Meta-Analysis of Wine and Beer Consumption in Relation to Vascular Risk

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Background—Many epidemiological studies have evaluated whether different alcoholic beverages protect against cardiovascular disease. We performed a meta-analysis of 26 studies on the relationship between wine or beer consumption and vascular risk.

Methods and Results—General variance-based method and fitting models were applied to pooled data derived from 26 studies that gave a quantitative estimation of the vascular risk associated with either beverage consumption. From 13 studies involving 209,418 persons, the relative risk of vascular disease associated with wine intake was 0.68 (95% confidence interval, 0.59 to 0.77) relative to nondrinkers. There was strong evidence from 10 studies involving 176,042 persons to support a J-shaped relationship between different amounts of wine intake and vascular risk. A statistically significant inverse association was found up to a daily intake of 150 mL of wine. The overall relative risk of moderate beer consumption, which was measured in 15 studies involving 208,036 persons, was 0.78 (95% confidence interval, 0.70 to 0.86). However, no significant relationship between different amounts of beer intake and vascular risk was found after meta-analyzing 7 studies involving 136,382 persons.

Conclusions—These findings show evidence of a significant inverse association between light-to-moderate wine consumption and vascular risk. A similar, although smaller association was also apparent in beer consumption studies. The latter finding, however, is difficult to interpret because no meaningful relationship could be found between different amounts of beer intake and vascular risk. (Circulation. 2002;105:2836-2844.)

Key Words: cardiovascular diseases ■ wine ■ beer ■ meta-analysis

An inverse association between moderate alcohol consumption and vascular risk has been shown in many epidemiological studies.1–6 All-cause mortality as a function of alcohol use has been depicted as a J-shaped curve.5–8 Reflecting a lower risk of coronary heart disease (CHD) at moderate consumption and an increased risk of certain cancers and cirrhosis at higher amounts.1,6–9 After wine intake was suggested as a possible explanation for the lower than expected CHD mortality rates in France,10 many studies have dealt with the question of whether different alcoholic beverages are equivalent in their ability to protect against CHD or if a specific beverage might offer a greater protection,2–4 but data are inconclusive.1,3,11,12 A recent advisory from the American Heart Association13 concluded that the usual standards to recommend alcohol consumption as a CHD prevention approach are not met and that wine is indistinguishable from other types of alcoholic beverages. These conclusions have been challenged.14–16

We did a systematic review of the literature and a meta-analysis of selected studies to evaluate the relationship between wine and beer consumption and vascular risk. We also tried to give a quantitative estimate of this relationship.

Methods

Search Strategy
First, a PUBMED search (http://www4.ncbi.nlm.nih.gov/PubMed) together with an assessment of the references of published studies was conducted until September 2001 to identify studies evaluating the relationship between alcohol consumption and vascular risk. More than 80 publications were identified. Next, publications reporting risks specific for wine and/or beer intake were selected, and 30 studies were identified.17–46 Some were multiple reports from the same cohort.20,43,44 One study46 was excluded because neither confidence intervals (CIs) nor precise probability values were reported. A total of 26 reports were identified for the meta-analysis. Two separate meta-analyses were conducted: the first used the 23 studies reporting data for wine,17–31,34–41 and the second used the 22 studies reporting data for beer.17,27,30–39,42 Finally, the studies were
divided into 2 groups. The first group included studies that consid-
er only a category of risk (drinkers versus nondrinkers), and the
second was formed by studies that reported "trend"
alyses of risk,
 ie, that considered more than one category of wine (beer)
 intake ("dose-response"
meta-analysis).

