Prior Alcohol Consumption and Mortality Following Acute Myocardial Infarction

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IN THE GENERAL POPULATION, INDIVIDUALS who have 1 alcoholic drink every 1 to 2 days have lower rates of nonfatal acute myocardial infarction (AMI) and coronary mortality compared with abstainers and heavier drinkers. Studies in patients with hypertension or diabetes also show a U-shaped relationship between alcohol consumption and coronary heart disease (CHD). However, the effect of drinking in patients with documented CHD is less certain.

Alcohol has diverse physiological actions. Although the balance of effects appears to favor prevention of CHD in asymptomatic persons, it may differ in those with established CHD. For example, alcohol lowers exercise tolerance in patients with angina, causes dose-dependent coronary vasoconstriction, raises heart rate and systolic blood pressure, and increases levels of plasminogen activator inhibitor. These effects could adversely influence the prognosis of patients with CHD.

To our knowledge, no previous study has determined the effect of alcohol consumption on mortality immediately following AMI. Two previous studies that examined the effect of alcohol consumption among men with remote history of MI produced differing conclusions. One of these studies found that consumption of 2 to 3 drinks/wk was associated with lower mortality among male physicians who reported a previous MI, while the other study found no effect of consuming 1 to 14 drinks/wk among men with a previous MI. However, these earlier studies were limited by their restriction to men with prevalent CHD and use of self-reported diagnoses.

We therefore studied mortality following AMI as a function of alcohol consumption in the year prior to AMI in patients enrolled in the Determinants of Myocardial Infarction Onset Study (the Onset study). This multicenter, prospective cohort study included chart reviews and face-to-face interviews with patients who were hospitalized with confirmed AMI.

Context Studies have found that individuals who consume 1 alcoholic drink every 1 to 2 days have a lower risk of a first acute myocardial infarction (AMI) than abstainers or heavy drinkers, but the effect of prior drinking on mortality after AMI is uncertain.

Objective To determine the effect of prior alcohol consumption on long-term mortality among early survivors of AMI.

Design and Setting Prospective inception cohort study conducted at 45 US community and tertiary care hospitals between August 1989 and September 1994, with a median follow-up of 3.8 years.


Main Outcome Measure All-cause mortality, compared by self-reported average weekly consumption of beer, wine, and liquor during the year prior to AMI.

Results Of the 1913 patients, 896 (47%) abstained from alcohol, 696 (36%) consumed less than 7 alcoholic drinks/wk, and 321 (17%) consumed 7 or more alcoholic drinks/wk. Compared with abstainers, patients who consumed less than 7 drinks/wk had a lower all-cause mortality rate (3.4 vs 6.3 deaths per 100 person-years; hazard ratio [HR], 0.55; 95% confidence interval [CI], 0.43-0.71) as did those who consumed 7 or more drinks/wk (2.4 vs 6.3 deaths per 100 person-years; HR, 0.38; 95% CI, 0.25-0.55; P<.001 for trend). After adjusting for propensity to drink and other potential confounders, increasing alcohol consumption remained predictive of lower mortality for less than 7 drinks/wk, with an adjusted HR of 0.79 (95% CI, 0.60-1.03), and for 7 or more drinks/wk, with an adjusted HR of 0.68 (95% CI, 0.45-1.05; P=.01 for trend). The association was similar for total and cardiovascular mortality, among both men and women, and among different types of alcoholic beverages.

Conclusion Self-reported moderate alcohol consumption in the year prior to AMI is associated with reduced mortality following infarction.


See also pp 1971 and 2004 and Patient Page.
centers. Between August 1989 and September 1994, 1935 patients (601 women and 1334 men) were interviewed a median of 4 days after having an AMI. Trained research interviewers identified eligible patients by reviewing coronary care unit admission logs and patient charts. For inclusion, patients were required to have a creatine kinase level higher than the upper limit of normal for each center, positive MB isoenzymes, an identifiable onset of AMI symptoms, and ability to complete a structured interview. We excluded patients with missing information about usual alcohol consumption (n=5) and those with a history of alcoholism who reported current abstinence (n=17), leaving 1913 patients eligible for analysis. The institutional review board of each center approved this protocol, and all participants provided informed consent.

