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# Life Years Gained From Smoking-Cessation Counseling After Myocardial Infarction

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**Introduction:** Hospitalization for acute myocardial infarction (AMI) is an opportune time to counsel smokers to quit. Studies have demonstrated lower short-term mortality for counseled versus non-counseled smokers; yet, little is known about the long-term survival benefits of post-AMI smoking-cessation counseling (SCC).

**Methods:** Data from the Cooperative Cardiovascular Project, a prospective cohort study of elderly patients with AMI between 1994 and 1996 with >17 years of follow-up, were used to evaluate the association of SCC with short- and long-term mortality in smokers with AMI. Life expectancy and years of potential life gained were used to quantify the long-term survival benefits of SCC. Cox proportional hazards models with exponential extrapolation were used to estimate life expectancy.

**Results:** The analysis included 13,815 smokers, of whom 5,695 (41.2%) received SCC. Non-counseled smokers had higher crude mortality than counseled smokers over all 17 years of follow-up. After adjustment for patient and hospital characteristics, SCC was associated with a 22.6% lower 30-day mortality and a 7.5% lower mortality over 17 years. These survival differences produced higher life expectancy estimates for counseled smokers than non-counseled smokers at all ages, which resulted in average gains in life years of 0.13 (95% CI=−0.31, 0.56) to 0.58 (95% CI=0.25, 0.91) years, with the largest gains observed in older smokers.

**Conclusions:** SCC is associated with longer life expectancy and gains in life years in elderly smokers with AMI, supporting the importance of post-AMI counseling efforts.

## INTRODUCTION

Hospitalization for acute myocardial infarction (AMI) is an opportune time to counsel smokers to quit smoking because patients are often motivated to quit. National guidelines strongly recommend smoking-cessation counseling (SCC) for smokers hospitalized with AMI,[1–3] and Centers for Medicare and Medicaid Services (CMS) has adopted SCC as a process-of-care measure to evaluate hospital performance.[4,5] Despite the robust support for SCC, most evidence for SCC after AMI comes from studies reporting higher quit rates in counseled versus non-counseled smokers[6–10] and other studies finding lower short- and long-term mortality in quitters than in persistent smokers.[10–14] Few

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studies have directly assessed the role of SCC on mortality after AMI, and only one has evaluated long-term mortality.[15–18] These studies have reported lower short-term mortality for counseled versus non-counseled smokers; however, the persistence of this survival benefit over the long term is unknown.

This long-term perspective is important to understand the overall benefits of SCC, for two reasons. First, quitters in the general population have higher mortality than never smokers, likely due to higher long-term cardiovascular and other smoking-related diseases.[19] Thus, the short-term benefits of SCC after AMI may be negated over the long term by the higher incidence of smoking-related deaths. Second, because smokers may initially quit in response to counseling but then restart,[8,20–22] it is important to assess both short- and long-term mortality benefits of SCC.

One method for evaluating both the short- and long-term benefits of SCC is to quantify its association with life expectancy. This approach measures the magnitude and persistence of survival benefits associated with SCC over the entire lifespan, and quantifies the years of potential life gained (YPLG) attributable to SCC. Accordingly, this study examines the differences in life expectancy after AMI between counseled and non-counseled smokers and quantifies the YPLG attributable to SCC.

## METHODS

### Study Design and Sample

The Cooperative Cardiovascular Project (CCP) was a prospective cohort study implemented by the Health Care Financing Administration (now CMS) to evaluate the care of patients with AMI in the U.S.[23,24] The database includes a random sample of all fee-for-service Medicare beneficiaries diagnosed with AMI (ICD-9-CM code 410) discharged from acute care, non-governmental hospitals in the U.S. during an 8-month period between January 1994 and February 1996. Repeat admissions for the same episode of care (ICD-9-CM code 410.x2) and patients with missing medical record numbers were excluded. Complete medical records for each patient were forwarded to a data center where trained medical abstractors performed the abstractions.[23] This study was approved by the Yale University IRB.