**Data Extraction**

Some studies had not taken the intake of different
types of alcoholic beverages into account (type A), whereas in others
(type B) the bias of combined drinking of different alcoholic
beverages in the same population was either formally excluded using
drinkers of only a specific type of beverages or was taken
into account in multivariate analyses of risk. In studies reporting more than one clinical end point, results on
combined (fatal and nonfatal) events and on CHD with respect to
other vascular events were used. Relative risks were extracted as a
measure of the relation between vascular events and wine or beer
consumption (whatever the amount consumed in drinkers versus
nondrinkers meta-analysis and for each specific consumption cate-
gory in the dose-response meta-analysis). Whenever possible, the
amount of a "drink" (mL/d) was taken as quantified by each author
(as it occurred in all beer studies); otherwise, to allow meaningful
comparisons among different categorizations of wine intake, a
"drink" was considered equivalent to 130 mL of wine. If not
otherwise reported in the study, it was assumed that wine contains
12% and beer 6% ethanol. Other sources of heterogeneity in the
methodological quality of the studies were taken into account by
performing sensitivity analysis, and prespecified subgroups were
considered according to type of cohort or event in case group, sex,
adjustment for different types of alcoholic beverages or for indicators

**TABLE 1. Characteristics of the Studies Included in the Drinkers vs Nondrinkers Meta-Analyses**

<table>
<thead>
<tr>
<th>Reference (Year)</th>
<th>Intake</th>
<th>Wine</th>
<th>Beer</th>
<th>Type of Cohort</th>
<th>Number of Subjects</th>
<th>Outcome and Sex Distribution</th>
<th>Type of Adjustment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kozararevic et al (1980)</td>
<td>$&lt;1$ drink/day</td>
<td>1</td>
<td>1</td>
<td>Prospective</td>
<td>11 121</td>
<td>CHD fatal and nonfatal</td>
<td>A</td>
</tr>
<tr>
<td>Friedman et al (1986)</td>
<td>$&lt;1$ drink/day</td>
<td>0.68</td>
<td>0.42–1.11</td>
<td>0.58</td>
<td>0.26–1.27</td>
<td>Men</td>
<td></td>
</tr>
<tr>
<td>Rimm et al (1991)</td>
<td>2 drinks/day</td>
<td>0.25</td>
<td>0.6–1.12</td>
<td>0.49</td>
<td>0.19–1.29</td>
<td>Both sexes</td>
<td>B</td>
</tr>
<tr>
<td>Klatsky et al (1990)</td>
<td>$&lt;2$ drinks/week</td>
<td>0.5</td>
<td>0.4–0.7</td>
<td>0.7</td>
<td>0.5–0.9</td>
<td>Both sexes</td>
<td>B</td>
</tr>
<tr>
<td>Wannamethee et al (1999)</td>
<td>Occasional drinkers</td>
<td>1</td>
<td>1</td>
<td>Prospective</td>
<td>7735</td>
<td>CHD fatal and nonfatal</td>
<td>A</td>
</tr>
<tr>
<td>Rosenberg et al (1991)</td>
<td>Regular drinkers</td>
<td>0.92</td>
<td>0.51–1.67</td>
<td>0.78</td>
<td>0.63–0.97</td>
<td>Men</td>
<td>B</td>
</tr>
<tr>
<td>Kaufman et al (1985)</td>
<td>Never drank</td>
<td>0.5</td>
<td>0.3–1.0</td>
<td>0.8</td>
<td>0.5–1.3</td>
<td>Women</td>
<td>B</td>
</tr>
<tr>
<td>Sacco et al (1999)</td>
<td>Nondrinkers</td>
<td>1.1</td>
<td>0.6–1.9</td>
<td>1.0</td>
<td>0.6–1.6</td>
<td>Men</td>
<td>B</td>
</tr>
<tr>
<td>Gaziano et al (1999)</td>
<td>Nondrinkers</td>
<td>0.40</td>
<td>0.23–0.70</td>
<td>0.56</td>
<td>0.35–0.90</td>
<td>Both sexes</td>
<td>A</td>
</tr>
<tr>
<td>Kaufman et al (1985)</td>
<td>Never drank</td>
<td>0.58</td>
<td>0.31–1.09</td>
<td>0.75</td>
<td>0.40–1.40</td>
<td>Both sexes</td>
<td>B</td>
</tr>
<tr>
<td>Sacco et al (1999)</td>
<td>Nondrinkers</td>
<td>0.5</td>
<td>0.2–0.9</td>
<td>0.9</td>
<td>0.5–1.7</td>
<td>Both sexes</td>
<td>B</td>
</tr>
<tr>
<td>Brenner et al (2001)</td>
<td>Nondrinkers</td>
<td>0.95</td>
<td>0.57–1.59</td>
<td>0.50</td>
<td>0.30–0.84</td>
<td>Both sexes</td>
<td>B</td>
</tr>
<tr>
<td>Marques-Vidal et al (1996)</td>
<td>0</td>
<td>1</td>
<td>Case-control</td>
<td>561/643</td>
<td>MI</td>
<td>B</td>
<td></td>
</tr>
<tr>
<td>Cleophas et al (1996)</td>
<td>Nondrinkers</td>
<td>0.86</td>
<td>0.64–1.15</td>
<td>Men</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Theobald et al (2000)</td>
<td>Nondrinkers</td>
<td>0.4</td>
<td>0.1–1.8</td>
<td>Men</td>
<td>A</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bianchi et al (1993)</td>
<td>Nondrinkers</td>
<td>1.15</td>
<td>0.67–1.99</td>
<td>1.0</td>
<td>0.5–1.7</td>
<td>Both sexes</td>
<td>A</td>
</tr>
<tr>
<td>Simons et al (1996)</td>
<td>Nondrinkers</td>
<td>0.89</td>
<td>0.66–1.18</td>
<td>Men</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Salonen et al (1983)</td>
<td>$&lt;5$ bottles/week</td>
<td>1</td>
<td>1</td>
<td>Prospective</td>
<td>4063</td>
<td>MI fatal and nonfatal</td>
<td>B</td>
</tr>
<tr>
<td>$\geq 5$ bottles/week</td>
<td>0.5</td>
<td>0.5–1.4</td>
<td>Men</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

