Seven-Year Changes in Alcohol Consumption and Subsequent Risk of Cardiovascular Disease in Men

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Background: Few studies have examined whether changes in alcohol consumption influence future cardiovascular risk.

Objective: To examine whether 7-year changes in alcohol consumption are associated with the subsequent risk of cardiovascular disease (CVD).

Methods: We prospectively followed up 18,455 men aged 40 to 84 years from the Physicians’ Health Study with no history of CVD or cancer. Alcohol consumption was reported on the baseline and the 7-year questionnaires; follow-up for this analysis began after the 7-year questionnaire (median follow-up, 5.8 years). There were 10,911 CVD cases, including myocardial infarction, angina pectoris, revascularization, stroke, and CVD-related death.

Results: Among men initially consuming 1 drink per week or less (n=7,460), those with moderate increases (>1 to <6 drinks per week) in alcohol consumption had a borderline significant (P=.05) 29% reduced risk of CVD compared with men with no changes (−1 to 1 drink per week). Among men initially consuming greater than 1 to 6 drinks per week (n=6,612), those with moderate increases had a nonsignificant (P=.32) 15% decrease in CVD risk compared with men with no changes. Finally, among men initially consuming 1 drink per day or more (n=4,483), those who increased intake had a 63% increased risk of CVD compared with men with no changes.

Conclusions: These prospective data suggest that, among men with initially low alcohol consumption (≤1 drink per week), a subsequent moderate increase in alcohol consumption may lower their CVD risk. The possible reduction in CVD risk from increasing alcohol intake did not extend to men initially consuming greater than 1 drink per week. Given the potential risks and benefits associated with alcohol consumption, physician counseling of patients must be individualized in the context of the primary prevention of CVD.

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SUBJECTS AND METHODS

PHYSICIANS' HEALTH STUDY

The subjects and methods of the Physicians' Health Study, a 2 × 2 factorial trial of aspirin and β-carotene for the primary prevention of CVD or cancer, have been described previously. Briefly, 22071 US male physicians, aged 40 to 84 years at enrollment, were free from prior myocardial infarction (MI), stroke, and cancer (except nonmelanoma skin cancer).

STUDY POPULATION AND DATA COLLECTION

At baseline and the 7-year follow-up, subjects were asked to report current alcohol intake. Among the 22071 randomized men at baseline, 3616 were excluded because of missing data on alcohol consumption (n=1261) or the development of either CVD (n=1718) or cancer (n=637) between the baseline and 7-year follow-up questionnaires. Therefore, 18455 men free of CVD and cancer through the 7-year follow-up questionnaire and reporting alcohol consumption at baseline and at the 7-year follow-up formed the study population for this investigation.

On each questionnaire, physicians responded to a question on their usual consumption of alcoholic beverages. Self-reported alcohol intake has been shown to be reliable and valid in male health professionals and other populations. We converted 7 response categories (≥2 per day, daily, 5–6 per week, 2–4 per week, 1 per week, 1–3 per month, and rarely or never) into the number of alcoholic drinks consumed weekly (18, 7, 5.5, 3, 1, 0.5, and 0 drinks per week, respectively). We calculated the 7-year change in alcohol consumption as the difference between the number of drinks consumed weekly on the 7-year questionnaire minus that reported at baseline. We then constructed 5 categories of 7-year alcohol change: a large decrease of 6 drinks per week or more, a modest decrease of greater than 1 to less than 6 drinks per week, no change of −1 to 1 drink per week, a modest increase of greater than 1 to less than 6 drinks per week, and a large increase of 6 drinks per week or more.

Follow-up of the 18455 participants comprising the study population began after completion of the 7-year questionnaire. On subsequent annual follow-up questionnaires, participants were asked whether they had experienced any CVD event since the return of the last questionnaire. Cardiovascular disease events included CHD (MI, angina pectoris, coronary artery bypass graft surgery, or percutaneous transluminal coronary angioplasty) and stroke. For men reporting MI or stroke, relevant medical records were obtained from more than 95% of the participants on receipt of their consent or, in the case of death, consent from next of kin. Confirmation of reported diagnosis was made after examination of medical records and other available information by an end points committee that included 2 internists, a cardiologist, and a neurologist. The diagnosis of nonfatal MI was confirmed by use of World Health Organization criteria. Nonfatal stroke was defined as a typical neurologic deficit, sudden or rapid in onset, lasting longer than 24 hours and attributable to a cerebrovascular event. Cardiovascular disease–related death was documented by convincing evidence of a cardiovascular mechanism from all available sources, including death certificates and medical records. All analyses are based on the first confirmed report of a CVD event, at which time intake of less than 1 drink per week, the multivariate RRs (95% CIs) of CVD were 0.99 (0.80-1.24) for 1 to less than 2 drinks per week, 0.79 (0.66-0.95) for 2 to less than 4 drinks per week, 0.78 (0.66-0.93) for 4 to less than 7 drinks per week, 0.77 (0.64-0.94) for 1 drink per day, and 0.95 (0.57-1.57) for 2 drinks per day or more.