Interviewers used a structured data abstraction and questionnaire form. Participants reported average frequency during the past year of consumption (and corresponding numbers of drinks) of wine, beer, and liquor individually. Because the average ethanol content of a typical drink differs by beverage type (ie, 13.2 g for beer, 10.8 g for wine, and 15.1 g for liquor), we determined each patient’s average weekly ethanol consumption from wine, beer, and liquor. We defined a standard serving of alcohol as 15.0 g of ethanol and categorized average alcohol consumption as none, less than 7 drinks/wk (<105 g of ethanol), or ≥7 or more drinks/wk (≥105 g of ethanol). We classified the 2 groups of drinkers as light and moderate drinkers, respectively. For beverage-specific analyses, we considered patients to be drinkers of a single type of alcoholic beverage (wine, beer, or liquor) if the other 2 beverages each represented less than 5% of the total number of drinks.

Other information collected included age, sex, medical history, and prescription and nonprescription medication use. During the chart review, interviewers recorded complications of congestive heart failure or ventricular arrhythmias based on clinical diagnoses documented in medical records, as well as blood pressure on admission and all creatine kinase values available at the time of chart review (median number of values = 4).

We defined initial hypotension and hypertension as systolic blood pressure on admission of less than 90 mm Hg and more than 200 mm Hg, respectively. We defined current aspirin use as the reported use of any aspirin or aspirin-containing product in the 4 days prior to the index AMI. We used 1990 US census data to derive median household income from ZIP codes (available for 1857 patients).15 We defined noncardiac comorbidity as any diagnosis of cancer, respiratory disease, renal failure, or stroke recorded in medical records.

We searched the National Death Index for deaths of Onset study participants through December 31, 1995, and requested death certificates from state offices of vital statistics records for all probable matches using a previously validated algorithm.16 Three physicians independently verified the determination of each death. Two physicians categorized the cause of each death as cardiovascular disease or noncardiovascular disease. Disagreements among raters were resolved by discussion. All-cause mortality was the primary outcome measure in all analyses, and cardiovascular mortality was a secondary outcome measure.

Propensity Score
To control for differences between groups of patients in factors other than alcohol consumption, we calculated propensity scores.37 Each patient’s score represents that individual’s probability of consuming a given amount of alcohol, relative to abstention, based on other demographic, behavioral, and clinical characteristics. We created 2 sets of models: those that compared abstainers with light drinkers (excluding moderate drinkers) and those that compared abstainers with moderate drinkers (excluding light drinkers). To do this, we used multivariable logistic regression models in which the dependent variable was light or moderate alcohol consumption, defined dichotomously, and the independent variables were covariates and interaction terms that could influence the probability of consuming a given amount of alcohol. Specifically, we included age and body mass index (as linear and quadratic terms), sex, previous MI, previous congestive heart failure, previous angina, diabetes mellitus, hypertension, noncardiac comorbidity, previous medication use (aspirin, β-blockers, calcium channel blockers, digoxin, and angiotensin-converting enzyme inhibitors individually), current/previous smoking, frequency of exertion (in 3 categories), household income (in quartiles), education (in 3 categories), and interaction terms of sex with age and race. The area under the receiver operating characteristic curve was 0.72 for the light-drinker model and 0.81 for the moderate-drinker model, indicating good discrimination between drinkers and abstainers.

We incorporated the actual propensity score into subsequent models, although we obtained similar results when we used indicator variables for quintiles or deciles of the propensity score.

In the propensity regression analysis, we assigned indicator variables to patients with missing data on education (n = 58) and household income (n = 56). For all other variables, we assigned individuals with missing covariate information (n ≤23 for any variable) mean levels of continuous covariates and modal levels of binary covariates. Models that deleted individuals with any missing covariate information (n = 177) yielded similar results (data not shown).

