the model using a dummy variable for missing data.

We defined life expectancy, or mean survival, as the area under the survival curve and used a 3-step process to calculate life expectancy for current smokers and non-smokers. These methods have been described previously and are detailed in the supplemental appendices. [21] Briefly, we used Cox proportional hazards models to plot the expected survival curves for current smokers and non-smokers at each age and then extrapolated the curves to age 100 using exponential models. Life expectancy was estimated by summing the areas under the Cox and exponential curves. Years of life lost after AMI attributable to current smoking were calculated by subtracting life expectancy in current smokers from that in non-smokers.

To determine whether the differences in life expectancy between current smokers and non-smokers were explained by differences in patient characteristics between the two groups, we repeated our calculations of life expectancy adjusting for the full list of covariates listed above. Adjusted years of life lost were again calculated by subtracting adjusted mean survival in current smokers from that in non-smokers. All statistical analyses were performed using SAS 9.2 (SAS Institute, Cary, NC).

RESULTS

Of the 158,349 patients with AMI in our cohort, 23,447 (14.8%) were current smokers. Compared with non-smokers, current smokers were younger and more likely to be male. In addition, they had lower rates of common cardiovascular risk factors including diabetes, hypertension, and obesity but higher rates of COPD and peripheral vascular disease (Table

1). They were also more likely to present with ST-elevation AMI and to receive acute reperfusion therapy.

Current smokers had lower crude mortality at 30-days, 1-year, and 5-years after AMI but slightly higher mortality over 17-years (Table 2). By the end of follow-up, 93.6% of current smokers were deceased compared with 92.9% of non-smokers. Unadjusted survival curves showed consistently higher mortality for non-smokers with the curves converging towards the end of follow-up (Figure 1); however, adjustment for age attenuated these effects (Table 2). Relative to non-smokers, the age distribution of current smokers was concentrated at younger ages and heavily right-skewed (Figure 2). After age adjustment, the hazard ratio of 30-day mortality in current smokers vs. non-smokers was not significantly different from one, but current smokers had significantly higher hazards of mortality at 1, 5, and 17 years. Further adjustment for medical history and clinical presentation again rendered the hazards of 30-day mortality in current smokers lower than for non-smokers (HR 0.91, 95% confidence interval (CI) 0.87–0.94) and attenuated this difference for 1-year mortality (HR 0.97, 95% CI 0.94–1.00). However, the higher hazards of 5- and 17-year mortality for current smokers vs. non-smokers persisted (5-year: 1.07, 95% CI 1.05–1.10; 17-year: 1.19, 95% CI 1.17–1.20).

Crude life expectancy estimates were lower for current smokers than non-smokers at all ages (Figure 3). These differences resulted in sizeable numbers of life-years lost attributable to current smoking; however, the magnitude of life-years lost due to smoking decreased as age at AMI increased (Figure 4). Before adjustment, 65-year-old smokers lived 2.90 (95% CI 2.68–3.12) fewer years of life after AMI, and 85-year-old smokers lived 0.41 (95% CI 0.30–0.52) fewer years. After full risk adjustment, the differences in life expectancy between current smokers and non-smokers persisted but decreased. Current smoking was associated with 1.96 (95% CI 1.76–2.16) life-years lost at age 65 and 0.16 (95% CI 0.06–0.26) life-years lost at age 85.

DISCUSSION

Using data from a large, nationally representative cohort of about 150,000 elderly patients with AMI, we found that the lower risk of short- and long-term mortality in current smokers after AMI was largely explained by their younger age at presentation. After adjustment for age, current smokers had comparable 30-day mortality and higher long-term mortality than non-smokers. However, after adjustment for clinical and treatment characteristics, current smoking was associated with lower 30-day, comparable 1-year, and higher 5- and 17-year mortality. The higher long-term mortality in current smokers resulted in significantly lower life expectancies after AMI for smokers at all ages and large numbers of life-years lost after AMI attributable to smoking.

Prior studies evaluating the relationship between smoking and short-term mortality after AMI have been inconsistent. Nearly all studies have reported lower 30-day and 1-year unadjusted mortality for smokers but lower,[6, 7, 10, 11] comparable,[4, 5, 8, 9, 13] or even higher[12] risk of mortality after adjustment. Inconsistencies between studies are likely due

to differences in study samples, length of follow-up, covariate adjustment, and the categorization of smokers.