Table 1 compares subjects according to categories of 7-year changes in alcohol consumption, stratified by their baseline alcohol consumption. In general, we found few differences in coronary risk factors. Among 7360 men consuming 1 drink per week or less at baseline, those with larger increases in alcohol consumption were less likely to be never smokers, and more likely to be past or current smokers. Among 6612 men consuming greater than 1 to 6 drinks per week at baseline, only 1.1% reported a large increase (≥6 drinks per week) in alcohol consumption. In this subgroup, men with any increase (>1 drink per week) in alcohol consumption were slightly more likely to be current smokers and to have a history of treatment for hypertension. Finally, among 4483 men consuming 1 drink per day or more at baseline, 6.9% increased from 1 drink per day at baseline to 2 drinks per day or more at 7 years. These men were older, were current smokers, had higher blood pressure, and had a greater prevalence of treatment for hypertension. Men with the largest decreases in alcohol consumption (≥6 drinks per week) had a slightly greater body mass index.
During 101,310 person-years of follow-up (median follow-up, 5.8 years), there were 1091 cases of incident CVD. There was a significant interaction between categories of baseline alcohol consumption and future 7-year changes in alcohol consumption in relation to CVD risk (interaction \( P=0.02 \)). Therefore, the association between 7-year changes in alcohol consumption and the risk of CVD differed according to the baseline alcohol level, justifying our use of stratified analyses (Table 2). Compared with age-adjusted models, there was no apparent net effect of confounding with additional adjustment for coronary risk factors. The exclusion of men who increased their alcohol consumption to 2 drinks per day or more did not materially alter the RR among men consuming either 1 drink per week or less or greater than 1 to 6 drinks per week at baseline. Treatment for high cholesterol level was not included in models since 15.3% of men had missing data, plus only 1.2% of men reported any history of treatment for high cholesterol level among those providing data. Its inclusion in multivariate models only slightly altered the RRs.

Among men consuming 1 drink per week or less at baseline, men with a moderate increase (>1 to <6 drinks per week) in alcohol consumption had a borderline significant 29% reduced risk of CVD compared with men with no change (−1 to 1 drink per week) in alcohol consumption. Larger increases (≥6 drinks per week) in alcohol consumption were not associated with the risk of CVD. However, because only 10 men developed CVD in this category, the 95% CI was wide. The addition of a squared ordinal trend variable was borderline significant (\( P=0.06 \)), suggesting the possible presence of a nonlinear trend. Among men consuming greater than 1 to 6 drinks per week at baseline, there was no association between changes in alcohol consumption and the risk of CVD. First, we excluded men with CVD during the first 3 years of follow-up to remove any biases due to underlying illnesses that may have affected their 7-year alcohol consumption. Next, since the relation may differ between alcohol and either CHD or stroke, we computed separate RRs for each end point.

In Table 3, we present RRs of CVD when considering the joint effect of alcohol consumption on the base-
line and 7-year questionnaires, using men consuming 1 drink per week or less on both questionnaires as the reference group. Men who increased from 1 drink per week or less at baseline to greater than 1 to 6 drinks per week at 7 years had a borderline significant (P=.04) 30% reduction in CVD risk. Insufficient power limited our ability to examine the risk of CVD in men who increased from 1 drink per week or less at baseline to 1 drink per day or more at 7 years. Conversely, men who decreased from 1 drink per day or more at baseline to 1 drink per week or less at 7 years still had a nonsignificant (P=.18) 24% reduced risk of CVD. Otherwise, any alcohol consumption greater than 1 drink per week—whether at baseline or 7 years—was associated with reductions in CVD risk ranging from 18% to 34%.

