Alcohol consumption, 29-y total mortality, and quality of life in men in old age

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ABSTRACT

Background: The negative effects of excessive alcohol consumption are well known, but moderate alcohol consumption is advocated for health reasons.

Objective: We compared 29-y total mortality and quality of life in old age by alcohol consumption in midlife.

Design: Cardiovascular disease risk factors and alcohol consumption were assessed in 1974 in 1808 men (aged 40–55 y) of high socioeconomic status. At baseline, the men were without signs of chronic diseases. Baseline alcohol consumption was divided as zero (n = 116), moderate (1–349 g/wk; n = 1519), and high (>349 g/wk; n = 173). Quality of life was surveyed in 2000 with the RAND-36 (SF-36) health survey (n = 1216). Mortality was retrieved from registers during the 29-y follow-up.

Results: Median alcohol consumption in 1974 and in 2000 was 123 (interquartile range: 56–238) and 84 (28–168) g/wk, respectively, and was significantly correlated. Values of cardiovascular disease risk factors measured in 1974 increased with increasing alcohol consumption. During the 29-y follow-up, 499 men (27.6%) died; mortality was significantly higher among men with the highest alcohol consumption (37.6%) than in abstainers (25.0%) or in men with moderate (26.7%) consumption. Quality of life was not significantly associated with baseline alcohol consumption in responding survivors but was worst in men with high consumption when deaths during follow-up were accounted for.

Conclusions: In this male cohort of high socioeconomic status, only the highest alcohol consumption (>3 drinks/d) affected mortality, and it was associated with worse quality of life in old age. Moderate alcohol consumption in middle age offered no special benefits compared with abstinence over the long term. Am J Clin Nutr 2004; 80:1366–71.

KEY WORDS Alcohol, aging, mortality, quality of life, RAND-36 (SF-36)

INTRODUCTION

Excessive alcohol consumption causes well-known health hazards and societal ills (1). On the other hand, the apparent benefit of moderate alcohol consumption on coronary heart disease has been documented in numerous studies (2–6), although not in all (7). Moderate consumption has also been associated with better cognitive function (8) and less dementia (9). Consequently, moderate alcohol consumption could even be advocated for preventive purposes in middle-aged and elderly persons. However, views clearly differ on this (10), and it has been suggested that moderate users may simply be protected by other beneficial effects of their lifestyle (ie, the healthy user bias; 11). Although a randomized controlled trial in which confounding effects are eliminated would give valuable information (11), it is ethically difficult to conduct such a trial with alcohol. Consequently, controlling for confounders is important in observational studies. Moreover, alcohol consumption is usually a lifelong habit, and thus the health effects should also be considered in the long-term, not for only 5- to 10-y follow-up times.

We considered that total mortality and quality of life are relevant endpoints for a follow-up study of total alcohol effects. To our knowledge, the effect of long-term alcohol consumption on the quality of life in old age has not been studied. Our cohort was socioeconomically homogeneous, and all men were middle-aged, working, and clinically healthy at baseline. This setting offered a clearer test of the effects of alcohol per se; on the other hand, those men with substantial problems related to alcohol before middle age were not included.

SUBJECTS AND METHODS

Baseline examinations in 1974 and follow-up examination in 1986

The cohort and examinations have been described before (12–14). Initially healthy men, mostly business executives born in 1919–1934, had participated in structured health check-ups during the 1960s and early 1970s at the Institute of Occupational Health in Helsinki. They were evaluated with questionnaires and clinical and laboratory examinations in 1974 (Figure 1). In the questionnaires, they were asked to report their average weekly alcohol consumption during the past year (available for 1808 men). Exdrinkers were not identified. In this study, one unit of alcohol (a “restaurant unit” equal to one bottle of beer, one glass of wine, or one drink) was calculated to contain 14 g alcohol. Consumption at baseline was initially categorized in 7 groups in g/wk (no alcohol, n = 116; 1–34, n = 199; 35–69, n = 203; 70–99, n = 200; 100–199, n = 203; 200–299, n = 203; and 300–699, n = 199). Mortality was retrieved from registers during the 29-y follow-up.