MI indicates myocardial infarction; N, number of patients, size of the cohort, or number of cases/number of controls; adjustment A, studies had not taken into account different types of alcoholic beverage; and adjustment B, bias of combined drinking was either excluded or taken into account.
of social class level, presence of ex-drinkers or light drinkers in the reference group, and use of the same reference group for both wine and beer.

Statistical Analyses

In the drinkers versus nondrinkers meta-analysis, data were combined using the general variance-based method that requires information on the relative risks (RR) or odds ratios estimate and their 95% CIs for each study. When no CIs were presented, they were calculated by transforming probability values (2 studies). The 95% CIs were used to assess the variance and the relative weight of each study. Adjusted RR, when available, was preferred. Publication bias was tested using funnel plot asymmetry. Results from subgroup analyses were reported with 99% CI to account for multiple comparisons. Data from studies reporting trend analysis were pooled with a weighted, least-squares regression model. In this method, the natural logarithm of the adjusted RR of vascular disease was regressed as a function of beverage intake. Midpoints of consumption categories were used for calculations. For open-ended, high-intake categories, the midpoint of the category was estimated to be 20% greater than the lower boundary specified by the original investigators. Study effect was modeled with indicator variables. This approach may be extended to fit a J-shaped trend, including linear and quadratic terms. The full model is as follows: log(RR) = γ × study + β1 × (beverage dose) + β2 × (square of beverage dose) + error. Statistical analyses were performed using the SAS package (version 8.2 for Windows). Statistical Analyses

Results

Wine

Drinkers Versus Nondrinkers Meta-Analysis

Thirteen studies on the association between wine intake and vascular risk (11 on CHD and 2 on cerebrovascular disease [CVD]) involved 201,308 persons (Table 1). Overall RR of drinkers with respect to nondrinkers was 0.68 (95% CI, 0.59 to 0.77; Figure 1). No heterogeneity was observed (P=0.10). Similar findings were obtained in prospective or case-control studies. Test for sample-size bias failed to show a funnel plot asymmetry (P=0.56). An extensive sensitivity analysis was performed (Table 2). The inverse association of wine with vascular risk remained statistically significant in pooling studies where either CHD or CVD were the only events considered or that separately considered either nonfatal vascular events or cardiovascular mortality. The RR of wine drinkers was also significantly reduced in studies that formally excluded ex-drinkers or “light or occasional” drinkers from the reference group or that had adjusted for different types of alcoholic beverages or for indicators of social class level or compared both wine and beer drinking groups with the same reference group. Six studies were conducted on men only, and meta-analysis

