Moderate Alcohol Consumption and the Risk of Sudden Cardiac Death Among US Male Physicians

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Background—Individuals who consume high amounts of alcohol (>5 drinks/d) have increased risks of ventricular arrhythmia and sudden cardiac death (SCD). However, the relationship is less clear for drinkers of light-to-moderate amounts.

Methods and Results—We prospectively assessed whether light-to-moderate alcohol drinkers have a decreased risk of SCD among 21,537 male participants in the Physicians Health Study who were free of self-reported cardiovascular disease and provided complete information on alcohol intake at study entry. Over 12 years of follow-up, 141 SCDs were confirmed. After control for multiple confounders, men who consumed 2 to 4 drinks/wk (RR = 0.40; 95% CI, 0.22 to 0.75; P = 0.004) or 5 to 6 drinks/wk (RR = 0.21; 95% CI, 0.08 to 0.56; P = 0.002) at baseline had significantly reduced risks of SCD compared with those who rarely or never consumed alcohol. The relationship for SCD was U-shaped (P = 0.002), with the risk approaching unity at ≥2 drinks/d. In contrast, the relationship of alcohol intake and nonsudden CHD death was L-shaped or linear (P for trend = 0.02).

Conclusions—In these prospective data, men who consumed light-to-moderate amounts of alcohol (2 to 6 drinks/wk) had a significantly reduced risk of SCD compared with those who rarely or never consumed alcohol. (Circulation. 1999;100:944-950.)

Key Words: alcohol | death, sudden | arrhythmia | coronary disease | epidemiology

Sudden cardiac death (SCD) accounts for 250,000 deaths in the United States every year1; therefore, identification of modifiable risk factors for SCD continues to be an important public health goal. One modifiable risk factor, alcohol consumption, may have a differential effect on the risk of sudden versus nonsudden coronary heart disease (CHD) death.2 It is well established that heavy alcohol consumption (>5 drinks/d) is associated with an increased risk of SCD3,4; however, the results of studies addressing light-to-moderate alcohol consumption and SCD have not been consistent. Prospective studies have found either no5-7 or a positive8 association, whereas case-control studies have reported inverse linear associations.9-11 In men, SCD is associated with underlying CHD in >90% of cases,12 and because light-to-moderate alcohol consumption has been associated with lower risks of other CHD end points,13-19 we hypothesized that men who drink light-to-moderate amounts of alcohol would have a lower risk of SCD. The Physicians’ Health Study of 22,071 apparently healthy men followed up for an average of 12 years presented a unique opportunity to explore whether individuals who consume light-to-moderate amounts of alcohol have a lower risk of SCD and whether risks differ for SCD compared with other forms of CHD death.

Methods
The methods of the Physicians’ Health Study have been described in detail elsewhere.20 Briefly, 22,071 male physicians who were 40 to 84 years old in 1982 and had no history of myocardial infarction (MI), stroke, transient ischemic attacks, and cancer were assigned to aspirin and/or b-carotene in a randomized, double-blind, placebo-controlled 2×2 factorial design. At baseline, the physicians completed questions on health status, risk factors for cardiovascular disease (CVD), alcohol consumption, antioxidant vitamin use, dietary intake of selected foods, and frequency of vigorous exercise. At baseline and at 84 months of follow-up, the physicians were asked, “How often do you usually consume alcoholic beverages (beer, wine, or liquor)?” The 7 possible response categories (2+1/d, daily, 5 to 6/wk, 2 to 4/wk, 1/wk, 1 to 3/mo, and never/rare) were interpreted as the number of drinks consumed per unit of time.

Population for Analysis
Because the development of preclinical CVD symptoms may influence the reporting of alcohol consumption, we chose to exclude from the primary analyses men who reported nonfatal CVD endpoints before ascertainment of alcohol intake. A total of 534 men who reported angina or coronary revascularization before randomization

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or who had missing data on alcohol consumption were excluded, leaving 21,537 participants for the baseline analysis. Participants contributed follow-up time from study entry to date of death or to the scheduled end of the randomized β-carotene component on December 31, 1995, whichever came first.

**Endpoint Ascertainment and Definitions**

Information on cardiovascular events was updated every 6 months for the first year and annually thereafter. The ascertainment of CVD events was by self-report on follow-up questionnaires, and deaths were generally reported by postal authorities or next of kin. All such events were reviewed by an Endpoints Committee of physicians for confirmation by medical records obtained from hospitals and attending physicians. The next of kin were interviewed regarding the circumstances surrounding the death if not adequately documented in the medical record. Deaths in which there was evidence of CHD at or before death and in which a noncoronary cause of death was not found were classified as CHD deaths (ICD-9 codes 410 to 414). Cases of nonfatal MI were confirmed by use of the World Health Organization criteria.