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In addition, the Finnish version of the RAND-36 Health Survey 1.0 (practically identical to the SF-36 health survey; 15, 16) was embedded in the questionnaire. The 8 RAND-36 scales (physical function, role physical, bodily pain, general health, vitality, social function, role emotional, and mental health) measure health-related quality of life, and the RAND-36 as a mailed questionnaire has been validated in the general Finnish population (15).

Mortality follow-up

The total mortality of the study population up to 31 December 2002 was retrieved from the Central Population Register, which keeps a register of all Finnish citizens. According to the register, the assessment of vital status is very reliable for persons having their permanent place of residence in Finland (>95% of the present cohort in 2000), irrespective of whether they die in Finland or abroad. Moreover, the assessment of vital status is reliable for Finnish citizens living permanently abroad. Causes of death up to 31 December 1999 were determined from the countrywide computerized Cause-of-Death Register of Statistics Finland, in which trained nosologists code the causes of death. During the follow-up, causes of death were assigned by one of the investigators (TES) to the following categories: coronary, stroke, other cardiovascular, cancer, violent (accidents or suicide), and other, which consisted of deaths outside the previous categories.

Statistical methods

NCSS statistical software (2000 version; NCSS Statistical Software, Kaysville, UT; Internet: www.ncss.com) was used for the analyses. Alcohol consumption was categorized as described above. * t-Tests, nonparametric tests, and analyses of covariance were used where appropriate to compare continuous variables, chi-square tests were used to compare proportions, and Spearman’s test was used to assess correlations. Logarithmic transformations were used where appropriate. Differences in survival curves were analyzed with log-rank tests. Relative risks (RRs) and their 95% CIs for mortality associated with baseline serum cholesterol concentrations were calculated by using Cox’s proportional hazards regression. Other risk factors were adjusted for in respective models. The 8 RAND-36 scales were calculated from questionnaires (15, 16). From these scales, the physical component summary (PCS) and the mental component summary (MCS) scores were calculated (17). Because a substantial proportion of the men had died by the time of the RAND-36 evaluation in 2000, we also used the technique described by Diehr et al (18) to account for deaths when assessing health-related quality of life. These analyses produced new values for the PCS and MCS scores in which persons who had died were coded as zero (18). Both original and death-accounted values are presented. Two-tailed tests were used, and P values < 0.05 were taken as significant.

RESULTS

Baseline

The baseline characteristics of the cohort according to weekly alcohol consumption (0, 1–349, and >349 g/wk) are shown in Table 1. Reported mean alcohol consumption in 1974 was 159 g/wk (n = 1808; SD: 153; median: 123, interquartile range: 56–238 g/wk). The values of several cardiovascular disease risk factors were normally distributed. In 1974 men reported a mean alcohol consumption of 159 g/wk, which was significantly higher than the consumption in 1986 (132 g/wk). Consumption was not significantly different between respondents and nonrespondents. Weight and height were measured, and body mass index (BMI) was calculated as weight (in kg) divided by height (in m) squared. A total of 1654 men were reexamined in 1979–1980; 1216 (79%) of the survivors responded (Figure 1). Baseline alcohol consumption was similar to the earlier surveys of 1974 and 1986.
factors measured in 1974 rose progressively with increasing alcohol consumption. Also, reported weight gain from the age of 25 y was positively associated with alcohol consumption in 1974. Self-report of subjective health and physical fitness in the 3 groups in 1974 showed that higher alcohol consumption was associated with worse profiles (Figure 2).

**Alcohol consumption during follow-up**

Age-adjusted alcohol consumption during the 29-y follow-up in those survivors who reported their current consumption in 2000 (n = 960) was relatively stable in lower categories but decreased in the highest category. Differences between the original groups prevailed, however. Mean consumption in 2000 was 117 g/wk (SD: 137, median: 84, interquartile range: 28–168 g/wk). Reported alcohol consumptions in 1974 and 2000 were significantly correlated (r = 0.53, P < 0.0001).