Figure 1. Odds ratios for vascular disease comparing wine intake versus no wine intake. Black squares indicate the odds ratio in each study, with the square sizes inversely proportional to the standard error of the odds ratio. Horizontal lines represent the 95% CI. The combined odds ratios are indicated by gray squares for subtotals and by a white square for grand total. The dashed vertical line shows the pooled estimate.
showed a RR of 0.87 compared with a RR of 0.53 in a pool of the other studies that were conducted on both sexes.

Dose-Response Meta-Analysis

Ten studies reported trend analysis of the association between different categories of wine intake and vascular risk (7 on CHD and 3 on CVD) involving 176,042 persons (Table 3). Dose-response curves (RRs at different amounts of wine intake) for each study are reported in Figure 2. The best fitting model includes a linear and a quadratic term and was used to construct an average dose-response curve. The complex relationship found was interpreted as a J-shaped curve because, after an initial progressive decrease in the vascular risk by increasing amounts of wine, the curve reaches a plateau at higher intake and tends to revert at the highest amounts explored. When only the 7 prospective studies were considered, the fitting of the quadratic model considerably improved, and this was used to construct the average dose-response curve in Figure 3. A maximum reduction was predicted at 750 mL/day, but statistical significance was only reached up to the amount of 150 mL/day. In subgroup analysis, studies considering CHD or CVD or cardiovascular mortality as separate end points showed similar J-shaped curves that did not reach statistical significance.

<table>
<thead>
<tr>
<th>Subgroup</th>
<th>Wine</th>
<th>Beer</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n  RR 99% CI</td>
<td>n  RR 99% CI</td>
</tr>
<tr>
<td>Overall</td>
<td>13 0.68 0.59–0.77*</td>
<td>15 0.78 0.70–0.86*</td>
</tr>
<tr>
<td>Type of cohort</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prospective studies</td>
<td>5 0.64 0.50–0.83*</td>
<td>8 0.79 0.67–0.94*</td>
</tr>
<tr>
<td>Case-control studies</td>
<td>8 0.71 0.56–0.90*</td>
<td>7 0.74 0.57–0.96*</td>
</tr>
<tr>
<td>Type of event</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coronary heart disease</td>
<td>11 0.71 0.59–0.85</td>
<td>13 0.79 0.68–0.91</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>2 0.43 0.24–0.78</td>
<td>2 0.67 0.41–1.10</td>
</tr>
<tr>
<td>Nonfatal vascular events</td>
<td>8 0.71 0.56–0.90</td>
<td>7 0.74 0.57–0.96</td>
</tr>
<tr>
<td>Cardiovascular mortality</td>
<td>2 0.49 0.34–0.70</td>
<td>3 0.76 0.55–1.05</td>
</tr>
<tr>
<td>Sex effect</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Only men</td>
<td>6 0.87 0.68–1.12</td>
<td>6 0.82 0.68–0.99</td>
</tr>
<tr>
<td>Both sexes</td>
<td>7 0.53 0.42–0.68</td>
<td>9 0.72 0.58–0.90</td>
</tr>
<tr>
<td>Adjustment for different types of alcoholic beverages</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not adjusted</td>
<td>3 0.53 0.39–0.73</td>
<td>4 0.79 0.62–1.01</td>
</tr>
<tr>
<td>Adjusted</td>
<td>10 0.75 0.61–0.93</td>
<td>11 0.77 0.65–0.92</td>
</tr>
<tr>
<td>Adjustment for indicators of social class</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not adjusted</td>
<td>3 0.78 0.56–1.08</td>
<td>3 0.68 0.41–1.14</td>
</tr>
<tr>
<td>Adjusted</td>
<td>10 0.64 0.52–0.79</td>
<td>12 0.78 0.68–0.91</td>
</tr>
<tr>
<td>Other</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No light or occasional drinkers in the reference group</td>
<td>10 0.73 0.59–0.91</td>
<td>11 0.80 0.66–0.97</td>
</tr>
<tr>
<td>No ex-drinkers in the reference group</td>
<td>5 0.61 0.47–0.79</td>
<td>5 0.77 0.63–0.94</td>
</tr>
<tr>
<td>Same reference group for both wine and beer</td>
<td>9 0.62 0.50–0.77</td>
<td>9 0.72 0.59–0.88</td>
</tr>
</tbody>
</table>

*95% CI.