Our results may offer insight into why studies of short-term mortality have been mixed. First, we found that the relationship between current smoking status and mortality varies by length of follow-up, even in the short-term. In our study, current smokers had a lower risk of 30-day mortality but comparable risk of 1-year mortality as non-smokers after full adjustment. Accordingly, most studies that have found lower mortality in smokers have evaluated hospital mortality,[8, 10, 11] whereas studies reporting no differences in mortality by smoking have generally examined 1-year mortality.[5, 8, 13] Second, we found that the association between current smoking status and short-term mortality depended on covariate selection. Using sequential adjustment, we found no differences in 30-day mortality between current smokers and non-smokers after adjustment for age but lower mortality for current smokers after adjustment for other clinical and treatment characteristics.

Over the long-term, we found that differences in mortality between current smokers and non-smokers were largely explained by differences in age. Current smokers in our sample were, on average, 4.8 years younger than non-smokers, which may also explain the lower prevalence of cardiovascular risk factors in smokers in our sample. The age difference in smokers and non-smokers is also consistent with observations from the general population suggesting that smoking causes premature AMIs in men by approximately 8.3–8.7 years and in women by 10.8–14.4 years.[22,23]

Very few studies have evaluated the relationship between smoking and long-term mortality after AMI; however, the few studies available have reported similar or higher mortality for smokers after adjustment.[14–17] Using data from the CRUSADE registry, Shen et al. found that smoking was associated with increased 3-year mortality in patients with non-ST-segment elevation AMI.[16] Likewise, Gerber et al. found that never smokers and quitters had significantly lower 13-year mortality after AMI than persistent smokers.[14] Our study improves upon prior long-term studies by 1) including large numbers of smokers and non-smokers, 2) extending follow-up to the entire remaining lifespan, 3) adjusting for multiple demographic and clinical characteristics, and 4) quantifying the burden of smoking after AMI using years of life lost. This metric has several strengths above traditional mortality estimates.[18,24] As indices of premature mortality, years of life lost can be used to quantify the effect of smoking after AMI over the entire lifespan and to compare the burden of smoking across multiple ages. In addition, quantifying the years of life lost attributable to smoking may be a more effective way to communicate the harmful effects of smoking to patients as opposed to relative mortality rates.[25] To our knowledge, only one other study has used years of life lost to evaluate the relationship between smoking and post-AMI mortality; however, this study estimated the years of life lost attributable to AMI in smokers and non-smokers rather than the years of life lost after AMI attributable to smoking as we have done in our study.[26]

Several explanations may account for our finding that smoking is associated with increased mortality after AMI over the long-term but not the short-term. Whereas mortality in the first 30-days after AMI is largely related to the extent of myocardial damage, comorbid

conditions, and timely receipt of therapies, mortality over the long-term depends largely on the extent and progression of atherothrombotic disease in the coronary arteries as well as other competing risks. We found that current smokers had less comorbidity but higher rates of acute reperfusion therapy after AMI, which may explain their lower mortality in the first 30 days. Although it is unclear why the effect of smoking on 30-day mortality became protective after full adjustment, one explanation may be that current smokers have the opportunity to alter a potent risk factor by quitting smoking and thereby rapidly reduce their risk of short-term death and recurrent AMI. In contrast, non-smokers likely have other risk factors that are more difficult to alter in the short-term. Prior studies have shown that up to 74% of smokers may quit, at least temporarily, in the first month after AMI[27] and that smoking cessation has immediate benefits after AMI, including reductions in angina and in-hospital complications.[28,29] Over the long-term, however, smoking causes endothelial dysfunction by increasing oxidative stress, vascular inflammation, and thrombosis, which predisposes patients to recurrent cardiac events.[30] Indeed, several studies have shown that smokers are at increased risk of recurrent infarction even in the first year after AMI.[13] Over time, this prothrombotic state places smokers at increased risk of cardiovascular mortality. In addition, smoking is associated with other competing risks and higher rates of noncardiovascular death in the general population from causes such as lung cancer and COPD. Not surprisingly, we found a higher prevalence of COPD in current smokers than non-smokers in our cohort. Thus, the higher risk of long-term mortality in smokers is likely mediated through both cardiac and non-cardiac pathways.

Interestingly, we observed that the years of life lost attributable to smoking narrowed as age at AMI increased. Although we might expect the gap in life expectancy to be smaller among older patients with lower life expectancies, it is also possible that smokers who reach the oldest ages represent a select group of patients with low frailty and low mortality. This phenomenon has been previously reported in the general population.[31] It is hypothesized that older smokers represent a healthier cohort than younger smokers because they have survived the increased risk of premature death due to smoking. Our study is the first to report this phenomenon in a population of patients with established cardiovascular disease.