In 1986 serum γ-glutamyl transferase activity, measured in 235 men, was significantly correlated with reported alcohol consumption (r = 0.41, P < 0.0001). Serum HDL-cholesterol and triacylglycerol concentrations (measured in 1211 men) were weakly positively correlated with reported alcohol consumption (r = 0.14, P < 0.0001, and r = 0.13, P < 0.0001, respectively), whereas there was no significant correlation between LDL-cholesterol concentrations and alcohol use (r = −0.05, P = 0.86).

**Mortality**

During the 29-y follow-up, 499 men (27.6% of the initial 1974 cohort) died. Mortality was significantly higher among men in the highest baseline category of reported alcohol consumption (37.6%) but was nonsignificantly different between the other 2 groups (25.0% and 26.7% in abstainers and moderate drinkers, respectively). Crude mortalities in the 3 groups (Figure 3, panel A) were significantly different (log-rank test, P = 0.01). For comparison, mortality in the 7 categories of alcohol consumption is shown in Figure 3, panel B. The log-rank test also showed a significant difference (P = 0.01) between the 7 groups, but this was due to the consistently higher mortality in the highest alcohol consumption group; differences between the 6 lower categories were not significant (log-rank test, P = 0.17). Causes of death (retrieved up to 1999) are shown in Table 2. There were no clear differences between the zero and moderate consumption groups, whereas there were more coronary, cancer, and other deaths in the highest consumption group. However, only the difference in other deaths was statistically significant (log-rank test, P = 0.004). It is of note that deaths due to accidents and suicide were almost identical in the groups.

Because alcohol consumption of >349 g/wk was associated with higher values of risk factors at baseline, we also performed multivariate analyses using proportional hazards regression to further compare total and other mortality. In these analyses, zero
and high alcohol consumption were compared with moderate intake (Table 3). When important risk factors were added to the model, alcohol consumption was no longer a significant predictor of total mortality. In contrast, other deaths (due to noncardiovascular, noncancer, and nonaccidental causes) were increased almost 3-fold in the highest alcohol consumption group, and this increase was insensitive to the added covariates. We also tested coronary and cancer mortality differences with covariates, but they remained statistically insignificant (data not shown).

Table 3

<table>
<thead>
<tr>
<th>Cause of death</th>
<th>None (n = 116)</th>
<th>1–349 g/wk (n = 1519)</th>
<th>&gt;349 g/wk (n = 173)</th>
<th>P²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary</td>
<td>60.3 (7)</td>
<td>60.6 (92)</td>
<td>75.1 (13)</td>
<td>NS</td>
</tr>
<tr>
<td>Stroke</td>
<td>0 (0)</td>
<td>11.8 (18)</td>
<td>5.8 (1)</td>
<td>NS</td>
</tr>
<tr>
<td>Other cardiovascular</td>
<td>8.6 (1)</td>
<td>16.5 (25)</td>
<td>17.3 (3)</td>
<td>NS</td>
</tr>
<tr>
<td>Cancer</td>
<td>77.6 (9)</td>
<td>69.1 (105)</td>
<td>98.3 (17)</td>
<td>NS</td>
</tr>
<tr>
<td>Accident or suicide</td>
<td>25.9 (3)</td>
<td>22.4 (34)</td>
<td>23.1 (4)</td>
<td>NS</td>
</tr>
<tr>
<td>Other</td>
<td>25.9 (3)</td>
<td>23.7 (36)</td>
<td>63.6 (11)</td>
<td>0.004</td>
</tr>
</tbody>
</table>

¹ Deaths per 1000 men; number of absolute events in parentheses.
² Log-rank test was used for comparisons between the groups.

The 2000 survey and quality of life

In 2000, 86% (n = 1216) of the survivors responded to the questionnaire. Nonrespondents (n = 200) had higher mean baseline alcohol consumption than did respondents (181 compared with 148 g/wk; P = 0.01). This was due to a greater proportion of high alcohol consumption among nonrespondents (12.7% compared with 8.1%), whereas the proportions of abstainers were the same (6.6%). In 2000, >90% of the respondents were retired and 97–99% were home dwelling, with no significant differences between the 3 groups. Risk factor differences between the groups had diminished among respondents, but there were still significantly more smokers (P = 0.001) and significantly fewer regular exercisers (P = 0.03) in the high alcohol consumption group.