To ascertain the specific end point of SCD, medical records and reports from next of kin for all cardiovascular deaths (excluding strokes) were rereviewed by 2 cardiologists unaware of exposure status, and agreement was reached. SCD was defined as death within 1 hour of symptom onset and/or a witnessed cardiac arrest or abrupt collapse not preceded by more than 1 hour of symptoms that precipitated the terminal event. Information from the death certificate was not used in the determination of the timing of the death. To increase our specificity for “arrhythmic death,” we excluded anyone who had evidence of collapse of the circulation (hypotension, exacerbation of congestive heart failure, and/or altered mental status) before the disappearance of the pulse.22

Unwitnessed deaths with no information on timing but with an autopsy consistent with arrhythmic cardiac death (ie, acute coronary thrombosis or severe coronary artery disease without myocardial necrosis or other pathological findings to explain death) were considered possible SCDs, and the analysis was performed both including and excluding these deaths. Deaths (often unwitnessed) in which the timing could not be accurately determined from the available information were not classified as sudden or nonsudden.

**Statistical Analysis**

Age-adjusted means or proportions of baseline risk factors and treatment group assignment were computed for the 7 levels of reported alcohol consumption. The significance of associations was tested by the Mantel-Haenszel $\chi^2$ test for trend for categorical variables and linear regression for continuous variables. Relative risks (RRs) were computed with Cox proportional hazards models,23 controlling for age and randomized aspirin and β-carotene assignment. A multivariate Cox proportional hazards model was used to control for potential confounders simultaneously (see legend of Table 2). Participants with missing data (3.5%) on covariates included in the multivariate model were excluded from analysis. Because fish consumption was ascertained at 12 months and was generally reported by postal authorities or next of kin. All such events were reviewed by an Endpoints Committee of physicians for confirmation by medical records obtained from hospitals and attending physicians. The next of kin were interviewed regarding the circumstances surrounding the death if not adequately documented in the medical record. Deaths in which there was evidence of CHD at or before death and in which a noncoronary cause of death was not found were classified as CHD deaths (ICD-9 codes 410 to 414). Cases of nonfatal MI were confirmed by use of the World Health Organization criteria.

To make the relationship of alcohol intake at baseline with subsequent risk of SCD is displayed in Figure 1. The relationship between moderate alcohol consumption and SCD was U-shaped in both age- and treatment-adjusted as well as multivariate analyses. The nadir was apparent at 5 to 6 drinks/wk, and the risk again approached unity at ≥2 drinks/d (Table 2). Participants who consumed 2 to 4 drinks/wk had a 60% reduced risk of SCD ($P=0.004$), and those who consumed 5 to 6 drinks/wk had a 79% reduced risk of SCD ($P=0.002$) compared with those who rarely or never consumed alcohol. The RRs in both these categories of light-to-moderate consumption were also significantly reduced compared with those who consumed ≥2 drinks/d. To assess the linear and nonlinear relations between alcohol intake and SCD, we performed analyses in which a continuous variable for alcohol (created by assigning the midpoint value to each response category) was entered into the multivariate model as both a linear and a quadratic term. There was no evidence for a linear trend when the linear term was entered alone ($P=0.43$). However, when the quadratic term was entered into the model to test for nonlinear trend, the results were significant ($P=0.002$), consistent with the observed U-shaped relation.

For SCD, we also performed secondary analyses using baseline alcohol exposure but excluding deaths in the first 4 years of follow-up to address the possibility that undiagnosed, preexisting CVD could have been present in these men at the time of study entry. In addition, to address potential misclassification of intake due to changing alcohol consumption over time, we repeated the analysis using the updated measure of exposure at 7 years in a time-varying covariate Cox model. The other covariates were also updated at 7 years with the most recently reported information. To maintain a population free of CVD at the time alcohol intake was assessed, 3093 participants who reported nonfatal CVD end points before ascertainment of alcohol consumption on the 84-month questionnaire were excluded from the time-varying analysis at 84 months.