This analysis included current smokers aged ≥65 years with confirmed AMI who were discharged to home alive after the index hospitalization. AMI was defined as an elevation of creatine kinase-MB level (>5% of total creatine kinase), or an elevation of lactate dehydrogenase enzyme level with isoenzyme reversal (lactate dehydrogenase-1 > lactate dehydrogenase-2), or the presence of at least two of the following: chest pain, twofold elevation in total creatine kinase, and diagnostic electrocardiographic changes. Current smoking was defined as chart-documented smoking on admission. A total of 23,447 smokers were included in CCP. Exclusions included in-hospital deaths ($n$=537), because SCC could be administered anytime during the hospitalization; and patients discharged to locations other than home, including nursing facilities and other hospitals ($n$=9,095), because physicians might be less likely to administer SCC to "sicker" patients going to other care facilities. These exclusions left 13,815 smokers who were discharged to home.

Information on receipt of SCC during hospitalization was obtained from the medical record and recorded as a yes/no variable. Information on vital status was obtained via linkage to the 1994–2012 Medicare Denominator files, which contain complete mortality data on all patients enrolled in Medicare during a given year. Data were analyzed in 2014.

### Statistical Analysis

Baseline characteristics were compared between counseled and non-counseled smokers using chi-square tests for categorical variables and Student's $t$-tests for continuous variables. Marginal Cox proportional hazards models were used to evaluate the unadjusted and adjusted associations between SCC and mortality at 30 days, 1 year, 5 years, and 17 years after AMI. Proportional hazards assumptions were checked graphically for all models using Schoenfeld residuals. All models assessed survival from the time of discharge, and marginal models were applied to account for clustering of patients within hospitals. Multivariable models included age, sex, race, ZIP code–level median household income percentile, diabetes, hypertension, BMI, prior AMI, percutaneous coronary intervention, coronary artery bypass grafting, congestive heart failure, chronic obstructive pulmonary disease, cerebrovascular accident, peripheral vascular disease, Killip class on admission, anterior AMI, ST-segment elevation AMI, pulse and systolic blood pressure on admission, revascularization within 30 days of admission, fibrinolytic therapy during hospitalization, and aspirin and beta blockers on admission. In addition, models adjusted for hospital characteristics, including annual AMI volume, rural versus urban location, ownership, and teaching status. Covariates were selected based on prior reports[15,16,21] and face validity. Patients with missing systolic blood pressure or income percentile 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.

A three-step process was used to calculate life expectancy after AMI, which was defined as mean survival or the area under the expected survival curve. First, a marginal Cox proportional hazards model was fit that included receipt of SCC, age, and their pairwise interaction. Second, the expected survival curves were plotted for counseled and non-counseled smokers at each age. Because 7% of patients were still living at the end of follow-up, the age-specific expected survival curves from the marginal Cox model were extrapolated to age 100 years using exponential models. Exponential models were selected because they provide a conservative decay function. The curves were extrapolated up to age 100 years because the Centers for Disease Control and Prevention also uses this age as the upper threshold for life expectancy estimates in the general population.[25] The constant hazard for the exponential model was specified as the average hazard over the last 2 years of follow-up. Finally, life expectancy was estimated by calculating and summing the areas under the expected and extrapolated regions of the survival curve. Upper and lower confidence bounds on the expected survival curves were used to calculate 95% CIs for the life expectancy estimates. The YPLG

from SCC was estimated as the difference in life expectancy between counseled and non-counseled smokers.

Life expectancy analyses were then repeated adjusting for patient and hospital characteristics. Age-specific covariate frequencies or mean values were used to plot the expected survival curves from the marginal Cox models. For example, the prevalence of diabetes among 65-year-old smokers in the sample was 21.8%. These covariate frequencies were used to plot the expected survival curves for 65-year-old counseled and non-counseled smokers. This approach estimates life expectancy for the "average" recipient and non-recipient of SCC at a given age and allows direct comparison of life expectancy in same-aged recipients and non-recipients of SCC with similar levels of baseline covariates. YPLG from SCC was again estimated as the difference in adjusted life expectancy between recipients and non-recipients of SCC.

To determine if selection bias was present in the analyses, a sensitivity analysis was conducted using propensity score matching. Propensity scores were calculated using the same covariates as in the fully adjusted models, and counseled and non-counseled smokers were matched in a greedy 1:1 fashion on both age (within 1 year) and propensity score (within 0.02). Cox proportional hazards models were used to compare short- and long-term mortality between counseled and non-counseled smokers while accounting for the matched design. These analyses were performed as sensitivity analyses rather than primary analyses because life expectancy could not be calculated with a matched design. Hazard ratios from the matched models were qualitatively compared with those from the fully adjusted models. All statistical analyses were performed using SAS, version 9.2.