Next, we considered models for the association between 7-year changes in alcohol consumption and the risk of CVD stratified by corresponding changes in selected coronary risk factors. Most subjects had no corresponding changes in hypertension status (89.1% of men), diabetes status (96.7% of men), smoking status (94.8% of men), physical activity (92.6% of men), and body weight (75.4% of men). As a result, limiting analyses to men with no corresponding changes in coronary risk factors yielded virtually identical results to the overall study population. Similarly, the exclusion of men with baseline hypertension or diabetes had little effect on the RRs. The exclusion of men with CVD during the first 3 years of follow-up had modest effects on the risk estimates. The potential inverse association for moderate increases (>1 to <6 drinks per week) in alcohol intake among men consuming 1 drink per week or less at baseline strengthened, with an RR of 0.49 (95% CI, 0.27-0.91) compared with men with no change in alcohol consumption.

The association between changes in alcohol consumption and either CHD (840 cases) or stroke (251 cases) yielded RRs that were similar in magnitude to those for CVD, although the low case counts for stroke resulted in considerably wider 95% CIs. Compared with men consuming 1 drink per week or less at baseline and 7 years, men who increased their alcohol consumption from 1 drink per week or less to greater than 1 to 6 drinks per week had RRs (95% CIs) of CHD and stroke of 0.72 (0.49-1.05) and 0.61 (0.27-1.40), respectively. Among men initially consuming 1 drink per day or more, increases in alcohol consumption were still associated with higher risks of CHD and stroke. Because most stroke cases were of ischemic origin (n=204), we were unable to examine the association between changes in alcohol consumption and risk of hemorrhagic stroke.

We found that among men initially consuming 1 drink per week or less, a small increase in alcohol consumption over 7 years of greater than 1 to less than 6 drinks per week may be associated with a 29% reduced risk of CVD. On the other hand, we found that men consuming greater than 1 to 6 drinks per week at baseline had no further reduction in CVD risk as a result of any 7-year increase in alcohol consumption, and had no increase in CVD risk from 7-year reductions in alcohol consumption. Men who increased their alcohol consumption from 1 drink per day at baseline to 2 drinks per day or more at 7 years had a 63% increased risk of CVD compared with men who did not change their consumption of 1 drink per day or more. We found no evidence for effect modification by corresponding changes in coronary risk factors, including hypertension, diabetes, smoking status, physical activity, and body weight, and found similar magnitudes of risk for CHD and stroke.

A wealth of epidemiological studies have consistently reported a J-shaped inverse association between alcohol consumption and the risk of CVD, with the lowest risk of CVD among those drinking approximately 1 drink per day. The increased risk of CVD at higher levels of alcohol consumption (>2 drinks per day) generally reflects an increased risk of stroke, beyond the range of alcohol consumption in our study. The reduction in CVD at moderate alcohol levels is primarily driven by a reduction in CHD. A meta-analysis by Maclure suggested an L-shaped inverse association for nonfatal CHD with reduced risks beginning at 3 drinks per week, and with no additional risk reduction at more than 1 drink per day. This finding is supported by the observation in our study that men who initially drank 1 drink per week or less had a lower CVD risk after increasing their alcohol consumption by only a few drinks per week. Several recent prospective studies reinforce the belief that these low levels of alcohol consumption still decrease CVD risk, including some from the present cohort of moderate-drinking physicians. Thun et al also recently reported a significant reduction in CVD-related death among those consuming less than 1 drink per day in a prospective study of 490000 men and women. Our findings are consistent with these effects.

Only 2 studies have investigated the association between changes in alcohol consumption and risk of CVD. Lazarus et al examined the relation between 9-year changes in alcohol consumption and the 11-year risk of CVD-related death in 1845 men and 2225 women. Compared with those always drinking moderately, men and women changing from no to any alcohol consumption had corresponding RRs (95% CIs) of CVD-related death
of 1.25 (0.53-2.97) and 1.33 (0.59-3.01). However, because overall only 95 and 92 CVD-related deaths occurred in men and women, respectively, they had limited power to detect modest RRs and collapsed several change categories. Goldberg et al13 observed 6069 Japanese-American men for the association between 6-year changes in alcohol consumption and the 15-year risk of CHD, and found a possible reduction in risk from initiating moderate alcohol consumption. Compared with men always abstaining from alcohol, men aged 51 to 64 years who changed from no alcohol consumption to 1 to 39 mL/d (approximately <3 drinks per day) had a significant 66% reduction in the risk of CHD. However, narrower categories of alcohol change (eg, abstainer to 1 drink per day) were not provided.