As to health-related quality of life assessed with the RAND-36 questionnaire, there were no significant differences in PCS or MCS scores between the alcohol consumption groups (Figure 4, adjusted for age). The results were essentially similar when the data were further adjusted for smoking, systolic blood pressure, serum cholesterol, and body mass index (data not shown) but were different when quality of life was further adjusted for death during follow-up. When adjusted for age only, both physical and mental health were clearly poorer in men with the highest alcohol consumption but were fairly similar between the men with zero consumption and those with moderate consumption (Figure 4). In the risk factor–adjusted model, the differences were marginally significant, and men with moderate alcohol consumption tended to have the best quality of life (data not shown).

Discussion

In our male cohort, alcohol use in midlife was associated with several cardiovascular disease risk factors in a significant and graded fashion. Despite this, only the highest category of consumption was associated with higher total mortality during the almost 30 y of follow-up. Even this association was not statistically significant, however, when baseline risk factors were adjusted for. However, deaths due to causes where alcohol could be directly involved were clearly and significantly higher in the highest alcohol consumption group. Among survivors in 2000, alcohol consumption was not associated with physical or mental quality of life. However, high consumption was harmful to quality of life if deaths during follow-up were accounted for. An important finding was that being an abstainer (at least from midlife) showed no special health disadvantages in this homogeneous cohort.

Although many previous studies of alcohol and mortality have been done, our study has special strengths for studying the effects of alcohol itself. The important confounders of socioeconomic class and comorbidity at baseline were eliminated, cardiovascular disease risk factors were measured, the follow-up time was almost 30 y, and we had information on alcohol consumption at different time points during the follow-up. In addition, we related alcohol consumption to quality of life in old age, when the original differences in alcohol consumption still prevailed.

The results of the present study are based on a relatively large cohort of men with a substantial number of mortality endpoints. The verification of mortality by the national registers was reliable. Because social class influences alcohol consumption and alcohol-related mortality (19), our study with all men from the...
highest socioeconomic class represents a cleaner test of the hypothesis in an area where social class may prove to have an important confounding effect. We also believe that the reporting of alcohol consumption was reliable in this cohort. This was supported by the consistent pattern of consumption over the decades. The tracking of consumption from 1974 to 2000 was good, and reported alcohol intake was significantly correlated with the values for several risk factors known to be associated with alcohol consumption. In addition, reported alcohol consumption was associated with serum γ-glutamyl transferase, a biochemical indicator of alcohol consumption (20). Nevertheless, when assessing the present results, it must be borne in mind that the cohort at baseline represented individuals who in midlife were healthy and professionally active, irrespective of their alcohol consumption habits. Thus, the results cannot be extrapolated to younger men with similar consumptions. Many alcohol-related deaths occur in young age groups (21), and a 25-y follow-up study in young conscripts found a clear association between alcohol use (>14 g/d) at conscription and risk of subsequent alcohol-related hospitalizations and deaths up to the age of 45 y (22). It is of note that violent deaths were not associated with alcohol consumption in our cohort.

An important question concerning alcohol consumption is the long-term tradeoff between harmful and possibly beneficial effects. Our results unexpectedly suggest that the long-term effects of alcohol on mortality and health in midlife and later are neutral. Specifically, alcohol is not needed for successful aging; moderate consumption does not seem to be associated with the length or the quality of life. In most of our analyses, however, the cohort was divided into 3 categories: a large intermediate group and smaller groups of zero and high consumption. This division may have hidden the benefits of, for example, very moderate consumption. However, this is not supported by the mortality curves for the 7 categories (Figure 3, panel B), in which all of the lower categories were totally intermingled. Part of the harmful effects associated with greater alcohol consumption may in fact be due to factors other than alcohol (such as smoking and weight gain) in this highly selected, middle-aged male cohort.