## RESULTS

The SCC analyses included 13,815 smokers, of whom 5,695 (41.2%) received SCC. Compared with non-counseled smokers, counseled smokers were slightly younger, more frequently non-white, and less likely to have diabetes, hypertension, and congestive heart failure, but more likely to have a history of chronic obstructive pulmonary disease and peripheral vascular disease (Table 1). Counseled smokers were also more likely to receive other guideline-recommended therapies, such as aspirin, beta-blockers, and acute reperfusion therapy.

Non-counseled smokers had higher crude mortality than counseled smokers at all follow-up timepoints; however, the magnitude of the difference at 17 years was relatively small (92.4% vs 93.4%, $p$=0.028) (Table 2). In unadjusted analyses, counseled smokers had significantly lower short- and long-term risk of death than non-counseled smokers, which persisted after adjustment for age and other covariates (Table 2 and Appendix Figure 1, available online). SCC was associated with a 22.6% lower hazard of death in the first 30 days after discharge, but only a 7.5% lower hazard after 17 years.

Life expectancy estimates after AMI were higher for counseled smokers than non-counseled smokers at all ages (Figure 1 and Appendix Table 1, available online). Figure 2 shows the YPLG attributable to SCC in smokers after AMI for both unadjusted and adjusted analyses. Before adjustment, SCC was associated with an average gain of 0.22 (95% CI= −0.28, 0.71) to 0.63 (95% CI=0.32, 0.92) years of life depending on patient age (Appendix Table 2, available online). After adjustment, YPLG decreased but remained significant for older patients. In sensitivity analyses, hazard ratios from the fully adjusted models were qualitatively similar to those from the age- and propensity-matched models at all follow-up timepoints, suggesting that the fully adjusted models adequately controlled for differences in observed characteristics between counseled and non-counseled smokers (Table 2).

## DISCUSSION

For smokers at the index admission, SCC was associated with a 23% lower hazard of death in the first 30 days after AMI and an 8% lower hazard over 17 years. The lower risk of death for counseled smokers resulted in average gains of 0.13 (95% CI=−0.31, 0.56) to 0.58 (95% CI=0.25, 0.91) years of life, with the largest gains observed in older smokers.

These results extend those of prior observational studies examining the association of SCC with mortality after AMI. Previous studies using CCP data have studied this issue. Using a sample of 16,743 CCP smokers discharged to home, Houston et al.[16] reported a 19% lower 30-day adjusted hazard of death after SCC but no difference at 2 years. Similarly, Brown and colleagues[15] studied the 788 CCP smokers from North Carolina and reported an adjusted 22% lower 5-year mortality with SCC. Although comparable to those in this study, these estimates differed, owing to differences in inclusion criteria, covariate adjustment, and analytic methods. In other AMI cohorts, Van Spall et al.[18] found that SCC was associated with a 37% lower risk in 1-year mortality. This study is the first to examine the effect of SCC on long-term mortality after AMI and its association with YPLG.

Only one trial has evaluated the effect of SCC on mortality after AMI. In this single-center trial, Mohiuddin and colleagues[17] randomized smokers with a diagnosis of acute coronary syndrome or decompensated heart failure to receive either intensive behavior modification with individualized pharmacotherapy or usual care, which also included routine SCC. This trial reported a much larger risk reduction in 2-year mortality for patients receiving intensive therapy (77%) than the 12% (hazard ratio, 0.88; 95% CI=0.78, 0.99) found in CCP,