We found an unexpected and significant 63% increased risk of CVD among men who initially consumed 1 drink per day or more and increased their alcohol consumption compared with men with no change in alcohol consumption. This extended to CHD (71% increased risk) and stroke (42% increased risk). Because 2 drinks per day or more was the highest recorded cat-

<table>
<thead>
<tr>
<th>7-Year Change in Alcohol Consumption</th>
<th>Baseline Alcohol Consumption</th>
<th>Large Decrease (&lt;6 Drinks/wk) (n = 724)</th>
<th>Moderate Decrease (&gt;1 to &lt;6 Drinks/wk) (n = 4110)</th>
<th>No Change (&gt;1 to 1 Drink/wk) (n = 11 208)</th>
<th>Moderate Increase (&gt;1 to &lt;6 Drinks/wk) (n = 1919)</th>
<th>Large Increase (&gt;6 Drinks/wk) (n = 494)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>53.9 ± 9.9</td>
<td>52.3 ± 8.8</td>
<td>55.0 ± 9.1</td>
<td>...</td>
<td>56.7 ± 9.3</td>
<td>...</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>25.0 ± 2.9</td>
<td>24.7 ± 2.8</td>
<td>24.5 ± 2.8</td>
<td>...</td>
<td>24.7 ± 2.8</td>
<td>...</td>
</tr>
<tr>
<td>Blood pressure, mm Hg†</td>
<td>Systolic</td>
<td>128.3 ± 13.0</td>
<td>126.1 ± 11.3</td>
<td>126.9 ± 11.6</td>
<td>...</td>
<td>129.3 ± 11.4</td>
</tr>
<tr>
<td>Diastolic</td>
<td>80.0 ± 7.8</td>
<td>78.9 ± 7.3</td>
<td>79.2 ± 7.3</td>
<td>...</td>
<td>80.9 ± 7.3</td>
<td>...</td>
</tr>
<tr>
<td>Ever treated for hypertension, %</td>
<td>15.1</td>
<td>11.7</td>
<td>14.0</td>
<td>...</td>
<td>19.5</td>
<td>...</td>
</tr>
<tr>
<td>Diabetes, %</td>
<td>2.2</td>
<td>1.4</td>
<td>2.1</td>
<td>...</td>
<td>1.6</td>
<td>...</td>
</tr>
<tr>
<td>Smoking status, %</td>
<td>Never</td>
<td>34.6</td>
<td>41.5</td>
<td>34.6</td>
<td>...</td>
<td>31.8</td>
</tr>
<tr>
<td></td>
<td>Former</td>
<td>48.5</td>
<td>47.4</td>
<td>49.2</td>
<td>...</td>
<td>49.7</td>
</tr>
<tr>
<td></td>
<td>Current</td>
<td>16.8</td>
<td>11.1</td>
<td>16.2</td>
<td>...</td>
<td>18.5</td>
</tr>
<tr>
<td></td>
<td>Vigorous exercise ≥1/wk, %</td>
<td>71.0</td>
<td>77.6</td>
<td>73.5</td>
<td>...</td>
<td>72.1</td>
</tr>
<tr>
<td></td>
<td>Parental history of MI before</td>
<td>11.1</td>
<td>14.1</td>
<td>11.7</td>
<td>...</td>
<td>11.0</td>
</tr>
<tr>
<td>age 60 y, %</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
</tbody>
</table>

*Data are given as mean ± SD unless otherwise indicated. MI indicates myocardial infarction; ellipses, data not applicable.
†Self-reported.
category of alcohol consumption, we may have been unable to identify the heaviest drinkers in the distribution of alcohol intake for our study population. However, only 2.9% of the men in our study consumed 2 drinks per day or more at baseline and 1.7% of the men increased their alcohol consumption from 1 to 2 drinks per day or more. These men were not more likely to start or restart smoking. The possible excess risk in men increasing their alcohol consumption from 1 to 2 drinks per day or more may be confined to the heaviest drinkers of the J-shaped association who have an increased risk of CVD relative to nondrinkers.30,37 Our highest category of 2 drinks per day or more may not identify the threshold for an increased risk of CVD, which may occur at 2, 3, or greater numbers of drinks per day. In any case, our study suggests that men consuming 1 drink per day will not achieve increased reductions in CVD or CHD with additional increases in alcohol consumption.