**Results**

**Alcohol Intake**

The alcohol intake of the 21,537 participants was fairly evenly distributed across categories, with the exception of the highest. Only 667 men (3.1%) reported consuming ≥2 drinks/d. Table 1 shows age-adjusted baseline risk factors according to level of alcohol intake. Baseline alcohol was directly associated with age, smoking, hypertension, physical activity, and fish consumption (ascertained at 12 months), as well as inversely with diabetes and antioxidant vitamin use. At 84 months, 32.7% of the cohort had decreased their alcohol intake by at least 1 category, and 15.1% increased their level. The proportion of participants who reported a change in their alcohol intake at 84 months did not differ significantly between the participants who had previously reported nonfatal CVD and those who had not.

**Sudden Cardiac Deaths**

Over 12 years, 141 SCDs (121 definite and 20 probable) were documented. The relationship of alcohol intake at baseline with subsequent risk of SCD is displayed in Figure 1. The relationship between moderate alcohol consumption and SCD was U-shaped in both age- and treatment-adjusted as well as multivariate analyses. The nadir was apparent at 5 to 6 drinks/wk, and the risk again approached unity at ≥2 drinks/d (Table 2). Participants who consumed 2 to 4 drinks/wk had a 60% reduced risk of SCD ($P=0.004$), and those who consumed 5 to 6 drinks/wk had a 79% reduced risk of SCD ($P=0.002$) compared with those who rarely or never consumed alcohol. The RRs in both these categories of light-to-moderate consumption were also significantly reduced compared with those who consumed ≥2 drinks/d. To assess the linear and nonlinear relations between alcohol intake and SCD, we performed analyses in which a continuous variable for alcohol (created by assigning the midpoint value to each response category) was entered into the multivariate model as both a linear and a quadratic term. There was no evidence for a linear trend when the linear term was entered alone ($P=0.43$). However, when the quadratic term was entered into the model to test for nonlinear trend, the results were significant ($P=0.002$), consistent with the observed U-shaped relation.

Although we excluded men with reported CVD at baseline, the subgroup who rarely or never consumed alcohol could include men who refrained from drinking because of early symptoms of CVD. If so, the higher SCD rate in this group could be due, at least in part, to undiagnosed preexisting
disease. To address this issue, we repeated our analyses after excluding deaths in the first 4 years of follow-up. The multivariate RRs from this analysis and their 95% CIs are displayed in Table 3. The U-shaped relationship defined by the multivariate RRs were essentially unchanged after the first 4 years of events were excluded.

Because ≈50% of this cohort increased or decreased their level of intake over time, we repeated the multivariate analysis using the updated measure of alcohol intake ascertained at year 7 to address potential misclassification in the baseline analyses. The multivariate RRs and 95% CIs from the time-varying analyses are displayed in Table 3. The