**Table 1.** Baseline Characteristics of Smokers on Admission by Receipt of Smoking-Cessation Counseling (n=13,815)

| Characteristics | Smokers counseled (n=5,695 [41.2%]) | Smokers not counseled (n=8,120 [58.8%]) | *p*-value |
|---|---|---|---|
| Demographics | | | |
| Age, y, M (SD) | 71.60 (5.32) | 72.68 (5.83) | **<0.001** |
| Female | 2,440 (42.8) | 3,551 (43.7) | 0.301 |
| Nonwhite race | 564 (9.9) | 1,083 (13.3) | **<0.001** |
| ZIP code–level median household income, M (SD) | 29,408 (10,556) | 29,212 (10,925) | 0.305 |
| Missing | 247 (4.3) | 380 (4.7) | |
| Risk factors | | | |
| Diabetes mellitus | 1,134 (19.9) | 1,909 (23.5) | **<0.001** |
| Hypertension | 3,098 (54.4) | 4,721 (58.1) | **<0.001** |
| BMI | | | 0.095 |
| Normal weight (<25) | 2,495 (43.8) | 3,581 (44.1) | |
| Overweight (25–30) | 1,817 (31.9) | 2,478 (30.5) | |
| Obese (>30) | 789 (13.9) | 1,118 (13.8) | |
| Missing | 594 (10.4) | 943 (11.6) | |
| Prior CAD | 917 (16.1) | 1,377 (17.0) | 0.183 |
| Comorbidities | | | |
| CHF | 711 (12.5) | 1,372 (16.9) | **<0.001** |
| COPD | 2,491 (43.7) | 3,129 (38.5) | **<0.001** |
| CVA/stroke | 584 (10.3) | 1,031 (12.7) | **<0.001** |
| PVD | 950 (16.7) | 1,185 (14.6) | **0.001** |
| Clinical presentation | | | |
| Killip class >2 | 1,680 (29.5) | 2,639 (32.5) | **<0.001** |
| SBP, mmHg, M (SD) | 116.1 (10.0) | 116.2 (9.9) | 0.405 |
| Missing | 13 (0.2) | 27 (0.3) | |
| HR, bpm, M (SD) | 85.6 (24.8) | 87.8 (24.8) | **<0.001** |
| STEMI | 1,939 (34.1) | 2,492 (30.7) | **<0.001** |
| Anterior infarction | 2,457 (43.1) | 3,554 (43.8) | 0.466 |
| Cardiac arrest on admission | 111 (2.0) | 171 (2.1) | 0.521 |
| Treatment | | | |
| Revascularization (PCI/CABG) within 30 days | 1,729 (30.4) | 2,302 (28.4) | **0.002** |
| Missing | 183 (3.2) | 212 (2.6) | |
| Fibrinolytic therapy | 1,365 (24.0) | 1,654 (20.4) | **<0.001** |
| Aspirin on admission among eligible, *n/N* (%) | 3,772/4,502 (83.8) | 4,988/6,283 (79.4) | **<0.001** |
| Beta-blockers on admission among eligible, *n/N* (%) | 1,465/2,283 (64.2) | 2,025/3,324 (60.9) | **0.014** |
| Hospital characteristics | | | |
| AMI volume, per year, M (SD) | 202.5 (161.4) | 198.9 (161.4) | 0.199 |
| Rural hospital | 981 (17.2) | 1,320 (16.3) | 0.147 |
| Missing | 47 (0.8) | 85 (1.1) | |
| Ownership | | | **<0.001** |
| Public | 733 (12.9) | 1,014 (12.5) | |
| Not-for-profit | 4,410 (77.4) | 6,129 (75.5) | |
| For-profit | 505 (8.9) | 892 (11.0) | |
| Missing | 47 (0.8) | 85 (1.1) | |
| Teaching hospital | 2,280 (40.0) | 3,311 (40.8) | 0.383 |

*Note:* Data are *n* (%) unless otherwise noted. Boldface indicates statistical significance ($p < 0.05$).
AMI, acute myocardial infarction; bpm, beats per minute; CAD, coronary artery disease; CHF, congestive heart failure; COPD, chronic obstructive pulmonary disease; CVA, cerebrovascular accident; HR, heart rate; PCI/CABG, percutaneous coronary intervention/coronary artery bypass grafting; PVD, peripheral vascular disease; SBP, systolic blood pressure; STEMI, ST-elevation myocardial infarction.

**Table 2.** Crude Mortality and HRs for Counseled versus Non-Counseled Smokers by Length of Follow-up ($n$=13,815)[a]

| | Crude mortality | | | HR (95% CI)[c] | | |
|---|---|---|---|---|---|---|
| Length of follow-up | Counseled smokers ($n$=5,695) | Non-counseled smokers ($n$=8,120) | $p$-value[b] | Unadjusted | Adjusted[d] | Matched analysis[e] |
| 30 days | 122 (2.1) | 250 (3.1) | **0.001** | **0.692 (0.558, 0.860)** | **0.774 (0.621, 0.964)** | **0.765 (0.601, 0.973)** |
| 1 year | 761 (13.4) | 1,470 (18.1) | **<0.001** | **0.718 (0.658, 0.784)** | **0.819 (0.750, 0.895)** | **0.815 (0.736, 0.902)** |
| 5 years | 2,378 (41.8) | 4,027 (49.6) | **<0.001** | **0.782 (0.744, 0.823)** | **0.860 (0.817, 0.906)** | **0.868 (0.814, 0.926)** |
| 17 years | 5,261 (92.4) | 7,580 (93.4) | **0.028** | **0.880 (0.850, 0.912)** | **0.925 (0.893, 0.959)** | **0.910 (0.862, 0.959)** |