This study was subject to several limitations. First, only current smoking status at the index hospitalization of the patient was recorded. No information on the duration or intensity of smoking or previous habits was collected. Thus, there is likely a fair amount of heterogeneity in risk among both current and non-smokers, and the estimates presented in this study represent averages across smokers and non-smokers with varying smoking histories and intensities. Second, we do not know how many patients quit smoking after AMI. Prior studies have noted that quitters have improved prognosis after AMI relative to persistent smokers.[14, 17, 32] As a result, including both quitters and persistent smokers together may attenuate our risk estimates. In fact, it is very likely that the life-years lost attributable to smoking differs in quitters and persistent smokers. Third, because the Medicare Denominator files record only all-cause mortality, we were unable to differentiate cardiovascular-related deaths from other causes and thus could not determine whether the higher long-term mortality in current smokers was attributable to cardiovascular disease or other smoking-related deaths. Fourth, our study sample included only elderly patients aged 65 and older and thus may not be generalizable to younger patients. Nevertheless, the

greatest differences in life expectancy between current and non-smokers occurred among younger patients suggesting that there may be even larger numbers of years of life lost attributable to smoking among patients <65 years of age. Fifth, in order to calculate life expectancy after AMI, we needed a cohort with long-term follow-up, which only a study like CCP could offer. As a result, the data used for this study were collected in the pre-primary PCI era and thus our life expectancy estimates may differ from those of patients today. It is possible that contemporary treatment with primary-PCI may be associated with better outcomes in both smokers and non-smokers. However, previous literature suggests that smokers may have higher rates of complications after reperfusion therapy such as major bleeding, stroke, or reinfarction.[33,34] Thus, it is unclear how the magnitude of life-years lost after AMI attributable to smoking differs today. Finally, we adjusted for several clinical and treatment characteristics, some of which may be in the causal pathway between smoking and mortality after AMI. We opted for thorough risk adjustment at baseline in order to determine which variables if any explained the association between current smoking status and long-term mortality after AMI. However, it is possible that we adjustment for certain variables such as COPD or stroke may have resulted in overadjustment of the models.

In summary, we showed that current smoking was associated with lower 30-day, comparable 1-year, and higher 5- and 17-year mortality. Although the smoker's paradox persisted in the short-term, the association between smoking and long-term mortality was largely explained by the younger age of current smokers at the time of AMI. Using data from the largest and longest follow-up study of elderly AMI patients in the U.S, we demonstrated that current smoking status was associated with lower life expectancy and large numbers of life-years lost after AMI. Our findings lend additional support to efforts that encourage smoking cessation in patients at risk for coronary heart disease by demonstrating poorer prognosis after AMI in current smokers. Years of life lost may be particularly helpful for communicating the harms of smoking to patients with AMI.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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KEY QUESTIONS**What is already known about this subject?**

Studies of patients with acute myocardial infarction (AMI) have consistently reported lower short-term crude mortality in smokers compared with nonsmokers; however, little is known about the effect of smoking on long-term mortality after AMI and whether the lower mortality in smokers can be explained by other patient characteristics.

What does this study add?

Using data from a large, nationally representative cohort of patients with AMI with detailed clinical information and over 17 years of follow-up, we found that current smokers had significantly shorter life expectancy after AMI than non-smokers. In patients 85 years of age, current smoking was associated with between 0.16 and 1.96 years of life lost after AMI relative to non-smokers. Years of life lost attributable to smoking were greatest in younger patients.

How might this impact on clinical practice?

Our findings lend additional support to smoking cessation efforts after AMI. In addition, years of life lost may be particularly helpful for communicating the harms of smoking to patients with coronary artery disease.

Figure 1. Unadjusted survival curves for smokers versus non-smokers calculated from admission Survival curves were modeled using Cox proportional hazards regression and thus represent expected survival for current smokers and non-smokers. Red line represents current smokers and blue line represents non-smokers.

Figure 2.
Age distribution of smokers and non-smokers at the time of AMI hospitalization.

Figure 3. Life expectancy estimates for smokers and non-smokers calculated from admission Estimates are calculated from a Cox proportional hazards model that includes only age, smoking status, and their interaction.

Figure 4. Unadjusted and adjusted years of potential life lost attributable to smoking at the time of AMI

Multivariable estimates are adjusted for patient demographics (sex and race), traditional cardiovascular risk factors (diabetes, hypertension, body mass index, history of coronary artery disease), medical history (congestive heart failure, chronic obstructive pulmonary disease, cerebrovascular accident, peripheral vascular disease), clinical presentation (Killip class, AMI location, ST-segment elevation AMI, pulse on admission, systolic blood pressure on admission), and therapies received (percutaneous coronary intervention or coronary artery bypass grafting in the first 30 days after admission, fibrinolytic therapy during hospitalization, and aspirin and beta-blockers on admission).