Statistical Analysis
We analyzed continuous and binary variables using t tests and Fisher exact tests, respectively. We compared unadjusted Kaplan-Meier survival plots using the log-rank test. We used Cox proportional hazards models to examine the independent effect of alcohol use on mortality. In these models, we controlled for the actual propensity score, age, sex, and measures of index AMI treatment and severity (peak creatine kinase level, receipt of thrombolytic therapy, and congestive heart failure and ventricular tachycardia during...
hospitalization). Models that incorporated the covariates used in the propensity regression analysis directly into Cox models (without use of a propensity score) with indicator variables for the 2 levels of alcohol consumption gave similar results (data not shown). We additionally controlled for Q-wave vs non-Q-wave AMI, initial hypotension, and initial hypertension (with indicator variables for missing information) in sensitivity analyses. The smaller numbers of patients in beverage-specific models precluded adjustment for the entire group of covariates; the truncated (adjusted for age, sex, smoking, and previous MI) and complete models gave similar results in analyses of the complete data set.

We repeated adjusted analyses within strata of covariates to explore possible effect modification. We tested hazard ratios (HRs) for linear trend across categories of alcohol consumption using a random-effects model.18 We tested the proportionality of hazards using time-varying covariates and found no significant violations. We present HRs from Cox models with 95% confidence intervals (CIs). All probability values presented are 2-sided.

RESULTS

Patient Characteristics

Characteristics of Onset study participants according to alcohol consumption are shown in Table 1.14 Of the 1913 patients, 896 (47%) reported abstinence in the year prior to their MI, 696 (36%) reported consumption of less than 7 drinks/wk, and 321 (17%) reported consumption of 7 or more drinks/wk. The median consumption in the heavier consumption group was 15 drinks/wk. Higher alcohol consumption was associated with younger age, male sex, current/former smoking, increased physical exertion, more frequent use of thrombolytic therapy, higher household income, higher educational attainment, and white race. It was inversely associated with hypertension, diabetes, previous CHD, use of calcium channel blockers and angiotensin-converting enzyme inhibitors, and congestive heart failure during index hospitalization.

Alcohol Consumption and Mortality

The figure shows unadjusted survival according to average weekly alcohol consumption. Survival was lowest among abstainers and greatest among patients who consumed 7 or more drinks/wk (P<.001).

Table 2 shows HRs for all-cause mortality according to average weekly alcohol consumption. Alcohol consumption was associated with lower mortality in unadjusted and adjusted models. Consumption of 7 or more drinks/wk was associated with somewhat lower mortality than consumption of less than 7 drinks/wk (HR, 0.83; 95% CI, 0.54-1.28). Table 2 also shows the adjusted HRs for mortality from cardio-