Our finding that a modest increase in alcohol consumption among men with low intake may lower the risk of CVD is biologically plausible. Recent studies9,10,38 confirm that the alcohol, rather than the specific type of alcoholic beverage, is responsible for the apparent reduction in CVD risk. The inverse associations for each beverage type appear to be mediated by increases in HDL-C levels.3 Alcohol increases HDL-C levels, which in turn promotes reverse cholesterol transport along the arterial wall and reverses the atherosclerotic process. Clinical trials7-10 have demonstrated these biological associations for HDL-C level and other lipid variables. Evidence from experimental and observational studies22 suggests that approximately 50% of the risk reduction attributable to alcohol consumption may be explained by changes in total HDL-C level. Alcohol may also increase the level of tissue plasminogen activator, a clot-dissolving enzyme,39 thereby prolonging bleeding time and reducing platelet aggregation.11 Small 7-year increases in alcohol consumption among drinkers of low levels of alcohol in our study may, therefore, produce these short-term cardiovascular benefits and potentially reduce subsequent CVD risk. Furthermore, men with 7-year decreases in alcohol consumption had no increased risk of CVD, suggesting that the risk reduction for CVD continues for a period after a reduction in alcohol consumption.

Some limitations in our study design should also be considered. First, we do not know why men changed their

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**Table 2. Relative Risks (RRs) of Cardiovascular Disease (CVD) According to Categories of Baseline Alcohol Consumption and Future 7-Year Changes in Alcohol Consumption**

<table>
<thead>
<tr>
<th>Baseline Alcohol Consumption</th>
<th>Large Decrease (≥ 6 Drinks/wk)</th>
<th>Moderate Decrease (1 to &lt; 6 Drinks/wk)</th>
<th>No Change (1 to 1 Drink/wk)</th>
<th>Moderate Increase (&lt; 1 to ≥ 6 Drinks/wk)</th>
<th>Large Increase (≥ 6 Drinks/wk)</th>
<th>Trend P†</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤ 1 Drink/wk</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CVD cases</td>
<td>...</td>
<td>424</td>
<td>37</td>
<td>10</td>
<td>...</td>
<td></td>
</tr>
<tr>
<td>Age-adjusted RR</td>
<td>...</td>
<td>1.00 (ref)</td>
<td>0.69 (0.50-0.97)</td>
<td>1.21 (0.65-2.28)</td>
<td>0.65</td>
<td></td>
</tr>
<tr>
<td>Multivariate-adjusted RR‡</td>
<td>...</td>
<td>1.00 (ref)</td>
<td>0.71 (0.50-1.00)</td>
<td>1.04 (0.51-2.10)</td>
<td>0.42</td>
<td></td>
</tr>
<tr>
<td>&gt; 1-6 Drinks/wk</td>
<td></td>
<td>141</td>
<td>129</td>
<td>61</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>CVD cases</td>
<td></td>
<td>1.07 (0.84-1.35)</td>
<td>1.00 (ref)</td>
<td>0.98 (0.72-1.33)</td>
<td>1.05 (0.39-2.84)</td>
<td>0.63</td>
</tr>
<tr>
<td>Age-adjusted RR</td>
<td></td>
<td>1.07 (0.84-1.36)</td>
<td>1.00 (ref)</td>
<td>0.85 (0.62-1.18)</td>
<td>0.97 (0.36-2.64)</td>
<td>0.26</td>
</tr>
<tr>
<td>Multivariate-adjusted RR‡</td>
<td></td>
<td>1.02 (0.64-1.34)</td>
<td>1.00 (ref)</td>
<td>...</td>
<td>1.61 (1.09-2.37)</td>
<td>0.04</td>
</tr>
<tr>
<td>≥ 1 Drink/d</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CVD cases</td>
<td>39</td>
<td>80</td>
<td>124</td>
<td>32</td>
<td>...</td>
<td></td>
</tr>
<tr>
<td>Age-adjusted RR</td>
<td>0.96 (0.67-1.38)</td>
<td>1.04 (0.79-1.39)</td>
<td>1.00 (ref)</td>
<td>...</td>
<td>1.61 (1.09-2.37)</td>
<td>0.04</td>
</tr>
<tr>
<td>Multivariate-adjusted RR‡</td>
<td>0.92 (0.64-1.34)</td>
<td>1.02 (0.76-1.36)</td>
<td>1.00 (ref)</td>
<td>...</td>
<td>1.63 (1.10-2.40)</td>
<td>0.02</td>
</tr>
</tbody>
</table>