The abstainer group was somewhat enigmatic because we do not have information on how many of them were actually lifelong abstainers and how many had quit earlier alcohol consumption. If the latter proportion were large, it could obviously dilute differences between groups. The proportion of abstainers in our total cohort (116/1808, or 6.4%), however, is similar to the proportion of reported never-users in a population survey conducted in Finland during the late 1960s (8%, or 23). The abstainer group is nevertheless a special group; for example, they had clearly lower risk factor status at baseline (Table 1), possibly reflecting their health-conscious nature. Therefore, they should also have a better prognosis, but this was not the case in our cohort.

Alcohol consumption as such seemed to be less harmful when adjusted for smoking and some other cardiovascular disease risk factors (and vice versa: zero consumption was less beneficial). We aimed not to adjust consumption for factors that may be in the pathogenetic pathway between alcohol and adverse outcomes. These factors are body weight (24), blood pressure, and serum triacylglycerols. Also, counting only survivors in old age may give a wrong impression, because those who have been able to cut their drinking may be better off. Therefore, we also accounted
for deaths when assessing the quality of life (25), and in these analyses greater alcohol consumption was clearly harmful, especially if cardiovascular disease risk factors were not adjusted for.

Although the effects of alcohol consumption on total mortality were smaller than expected, closer analysis of causes of death nevertheless showed that deaths due to noncardiovascular, non-cancer, and nonaccidental causes (other) were almost 3-fold higher in the highest alcohol consumption group. The other causes have not been described in more detail, but they include, for example, deaths due to cirrhosis of the liver. The insensitivity to covariates further supports the central role of alcohol in these deaths. Thus, not even this cohort was immune to the toxic effects of excess alcohol consumption.

As already implicated, our study has some important limitations. The cohort consisted mainly of low to moderate middle-aged drinkers and included only a limited number of heavy drinkers, probably because the men were professionally active and healthy at baseline in 1974. Because our population was selected, all participants being men from the highest social class, any extrapolation of the results to the general population (and especially to women) needs to be done cautiously. Population surveys conducted in Finland during the 1960s (23) and in 2000 (STAKES, the National Research and Development Centre for Welfare and Health, Helsinki; data on file) suggest that drinking patterns differ by socioeconomic classes. Although the annual amount may be higher and there are more drinking occasions, consumption is more even, probably connected to social occasions and meals; there is less binge drinking; and consumption at one occasion is less in higher than in lower socioeconomic classes. In our cohort, we have data only on the amount of alcohol, not the pattern of drinking, which may affect the health outcomes (6). Another limitation of our study is that quality of life could not be assessed in 1974 because the RAND-36 did not exist at that time. However, the data in Figure 2 indicate that high consumption of alcohol was associated with at least a slight impairment in health-related quality of life already in midlife. A question may arise as to whether these differences are a cause or a consequence of alcohol consumption. In 2000, nonrespondents included a slightly greater proportion of heavy drinkers at baseline, and this may have attenuated the differences in quality of life, risk factors, and reported diseases among respondents. Finally, an interesting issue is the type of alcoholic beverage consumed (beer, wine, or liquor), the significance and exact mechanisms of which are still controversial (6, 26, 27). We have data on the preferences for beverage type in our cohort, but these will be communicated in a separate report.

In conclusion, in this selected group of men from the highest social class in Finland, the long-term overall health effects of alcohol consumption were unexpectedly neutral. Specifically, moderate alcohol consumption as such did not offer special health benefits compared with zero consumption. Even our cohort, however, showed the harm of high alcohol consumption. Taking into account the clear and substantial problems of excessive use at the societal level, our study clearly challenges the need to propagate alcohol consumption for health reasons.

Author contributions were as follows: design of the experiment (AYS, TES, and TAM), collection of data (TES, VVS, and TAM), analysis of data (AYS, TES, and KP), and writing and revising the manuscript (AYS, TES, VVS, KP, and TAM). None of the authors had a conflict of interest.

REFERENCES