<table>
<thead>
<tr>
<th>Average Alcohol Consumption, Drinks/wk</th>
<th>&lt;7 (n = 696)</th>
<th>≥7 (n = 321)</th>
<th>P Value†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean (SD), y</td>
<td>65 (12)</td>
<td>60 (12)</td>
<td>57 (12)</td>
</tr>
<tr>
<td>Female sex</td>
<td>390 (44)</td>
<td>173 (25)</td>
<td>32 (10)</td>
</tr>
<tr>
<td>White race</td>
<td>788 (89)</td>
<td>637 (93)</td>
<td>292 (92)</td>
</tr>
<tr>
<td>Income, $‡</td>
<td>36 641 (12 232)</td>
<td>39 748 (13 883)</td>
<td>40 063 (13 251)</td>
</tr>
<tr>
<td>Education</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Less than high school‡</td>
<td>263 (31)</td>
<td>137 (20)</td>
<td>48 (15)</td>
</tr>
<tr>
<td>Completed high school</td>
<td>379 (44)</td>
<td>276 (40)</td>
<td>115 (37)</td>
</tr>
<tr>
<td>Some college</td>
<td>217 (25)</td>
<td>270 (40)</td>
<td>151 (48)</td>
</tr>
<tr>
<td>Body mass index, mean (SD), kg/m²</td>
<td>27.6 (5.7)</td>
<td>27.1 (4.7)</td>
<td>26.9 (4.5)</td>
</tr>
<tr>
<td>Smoking status</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current</td>
<td>253 (29)</td>
<td>242 (35)</td>
<td>132 (41)</td>
</tr>
<tr>
<td>Former</td>
<td>341 (38)</td>
<td>307 (44)</td>
<td>145 (45)</td>
</tr>
<tr>
<td>Morbidity</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Hypertension</td>
<td>433 (48)</td>
<td>290 (42)</td>
<td>122 (38)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>238 (27)</td>
<td>101 (15)</td>
<td>20 (6)</td>
</tr>
<tr>
<td>Previous myocardial infarction</td>
<td>293 (33)</td>
<td>177 (26)</td>
<td>70 (22)</td>
</tr>
<tr>
<td>Angina</td>
<td>268 (30)</td>
<td>151 (22)</td>
<td>64 (20)</td>
</tr>
<tr>
<td>Previous congestive heart failure</td>
<td>56 (6)</td>
<td>24 (3)</td>
<td>4 (1)</td>
</tr>
<tr>
<td>Regular medication use</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Angiotensin-converting enzyme inhibitors</td>
<td>133 (15)</td>
<td>62 (9)</td>
<td>30 (9)</td>
</tr>
<tr>
<td>Aspirin</td>
<td>274 (31)</td>
<td>257 (37)</td>
<td>111 (35)</td>
</tr>
<tr>
<td>β-Blockers</td>
<td>199 (22)</td>
<td>128 (18)</td>
<td>61 (19)</td>
</tr>
<tr>
<td>Calcium channel blockers</td>
<td>256 (29)</td>
<td>149 (21)</td>
<td>62 (19)</td>
</tr>
<tr>
<td>Digoxin</td>
<td>82 (9)</td>
<td>50 (7)</td>
<td>11 (3)</td>
</tr>
<tr>
<td>Index hospitalization</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thrombolytic use</td>
<td>263 (29)</td>
<td>280 (40)</td>
<td>142 (44)</td>
</tr>
<tr>
<td>Initial systolic blood pressure &lt;90 mm Hg</td>
<td>23 (3)</td>
<td>22 (3)</td>
<td>9 (3)</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>146 (16)</td>
<td>106 (15)</td>
<td>29 (9)</td>
</tr>
<tr>
<td>Ventricular tachycardia</td>
<td>91 (10)</td>
<td>100 (14)</td>
<td>44 (14)</td>
</tr>
<tr>
<td>Q-wave myocardial infarction¶</td>
<td>261 (54)</td>
<td>230 (60)</td>
<td>97 (55)</td>
</tr>
<tr>
<td>Peak creatine kinase level, U/L</td>
<td>918 (838)</td>
<td>1102 (1037)</td>
<td>1074 (943)</td>
</tr>
<tr>
<td>Exertion¶</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;1</td>
<td>786 (88)</td>
<td>538 (77)</td>
<td>237 (74)</td>
</tr>
<tr>
<td>1-4</td>
<td>48 (5)</td>
<td>97 (14)</td>
<td>46 (14)</td>
</tr>
<tr>
<td>&gt;4</td>
<td>62 (7)</td>
<td>61 (9)</td>
<td>38 (12)</td>
</tr>
</tbody>
</table>

*Data are No. (%) unless otherwise noted.
†P values for binary and continuous variables were calculated using the Fisher exact test and analysis of variance, respectively.
‡Household income data were derived from ZIP codes according to 1990 US census data for 1857 participants.
§Education data were available for 1855 participants.
¶Income data were derived from ZIP codes according to 1990 US census data for 1857 participants.
| One metabolic equivalent is equal to the energy expended per minute by a sitting adult and is 3.5 mL of oxygen uptake per kilogram of body weight per minute. We measured exertion by the number of times per week (<1, 1-4, or >4) participants exerted 6 or more METs.

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vascular causes. Alcohol consumption was also inversely associated with cardiovascular mortality, again with somewhat lower mortality among heavier drinkers than among lighter drinkers.

**Sensitivity Analyses**

To evaluate the robustness of our findings, we repeated our adjusted analyses within prespecified patient subgroups. Among men, the adjusted HRs for all-cause mortality were 0.83 (95% CI, 0.60-1.15) and 0.76 (95% CI, 0.48-1.19) among patients who consumed less than 7 or 7 or more drinks/wk, respectively. The corresponding HRs for women were 0.64 (95% CI, 0.38-1.07) and 0.37 (95% CI, 0.04-3.21). These results were similar but more precise when modeled as indicator variables in stratified Cox analyses, rather than in propensity analyses. We also found similar associations between alcohol consumption and survival among patients with a first or recurrent MI, with or without diabetes, with or without hypertension, and with incomes higher or lower than the mean for this study.