**TABLE 1. Relationship of Alcohol Consumption to Coronary Heart Disease Risk Factors at Baseline***

<table>
<thead>
<tr>
<th>Variable</th>
<th>&lt;1/mo</th>
<th>1–3/mo</th>
<th>1/wk</th>
<th>2–4/wk</th>
<th>5–6/wk</th>
<th>Daily</th>
<th>≥2/d</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of men</td>
<td>3190</td>
<td>2396</td>
<td>3012</td>
<td>4847</td>
<td>2735</td>
<td>4690</td>
<td>667</td>
</tr>
<tr>
<td>Age, y (mean)</td>
<td>54.0</td>
<td>51.6</td>
<td>51.4</td>
<td>51.8</td>
<td>52.6</td>
<td>55.3</td>
<td>55.7</td>
</tr>
<tr>
<td>Smoking, %</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥20 cigarettes/d</td>
<td>5.6</td>
<td>7.1</td>
<td>5.9</td>
<td>5.8</td>
<td>5.7</td>
<td>9.9</td>
<td>17.6</td>
</tr>
<tr>
<td>&lt;20 cigarettes/d</td>
<td>2.3</td>
<td>3.6</td>
<td>3.1</td>
<td>3.6</td>
<td>5.0</td>
<td>4.8</td>
<td>5.3</td>
</tr>
<tr>
<td>Past</td>
<td>24.0</td>
<td>33.1</td>
<td>35.5</td>
<td>41.3</td>
<td>45.6</td>
<td>47.4</td>
<td>49.3</td>
</tr>
<tr>
<td>Never</td>
<td>68.1</td>
<td>56.2</td>
<td>55.6</td>
<td>49.3</td>
<td>43.8</td>
<td>37.9</td>
<td>27.9</td>
</tr>
<tr>
<td>Reported diagnosis of, %</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes</td>
<td>3.4</td>
<td>3.8</td>
<td>2.7</td>
<td>1.5</td>
<td>1.3</td>
<td>1.9</td>
<td>1.6</td>
</tr>
<tr>
<td>High cholesterol†</td>
<td>5.6</td>
<td>5.6</td>
<td>6.2</td>
<td>5.7</td>
<td>5.9</td>
<td>6.4</td>
<td>6.4</td>
</tr>
<tr>
<td>Hypertension‡</td>
<td>13.0</td>
<td>14.1</td>
<td>13.0</td>
<td>12.7</td>
<td>14.1</td>
<td>14.1</td>
<td>17.3</td>
</tr>
<tr>
<td>Vigorous exercise (≥1×wk), %</td>
<td>68.1</td>
<td>65.4</td>
<td>71.0</td>
<td>75.8</td>
<td>76.4</td>
<td>74.2</td>
<td>66.1</td>
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<tr>
<td>Body mass index, kg/m² (mean)</td>
<td>25.0</td>
<td>25.2</td>
<td>25.2</td>
<td>24.9</td>
<td>24.7</td>
<td>24.6</td>
<td>25.0</td>
</tr>
<tr>
<td>Parental history of MI before age 60 y, %</td>
<td>11.8</td>
<td>12.6</td>
<td>13.4</td>
<td>13.7</td>
<td>13.1</td>
<td>13.2</td>
<td>9.4</td>
</tr>
<tr>
<td>Vitamin supplement use, %</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vitamin E</td>
<td>12.6</td>
<td>10.5</td>
<td>9.6</td>
<td>9.8</td>
<td>9.6</td>
<td>10.0</td>
<td>9.4</td>
</tr>
<tr>
<td>Vitamin C</td>
<td>25.0</td>
<td>23.9</td>
<td>22.3</td>
<td>22.5</td>
<td>22.9</td>
<td>22.5</td>
<td>22.0</td>
</tr>
<tr>
<td>Multivitamin</td>
<td>39.3</td>
<td>37.0</td>
<td>33.8</td>
<td>34.2</td>
<td>34.6</td>
<td>35.7</td>
<td>36.1</td>
</tr>
<tr>
<td>Fish intake, ≥1 servings/wk, %§</td>
<td>80.3</td>
<td>86.9</td>
<td>89.3</td>
<td>90.8</td>
<td>91.3</td>
<td>90.3</td>
<td>87.8</td>
</tr>
<tr>
<td>Treatment group, %</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aspirin</td>
<td>49.3</td>
<td>48.5</td>
<td>51.3</td>
<td>50.0</td>
<td>50.9</td>
<td>49.8</td>
<td>50.2</td>
</tr>
<tr>
<td>β-Carotene</td>
<td>49.6</td>
<td>49.0</td>
<td>51.2</td>
<td>49.3</td>
<td>51.9</td>
<td>49.5</td>
<td>49.0</td>
</tr>
</tbody>
</table>

*Standardized for age to the total cohort.
†Self-reported high cholesterol, cholesterol level ≥240 mg/dL, or on cholesterol-lowering medications.
‡Self-reported systolic blood pressure of ≥160 mm Hg, diastolic blood pressure of ≥90 mm Hg, or taking antihypertensive medication.
§Information ascertained on the 12-month questionnaire.

**Figure 1.** Age-adjusted and multivariate RR of SCD according to level of alcohol intake. Vertical bars represent 95% CI. *RRs that are significantly <1. See Table 2 legend for covariates used in multivariate model.
U-shaped relationship defined by the multivariate RRs persisted after the exposure was updated (P for the quadratic term=0.02) despite updating of potential confounders, even those that might be in the causal pathway.

To explore whether the effect of alcohol differed by mode of death (sudden versus nonsudden), we analyzed the relationship of baseline moderate alcohol consumption with nonsudden CHD death. The results of the age-adjusted and multivariate analysis involving the 157 cases of nonsudden CHD death. The lowest risks were found in those who consumed ≥2 drinks/d (RR, 0.50; 95% CI, 0.30 to 0.83), a level of consumption that was associated with an apparent increase in the RR for SCD.