*Data in parentheses are 95% confidence intervals. Ref indicates reference; ellipses, data not applicable.
†Test for linear trend. The test for nonlinear trend was borderline significant (P = .07) for men with a baseline alcohol consumption of 1 drink per week or less in the multivariate model.
‡Adjusted for baseline age (40-49, 50-59, 60-69, and ≥ 70 years), randomized aspirin treatment (yes or no), randomized β-carotene assignment (yes or no), smoking status (never, past, currently < 1 pack per day, or currently ≥ 1 pack per day), parental history of myocardial infarction before age 60 years (yes or no), vigorous exercise once a week or more (yes or no), body mass index (< 23, 23-<24.5, 24.5-<26, and ≥26), any history of treatment for hypertension (yes or no), and diabetes (yes or no).

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**Table 3. Multivariate Relative Risks of Cardiovascular Disease (CVD) According to Alcohol Consumption at Baseline and at 7 Years**

<table>
<thead>
<tr>
<th>Baseline Alcohol Consumption</th>
<th>≤ 1 Drink/wk</th>
<th>&gt; 1-6 Drinks/wk</th>
<th>≥ 1 Drink/d</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤ 1 Drink/wk</td>
<td>1.00 (ref) [424]</td>
<td>0.70 (0.49-0.99) [37]</td>
<td>1.00 (0.50-2.02) [10]</td>
</tr>
<tr>
<td>&gt; 1-6 Drinks/wk</td>
<td>0.82 (0.66-1.03) [98]</td>
<td>0.79 (0.67-0.94) [206]</td>
<td>0.66 (0.46-0.94) [41]</td>
</tr>
<tr>
<td>≥ 1 Drink/d</td>
<td>0.76 (0.51-1.13) [29]</td>
<td>0.73 (0.57-0.93) [83]</td>
<td>0.77 (0.64-0.93) [163]</td>
</tr>
</tbody>
</table>

*Adjusted for baseline age (40-49, 50-59, 60-69, and ≥ 70 years), randomized aspirin treatment (yes or no), randomized β-carotene assignment (yes or no), smoking status (never, past, currently < 1 pack per day, or currently ≥ 1 pack per day), parental history of myocardial infarction before age 60 years (yes or no), vigorous exercise once a week or more (yes or no), body mass index (< 23, 23-<24.5, 24.5-<26, and ≥26), any history of treatment for hypertension (yes or no), and diabetes (yes or no). Data in parentheses are 95% confidence intervals; brackets, number of CVD cases.
alcohol consumption, since drinking patterns among middle-aged and older men tend to be stable over time. When we restricted our analysis to men with no corresponding changes in hypertension status, diabetes status, smoking status, physical activity level, or body weight, the overall results remained the same. However, we were unable to consider corresponding changes in self-reported cholesterol level since only 28.7% of the men reported such data. Second, we do not know when, during the 7-year interval, the change in alcohol consumption occurred. Therefore, we cannot definitively conclude whether the association of changes in alcohol consumption on CVD risk is immediate or delayed. Third, our study relied on self-reported alcohol consumption. However, studies have found self-reports to be reliable for the general classification of drinking habits, including those of health professionals. Fourth, our findings may not apply to women, populations with a lower socioeconomic status, and minority groups, who may have different drinking patterns and physiological responses to alcohol. Finally, unaccounted personality characteristics or other lifestyle factors associated with changes in alcohol consumption and risk of CVD may introduce a modest degree of residual confounding, thereby affecting our risk estimates.

Although we found a possible inverse association for a small increase in alcohol consumption on the risk of CVD among men initially consuming 1 drink per week or less, larger increases in alcohol consumption may have adverse health effects. Alcohol consumption is positively correlated with blood pressure, body weight, and triglycerides, and a greater prevalence of hypertension and diabetes. Other potential medical problems (liver disease and alcoholism) should also be considered. Any individual recommendation must consider the complexity of alcohol’s metabolic, physiological, and psychological effects. Men consuming greater than 1 drink per week of alcohol have no apparent reductions by increasing their alcohol consumption. A public health recommendation for widespread alcohol consumption is viewed as neither a primary prevention strategy nor an unhealthy behavior. A study for moderate alcohol intake in fixed or variable amounts on concentration of serum lipids and liver enzymes in healthy young men. Am J Clin Nutr. 1989;50:987-991.

References


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