Models that additionally controlled for initial hypotension, initial hypertension, and presence of Q-wave (vs non-Q-wave) AMI did not materially change our results. Our results were also unchanged when we excluded hypertension as a covariate in the full model and when we excluded patients with non-cardiac comorbidity. Finally, we examined the effect of alcohol consumption among patients who consumed 1 type of alcoholic beverage exclusively, although our power was limited by a smaller number of patients (Table 3). The effect of alcohol consumption was generally similar among the 3 beverage types.

**COMMENT**

In this prospective cohort study of early survivors of AMI, moderate alcohol consumption during the prior year, as measured at the time of index AMI, was associated with lower subsequent mortality following AMI. This association was present in unadjusted and adjusted analyses, extended to both men and women, and was similar for cardiovascular and all-cause mortality.

We know of no prior studies that specifically studied alcohol use and mortality immediately following AMI. Muntwyler et al studied the mortality of male physicians who reported history of MI and found that moderate drinkers had a relative risk of death of 0.7, a result that corresponds well to our findings. However, those men survived their MIs long enough to be considered for enrollment in the Physicians’ Health Study. Hence, the authors could not exclude the possibility of differential mortality between drinkers and abstainers after MI but prior to consideration for enrollment in the study. Because we specifically included early survivors of AMI in the Onset study, our results argue against that possibility; in addition, we extend their findings to women and patients of lower socioeconomic status.

Shaper and Wannamethee, in a study of 455 men, found no association between consumption of 1 to 14 drinks/wk and survival in men with history of MI. However, the 95% CIs for the effect of alcohol consumption were wide (0.77-1.56) and compatible with the results of this study. Also, moderate drinkers were previously found to have a lower case-fatality rate of AMI than abstainers in the same population, raising the possibilit-
ity that abstainers at the highest risk of death may have died before entry into that analysis.

In exploratory analyses, we found no substantial difference in survival among patients who reported predominant consumption of wine, beer, or liquor. Although some studies have found that wine consumption is associated with a lower risk of CHD than is consumption of other alcoholic beverages,20,21 a systematic review found no consistent effect of beverage type on CHD.22 Indeed, studies in countries where wine consumption is uncommon report similar results to studies done elsewhere.23,24 Several physiological effects of alcohol could possibly explain our findings. In experimental studies, alcohol consumption increases high-density lipoprotein cholesterol (HDL-C) levels25,26 and decreases levels of prothrombotic factors, particularly fibrinogen.27,28 Alcohol also reduces platelet aggregability and ablates cyclic flow reductions in mechanically stenosed canine coronary arteries.29,30 Other potentially beneficial effects of alcohol include inhibition of ischemic-induced arrhythmias31 and lowering of pulmonary artery pressure among patients with congestive heart failure.32

Whether the observed association between alcohol consumption and post-AMI survival reflects the physiological effects of alcohol or confounding by other factors associated with alcohol can only be answered in a long-term randomized trial, although such a trial is unlikely to be performed in the near future.

**Study Limitations**

As with any observational study, the associations we observed could be accounted for, at least in part, by differences between alcohol consumers and abstainers. For example, alcohol consumers tend to engage in more physical activity and have higher socioeconomic status than abstainers.33 In fact, the association between alcohol consumption and lower mortality was substantially attenuated after adjusting for an alcohol propensity score that incorporates measures of physical activity and socioeconomic status (Table 2). Despite our inclusion of frequency of physical exertion, educational attainment, race, and median household income by ZIP code of residence in the propensity score, residual confounding by these factors (as well as by unmeasured factors) may exist. Some abstainers may be former drinkers who quit because of illness. However, several studies have shown that former drinkers and lifelong abstainers have similarly elevated risks of CHD,13,14 and we excluded patients who reported history of alcohol abuse and had become abstainers. Our results also remained robust when we excluded patients with noncardiac comorbidity.

While controlling for possible confounding in our analyses, we may have overadjusted for some covariates that are actually intermediates in the causal pathway between alcohol consumption and lower mortality. For example, if alcohol consumption lowers the risk of diabetes,35 which, in turn, lowers long-term mortality following AMI, then adjusting for diabetes may have caused us to underestimate the actual survival difference associated with alcohol consumption. However, given the important concerns about confounding in observational studies of alcohol use, we believe this conservative approach is most appropriate.