**Discussion**

In this large, prospective cohort study of apparently healthy male physicians who were free of reported MI, stroke, or angina at study entry, alcohol consumption had a U-shaped association with SCD (P for quadratic term=0.002). Compared with those who rarely or never consumed alcohol, the nadir in risk occurred in those who reported consuming 5 to 6 drinks/wk (RR, 0.21; 95% CI, 0.80 to 0.56). The RR of SCD was also significantly decreased among those consuming 2 to 4 drinks/wk (RR, 0.40; 95% CI, 0.22 to 0.75). The RRs in both of these categories of light-to-moderate consumption were also significantly reduced compared with those who consumed ≥2 drinks/d. These results were similar

**TABLE 2. RR of SCD and Nonsudden CHD Death According to Level of Alcohol Intake**

<table>
<thead>
<tr>
<th>SCD</th>
<th>Average Frequency of Alcohol Intake at Baseline</th>
<th>P for Association</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Linear</td>
<td>Curvilinear</td>
</tr>
<tr>
<td>Cases (N=141)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age-adjusted RR</td>
<td>1.0</td>
<td>0.77</td>
</tr>
<tr>
<td>95% CI</td>
<td>Referent</td>
<td>0.43–1.39</td>
</tr>
<tr>
<td>Multivariate RR*</td>
<td>1.2</td>
<td>0.72</td>
</tr>
<tr>
<td>95% CI</td>
<td>Referent</td>
<td>0.39–1.33</td>
</tr>
</tbody>
</table>

**Nonsudden CHD Death**

| Cases (N=157) |       |             |       |             |
| Age-adjusted RR | 1.0  | 1.28        | 1.19  | 0.97        | 0.59  | 0.70        | 1.07  |
| 95% CI       | Referent | 0.73–2.24  | 0.69–2.04 | 0.59–1.62 | 0.30–1.15 | 0.43–1.16 | 0.47–2.43 | 0.07 | 0.07 |
| Multivariate RR* | 1.0 | 1.24        | 1.01  | 0.91        | 0.63  | 0.61        | 0.73  |
| 95% CI       | Referent | 0.70–2.22  | 0.62–1.93 | 0.52–1.57 | 0.32–1.25 | 0.36–1.03 | 0.03–1.80 | 0.02 | 0.18 |

*Multivariate model includes age (continuous), aspirin and β-carotene treatment assignment, body mass index (quartiles), smoking (current: <20 cigarettes/d, ≥20 cigarettes/d, past, never), history of diabetes, history of hypertension, history of hypercholesterolemia, vigorous exercise (<1×wk, 1–4×wk, ≥5×wk), and vitamin E, vitamin C, and multivitamin use at baseline and fish consumption at 12 months (<1×mo, 1–3×mo, daily, 2–4×wk, ≥5×wk).

**TABLE 3. Secondary Analyses of RR of SCD According to Level of Alcohol Intake**

<table>
<thead>
<tr>
<th>Average Frequency of Alcohol Intake</th>
<th>P for Association*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Linear</td>
</tr>
<tr>
<td>Excluding first 4 y of follow-up</td>
<td></td>
</tr>
<tr>
<td>Multivariate RR*</td>
<td>1.0</td>
</tr>
<tr>
<td>95% CI</td>
<td>Referent</td>
</tr>
<tr>
<td>Updating alcohol intake</td>
<td></td>
</tr>
<tr>
<td>Multivariate RR†</td>
<td>1.0</td>
</tr>
<tr>
<td>95% CI</td>
<td>Referent</td>
</tr>
</tbody>
</table>

*Multivariate model includes age (continuous), aspirin and β-carotene treatment assignment, body mass index (quartiles), smoking (current: <20 cigarettes/d, ≥20 cigarettes/d, past, never), history of diabetes, history of hypertension, history of hypercholesterolemia, vigorous exercise (<1×wk, 1–4×wk, ≥5×wk), and vitamin E, vitamin C, and multivitamin use at baseline and fish consumption at 12 months (<1×mo, 1–3×mo, daily, 2–4×wk, ≥5×wk).

†Multivariate model includes above and time-varying covariates for alcohol consumption, smoking, diabetes, hypertension, body mass index, and vigorous exercise.
when baseline alcohol intake was used alone, when this
measure was updated by the value obtained at 84 months of
follow-up, or when the first 4 years of SCDs were excluded
from the analysis.

Most prospective studies have suggested that moderate
alcohol intake was not associated with a reduction in the risk
of SCD despite observed reductions in the risk of other CHD
end points.5–7 The small numbers of SCDs in these previous
studies, the 50% of the number in this study, may have limited
the power to detect an association. In addition, previous
studies tested only for linear and not quadratic associations.
However, 1 prospective study from Finland reported a posi-
tive association between moderate alcohol intake (>200
grams/mo, >3 to 5 drinks/wk) and SCD.8 The disparate results of
their study might have been due, at least in part, to the Finnish
pattern of heavy consumption at less frequent settings per
week (binge drinking), which was probably uncommon in the
US physicians.