Table 1

Baseline characteristics of patients by current smoking status on admission (n=158,349): the Cooperative Cardiovascular Project, 1994–1996*

Characteristics	Current Smoker n=23,447 (14.8%)	Non-Smoker n=134,902 (85.2%)	p-value
Demographics			
Age, mean (SD)	72.41 (5.82)	77.20 (7.40)	<0.001
Female	10,336 (44.1)	66,967 (49.6)	<0.001
Nonwhite race	2,540 (10.8)	13,035 (9.7)	<0.001
Risk Factors			
Diabetes mellitus	5,267 (22.5)	43,228 (32.0)	<0.001
Hypertension	13,189 (56.3)	84,624 (62.7)	<0.001
BMI			<0.001
Normal Weight (<25)	10,052 (42.9)	47,522 (35.2)	
Overweight (25–30)	6,869 (29.3)	41,553 (30.8)	
Obese (>30)	3,068 (13.1)	21,595 (16.0)	
Missing	3,458 (14.8)	24,232 (18.0)	
Prior MI/PCI/CABG	3,522 (15.0)	23,247 (17.2)	<0.001
Comorbidities			
CHF	3,655 (15.6)	30,080 (22.3)	<0.001
COPD	9,638 (41.1)	22,667 (16.8)	<0.001
CVA/stroke	2,905 (12.4)	19,274 (14.3)	<0.001
PVD	3,645 (15.6)	12,846 (9.5)	<0.001
Clinical Presentation			
Killip class >2	7,941 (33.9)	51,120 (37.9)	<0.001
SBP (mmHg), mean (SD)	115.2 (11.6)	116.0 (10.8)	<0.001
Missing	145 (0.6)	702 (0.5)	
HR (bpm), mean (SD)	87.6 (25.4)	88.0 (24.7)	<0.001
STEMI	7,847 (33.5)	38,135 (28.4)	<0.001
Anterior infarction	10,731 (45.8)	63,047 (46.7)	0.006
Cardiac arrest on admission	950 (4.1)	4,844 (3.6)	0.001
Treatment			
PCI/CABG in first 30 days	7,950 (33.9)	37,451 (27.8)	<0.001
Missing	675 (2.9)	4,219 (3.1)	
Fibrinolytic therapy	5,632 (24.0)	22,170 (16.4)	<0.001
Aspirin on admission among eligible	14,239/18,291 (77.9)	75,427/102,017 (73.9)	<0.001
Beta-blockers on admission among eligible	5,277/9,034 (58.4)	38,244/64,435 (59.4)	0.089

* Only variables with missing data have “missing” rows. Variables without “missing” have no missing data.

Table 2

Crude mortality and hazard ratios for mortality in current smokers versus non-smokers by length of follow-up from admission^a

	Crude Mortality		p-value ^b	Unadjusted	Hazard Ratios (95% CI)		
	Current Smokers	Non-Smokers			Adjusted for Age ^c	Adjusted for Medical History ^d	Fully Adjusted ^e
30-Days	3222 (13.7)	23,967 (17.8)	<0.001	0.76 (0.73, 0.79)	1.00 (0.96, 1.03)	0.97 (0.93, 1.01)	0.91 (0.87, 0.94)
1-Year	6454 (27.5)	44854 (33.3)	<0.001	0.79 (0.77, 0.82)	1.08 (1.05, 1.11)	1.02 (0.99, 1.05)	0.97 (0.94, 1.00)
5-Years	12,344 (52.7)	77,355 (57.3)	<0.001	0.86 (0.84, 0.88)	1.18 (1.16, 1.21)	1.12 (1.09, 1.14)	1.07 (1.05, 1.10)
17-Years	21,945 (93.6)	125,273 (92.9)	<0.001	0.95 (0.93, 0.96)	1.27 (1.25, 1.29)	1.22 (1.20, 1.24)	1.19 (1.17, 1.20)

^aSurvival times are calculated from admission.

^bP-value for chi-squared comparison of crude mortality in current smokers and non-smokers.

^cAdjusted for age only.

^dAdjusted for age, gender, race, and medical history (diabetes, hypertension, obesity, prior MI, CHF, COPD, cerebrovascular accident, and peripheral vascular disease)

^eAdjusted for age, gender, race, medical history (diabetes, hypertension, obesity, prior MI, CHF, COPD, cerebrovascular accident, and peripheral vascular disease), clinical presentation (Killip class, AMI location, ST-elevation AMI, pulse on admission, systolic blood pressure on admission), and therapies received (PCI/CABG within 30 days of admission, fibrinolytic therapy during the index admission, and among those eligible, aspirin on admission and beta-blockers on admission).