We asked patients to report their usual alcohol consumption prior to the AMI that resulted in their hospitalization. If pre-AMI and post-AMI drinking patterns do not correlate well, the true effect of post-AMI alcohol consumption on survival could be different than that reported herein. However, among 711 men who had an AMI in the Health Professionals Follow-Up Study, the correlation between alcohol consumption before and after AMI was high (\(r=0.79\)), while the median absolute change in consumption was minimal (Eric B. Rimm, ScD, written communication, March 2000). Similarly, alcohol consumption following AMI was similar to, but lower than, consumption prior to AMI among Dutch men.36 Our results could also be influenced by inaccuracies in identification of deaths among Onset study participants. However, we used a validated method to search the National Death Index,16 and 3 physicians blinded to alcohol consumption confirmed each death.

Our power to evaluate heavier alcohol consumption was limited. For example, only 107 patients reported consumption of 21 or more drinks/wk, of whom only 8 died. Hence, we could not assess these 107 patients as a separate subgroup and cannot generalize our results to individuals who consume that quantity of alcohol or more.

Because individuals tend to under-report alcohol intake,37 the actual number of servings that Onset study participants consumed may differ from the values reported here. However, such underreporting is unlikely to affect the rank order of alcohol consumption among Onset study participants and, therefore, should not affect the internal validity of our results.

Although we only interviewed early survivors of AMI who could complete a structured interview, it is possible that AMI severity may have produced differential recall of usual alcohol consumption. However, we have previously shown that neither recent nor habitual alcohol consumption influences AMI severity in this population.38 Moreover, in a subset of 115 Onset study participants who had HDL-C levels measured during hospitalization, estimated alcohol consumption and HDL-C level were correlated to the same degree found in the Second National Health and Nutrition Examination Survey (Pearson \(r=0.21; P=0.02\), confirming the validity of our instrument.39

We do not have information on how Onset study participants may have changed their smoking habits following hospitalization. Numerous studies link heavier alcohol consumption to lower success in smoking cessation and greater likelihood of relapse among ex-smokers.40 If alcohol consumption mediates these smoking behaviors directly, lack of control for differences in posthospitalization smoking habits is inappropriate. If not, confounding by changes in smoking habits may actually have led us to underestimate the true difference between drinkers and abstainers, because
abstainers will tend to disproportionately become and remain nonsmokers.

**Conclusions**

Our results have 2 somewhat different implications for clinical practice. First, our results provide prognostic information for patients who survive an AMI. Adults who abstained from alcohol prior to AMI appeared to be at particularly high risk of long-term mortality; specialized strategies for secondary prevention may be appropriate for these individuals. Second, our results are consistent with the hypothesis that light or moderate alcohol use following AMI is safe, although studies that formally assess post-MI consumption are needed to confirm this. Even given the associations demonstrated in this study, individuals should continue to consult their physicians regarding the advisability of consuming alcohol following an AMI. Determination of the risks and benefits of alcohol consumption for an individual requires consideration of numerous personal, clinical, and social factors that cannot be addressed with aggregate-level observational data such as ours.

In summary, we found that Onset study participants who reported alcohol consumption prior to AMI had lower mortality following AMI than abstainers. This finding was consistent across patient subgroups and beverage types, was similar for cardiovascular and all-cause mortality, and persisted even after controlling for potentially confounding factors in a rigorous propensity analysis. Thus, taken together, the overall epidemiological evidence suggests that moderate alcohol consumption is associated with a lower risk of AMI and a lower risk of long-term mortality following AMI.

**Author Contributions:** Study concept and design: Mukamal, Maclure, Muller, Sherwood, Mittleman. Acquisition of data: Maclure, Sherwood, Mittleman. Analysis and interpretation of data: Mukamal, Maclure, Mittleman. Drafting of the manuscript: Mukamal, Mittleman. Critical revision of the manuscript for important intellectual content: Maclure, Muller, Sherwood, Mittleman. Statistical expertise: Mukamal, Maclure, Mittleman. Obtained funding: Maclure, Muller, Sherwood, Mittleman. Administrative, technical, or material support: Maclure, Muller, Sherwood, Mittleman.

**Study supervision:** Sherwood, Mittleman.

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**REFERENCES**