In contrast to previous prospective studies, retrospective
case-control studies have reported a reduced risk of SCD at
all levels of moderate alcohol intake.9–11 In the present
prospective cohort study, the risk of SCD was no longer
reduced among men who consumed >2 drinks/d. Our results
may be compatible with these case-control studies. The
open-ended highest intake category (>2 drinks/d) includes
both moderate and excessive drinking, and several studies
have documented the increased risk of SCD associated with
heavy alcohol consumption (>5 to 6 drinks/d).3,4 The ele-
vated risks in the highest intake category may have been
limited to the heavy drinkers and may not apply to more
moderate drinkers.

The results of previous prospective studies have raised the
concern that even low doses of alcohol may have an adverse
effect on arrhythmogenesis, thereby diminishing the magni-
tude of the beneficial effects of alcohol on atherogenesis and
possibly thrombosis, resulting in a neutral or increased risk of
SCD. Our data do not support this possibility. We found
reduced risks of SCD at consumption levels up to 1 drink/d,
suggesting that up to these levels, the arrhythmogenic effect
of alcohol is minimal and is outweighed by other beneficial
effects. In fact, although the confidence bounds were wide,
the odds ratio for sudden compared with nonsudden CHD
death among the men who died was significantly lower at 2
to 6 drinks/wk, suggesting an added benefit of moderate
alcohol consumption on SCD. This possible added benefit
against SCD could be mediated by the beneficial effects on
plaque rupture19 or thrombosis25 often documented on autopsies
of SCD victims26,27 or through effects on the autonomic
nervous system.28

In contrast to the findings for SCD, the relationship with
nonfatal MI and nonsudden CHD death was linear, without an
increase in RR at the highest level of intake. In addition, risks
are not significantly reduced for nonfatal MI until consump-
tion levels reach 1 drink/d, the same level at which risk for
SCD begins to increase. Presumably the differential effect on
SCD could be due to the superimposed effect of alcohol (perhaps
U- or J-shaped) on arrhythmogenesis on a background effect
on atherogenesis and thrombosis. Although it is difficult to
classify the mechanism of death, the definition of SCD used

<table>
<thead>
<tr>
<th>Average Frequency of Alcohol Intake</th>
<th>&lt;1/mo</th>
<th>1/wk</th>
<th>2–6/wk</th>
<th>≥1/d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total No. of deaths</td>
<td>63</td>
<td>82</td>
<td>68</td>
<td>85</td>
</tr>
<tr>
<td>Odds ratio*</td>
<td>1.0</td>
<td>0.54</td>
<td>0.38</td>
<td>0.99</td>
</tr>
<tr>
<td>95% CI Referent</td>
<td>0.24–1.22</td>
<td>0.16–0.91</td>
<td>0.43–2.25</td>
<td></td>
</tr>
<tr>
<td>P for association</td>
<td>0.13</td>
<td>0.03</td>
<td>0.97</td>
<td></td>
</tr>
</tbody>
</table>

*Multivariate model includes age (continuous), aspirin and β-carotene
treatment assignment, body mass index (quartiles), smoking (current: <20
cigarettes/d, ≥20 cigarettes/d, past, never), history of diabetes, history of
hypertension, history of hypercholesterolemia, vigorous exercise (<1×/wk,
1–4×/wk, ≥5×/wk), and vitamin E, vitamin C, and multivitamin use at
baseline and fish consumption at 12 months (<1×/mo, 1–3×/mo, daily,
2–4×/wk, ≥5×/wk).

Table 4. Relative Odds of Sudden Versus Nonsudden CHD
Death Among Men Who Died

Figure 2. Multivariate RR of nonfatal MI according
to level of alcohol intake. Vertical bars represent
95% CI. *RRs that are significantly <1. See Table
2 legend for covariates used in multivariate model.
SCD is the most common cause of death in adults <65 years old and therefore is a major public health problem in the United States and other industrialized countries. Because only 25% of out-of-hospital ventricular fibrillation arrest victims will survive to hospital discharge, any substantial reduction in the incidence of SCD will require effective preventive interventions. Further research directed at understanding the underlying mechanism by which alcohol may protect against SCD specifically and CHD in general could lead to the development of preventive therapeutics that have the benefits of alcohol without the accompanying risks.

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