*Note:* Boldface indicates statistical significance ($p<0.05$).
[a]Survival times are calculated from discharge.
[b]Chi-squared comparison of crude mortality in counseled smokers and non-counseled smokers.
[c]HRs comparing risk of death in recipients of smoking-cessation counseling to non-recipients.
[d]Adjusted for demographics (age, gender, race, ZIP code–level median household income percentile), medical history (diabetes, hypertension, obesity, prior AMI, CHF, COPD, cerebrovascular accident, and peripheral vascular disease), AMI severity (Killip class, AMI location, ST-elevation AMI, pulse on admission, systolic blood pressure on admission), AMI 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), and hospital characteristics (annual AMI volume, location, ownership, and teaching status).
[e]Results from matched pair analyses ($n$=10,960). Counseled and non-counseled smokers were matched in a greedy 1:1 fashion on age and propensity scores. Propensity scores were generated using demographics (gender, race, ZIP code–level median household income percentile), medical history (diabetes, hypertension, obesity, prior MI, CHF, COPD, cerebrovascular accident, and peripheral vascular disease), AMI severity (Killip class, AMI location, ST-elevation AMI, pulse on admission, systolic blood pressure on admission), and AMI 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).
AMI, acute myocardial infarction; CHF, congestive heart failure; COPD, chronic obstructive pulmonary disease; HR, hazard ratio; PCI/CABG, percutaneous coronary intervention/coronary artery bypass grafting.

which is likely attributable to differences in treatment intensity between studies.

Interestingly, the largest gains in life expectancy attributable to SCC occurred in the oldest patients in this cohort. Given that elderly patients have shorter life expectancies and more competing risks than younger patients, life expectancy gains of between 5 and 8 months are both meaningful and impressive. Although prior studies have not examined interactions between SCC and age, there are several plausible explanations for this observation. Both clinical and population-based studies have shown that older age is an independent predictor of smoking cessation and maintenance after smoking-cessation intervention.[26–29] Thus, the effect may be greater in older patients because more patients may quit smoking and remain abstinent after SCC than in younger patients. Alternatively, differences in life expectancy among quitters and persistent smokers may be more pronounced in smokers with a longer smoking history. Because older smokers are likely to have accrued more pack years than younger smokers by virtue of their age, these differences may translate into greater gains in life expectancy for the oldest smokers.

The largest YPLG due to SCC were observed shortly after discharge and became attenuated over time. The immediate benefits of smoking cessation have been documented in prior studies showing reductions in angina and in-hospital complications among patients who quit smoking even for short periods.[30,31] These early improvements in outcomes are likely due to elimination of nicotine and carbon monoxide from the bloodstream, which rapidly reduces the risk of recurrent ischemia by decreasing heart rate, blood pressure, and myocardial demand.[32–34] Longer-term effects of smoking cessation include reversal of endothelial damage and reduced platelet aggregation, which occur as early as 8–11 weeks after cessation.[35–37] The attenuation of this effect over the long term may be due to high rates of relapse in quitters. Prior studies have reported relapse rates up to 40% at 1 year in smokers who temporarily quit after AMI.[8,20–22] This effect may indicate a need for reinforcement of SCC in the months or years following index hospitalization. Alternatively, because smoking is associated with higher mortality from non-cardiovascular causes, the attenuation of the effect over the long term may reflect higher mortality from other conditions.

This study was developed under the assumption that inpatient SCC decreases post-AMI mortality through smoking cessation. Although this assumption could not be tested because the CCP does not include longitudinal data on smoking behaviors, prior trials have demonstrated significant improvements in post-AMI cessation of 7%–36% after inpatient SCC,[6,8,9,21] and observational studies have shown that patients who quit smoking after AMI have improved short- and long-term mortality compared with persistent smokers.[10–14] As a result, national guidelines strongly recommend SCC for smokers hospitalized with AMI,[38,39] and CMS recognizes SCC

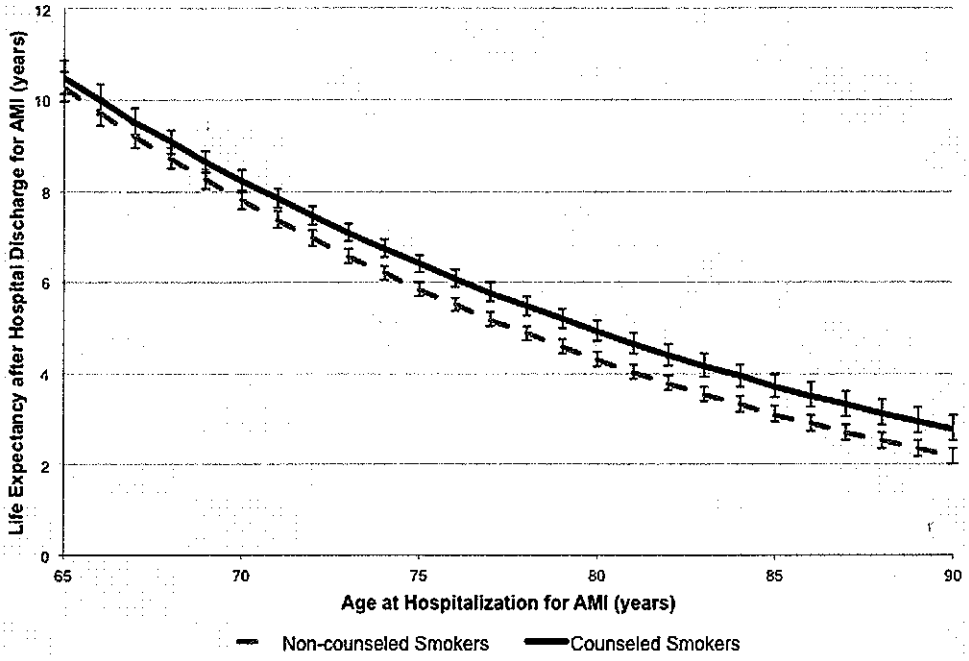


**Figure 1.** Life expectancy estimates for counseled smokers and not counseled smokers calculated from discharge.
*Note:* Estimates are calculated from a Cox proportional hazards model that includes only age, smoking cessation counseling, and their interaction.

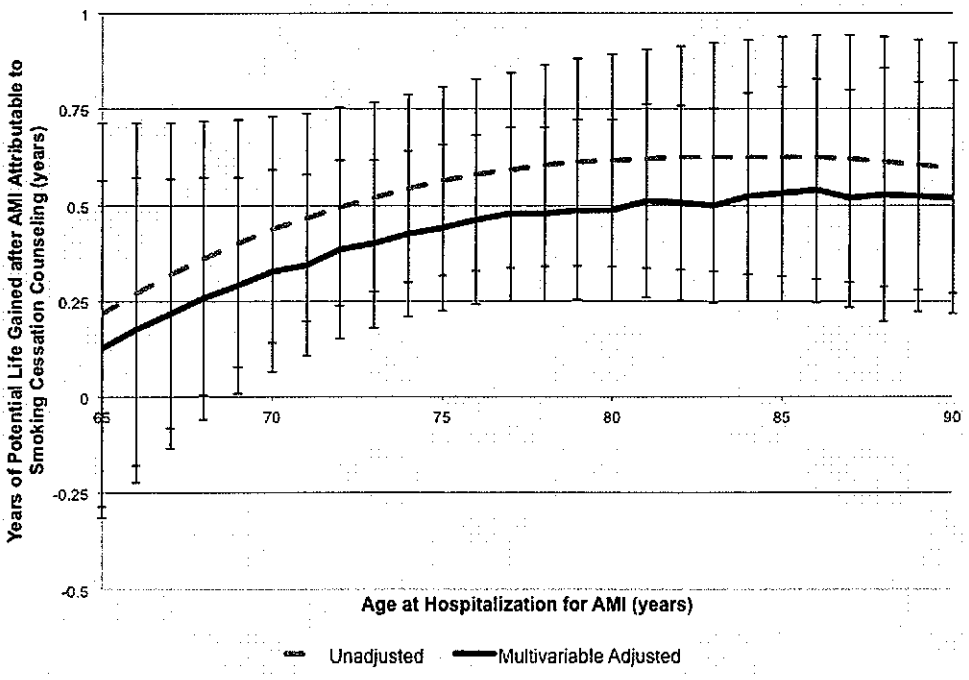


**Figure 2.** Unadjusted and adjusted years of potential life gained after AMI attributable to smoking cessation counseling.
*Note:* Multivariable estimates are adjusted for patient demographics (sex and race), traditional cardiovascular risk factors (diabetes, hypertension, BMI, history of coronary 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).
AMI, acute myocardial infarction.

as a national hospital inpatient quality measure.[40] In fact, CMS has determined that "the evidence is adequate to conclude that smoking and tobacco use cessation counseling, based on the current U.S. Public Health Service Guideline, is reasonable and necessary for a patient with a disease or an adverse health effect that has been found by the U.S. Surgeon General to be linked to tobacco use."[41] Nevertheless, the likelihood of successfully quitting may vary by intensity and duration of SCC, with some studies of lower intensity or shorter interventions finding no effect.[42–44]

The findings from this study reinforce the utility of SCC after AMI by demonstrating a survival benefit for recipients of SCC that persisted over 17 years of follow-up and was associated with meaningful gains in life expectancy. National guidelines from the American Heart Association and the American College of Cardiology recommend SCC after AMI, and rates of SCC approach 99% at most U.S. hospitals today, as opposed to the mid-1990s. However, guidelines around the content and intensity of inpatient SCC efforts are lacking. Moreover, SCC is frequently undermined by ineffective implementation.[45] Prior trials of intensive SCC or pharmacotherapy in patients with AMI have yielded larger risk differences that those observed in this study, suggesting that the benefits associated with SCC in this study are likely an underestimate of what would be attainable with more-intensive efforts or more-comprehensive SCC guidelines.[17,46–48] Similarly, pharmacologic agents to support smoking cessation have improved since this study was conducted. As a result, risk estimates may be greater than those reported in this study if counseling is accompanied by nicotine replacement therapies or other agents, such as bupropion or varenicline. Additional research is needed to better understand which SCC approaches are most beneficial and cost effective and how to support patients in the transition from inpatient to outpatient smoking cessation. Ultimately, the development of detailed guidelines around SCC may help to further improve outcomes for smokers hospitalized with AMI.

### Limitations

This study has some limitations. First, receipt of SCC was collected through medical record abstraction and thus may be subject to variations in documentation between providers and hospitals.[49] Second, the results from this study may be subject to selection bias if physicians preferentially selected "healthier" patients, who they deemed more likely to benefit from SCC. Alternatively, patients presenting at hospitals with better performance indicators may have been more likely to receive SCC. This issue was addressed by limiting the sample to patients who were healthy enough to be discharged home and by adjusting for patient- and hospital-level characteristics, but only observed variables were included. Indeed, the rate of SCC was substantially lower in patients discharged to locations other than home compared with those in this analysis (20.8% vs 41.2%). The similarity in hazard ratios between the fully adjusted and propensity-matched analyses suggests that risk adjustment was appropriate in accounting for observed variables. Residual confounding by an unobserved variable remains a possibility, as always in observational studies. Third, information on the type and intensity of inpatient SCC provided to patients and whether outpatient SCC was provided after discharge was not available. Fourth, it is unclear how many patients actually quit smoking after AMI. Fifth, because the Medicare Denominator files record all-cause mortality only, it was not possible to differentiate cardiovascular-related deaths from other causes. Sixth, although hospital characteristics were included in the adjusted models, information on physician characteristics was not available. These data have been known to influence the likelihood, and presumably the quality, of delivered SCC.[50–52]

## CONCLUSIONS

This study is the first to quantify the benefits of SCC after AMI using YPLG. Using data from the largest and longest follow-up study of elderly AMI patients in the U.S., this study found that SCC is associated with gains in life years after AMI. The finding of a sustained survival benefit in smokers receiving SCC has important implications for clinicians, who often fail to effectively intervene against smoking.[53,54] Future research is needed to understand which SCC interventions are most effective and how to maximize smoking-cessation efforts after AMI.

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## SUPPLEMENTAL MATERIAL

Supplemental materials associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.amepre.2016.08.013.

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