Beer

Drinkers Versus Nondrinkers Meta-Analysis

Fifteen studies on the association between beer intake and vascular risk (13 on CHD and 2 on CVD) involved 208,096 persons (Table 1). Overall RR reduction in favor of beer drinkers was 0.78 (95% CI, 0.70 to 0.86; Figure 4). No heterogeneity (P=0.82) or funnel plot asymmetry (P=0.90) was observed. Similar results were observed in prospective and case-control studies and when studies in which CHD or nonfatal vascular events were considered separately. Significant results were also obtained by pooling studies that had adjusted for different types of alcoholic beverages or for indicators of social class or excluded ex-drinkers or light or occasional drinkers from the reference groups. The RR of beer drinkers was lower in the studies that included both sexes than in those with only men; both results were statistically significant.

Dose-Response Meta-Analysis

Seven studies reported trend analysis of the association between different categories of beer intake and vascular risk (5 on CHD and 2 on CVD); they involved 136,382 persons (Table 3). Dose-response curves for each study are reported in Figure 5. Both a linear and a quadratic model failed to
<table>
<thead>
<tr>
<th>Reference (Year)</th>
<th>Wine</th>
<th>Beer</th>
<th>Type of Study</th>
<th>Number of Patients</th>
<th>Outcome and Sex Distribution</th>
<th>Type of Adjustment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yano et al 34 (1977)</td>
<td>0 1</td>
<td>0 1</td>
<td>Prospective 7705</td>
<td>CHD fatal and nonfatal</td>
<td>A</td>
<td></td>
</tr>
<tr>
<td>Stampfer et al 35 (1988)</td>
<td>0 1</td>
<td>0 1</td>
<td>Prospective 87 526</td>
<td>CHD fatal and nonfatal</td>
<td>B</td>
<td></td>
</tr>
<tr>
<td>Truelsen et al 36 (1998)</td>
<td>0 1</td>
<td>0 1</td>
<td>Prospective 13 329</td>
<td>Stroke fatal and nonfatal</td>
<td>B</td>
<td></td>
</tr>
<tr>
<td>Grønbaek et al 37 (2000)</td>
<td>0 1</td>
<td>0 1</td>
<td>Prospective 24 523</td>
<td>CHD nonfatalit</td>
<td>B</td>
<td></td>
</tr>
<tr>
<td>Tavani et al 38 (1996)</td>
<td>0 1</td>
<td>0 1</td>
<td>Case-control 787/959</td>
<td>MI</td>
<td>A</td>
<td></td>
</tr>
<tr>
<td>Malarcher et al 39 (2001)</td>
<td>0 1</td>
<td>0 1</td>
<td>Case-control 224/392</td>
<td>Stroke</td>
<td>A</td>
<td></td>
</tr>
<tr>
<td>Farchi et al 40 (1992)</td>
<td>240 1</td>
<td>0 1</td>
<td>Prospective 1536</td>
<td>CHD mortality</td>
<td>A</td>
<td></td>
</tr>
<tr>
<td>Renaud et al 41 (1999)</td>
<td>0 1</td>
<td>0 1</td>
<td>Case-control 202/735</td>
<td>MI</td>
<td>B</td>
<td></td>
</tr>
<tr>
<td>Bianchi et al 42 (1991)</td>
<td>0 1</td>
<td>0 1</td>
<td>Case-control 298/685</td>
<td>MI</td>
<td>A</td>
<td></td>
</tr>
<tr>
<td>Theobald et al 43 (2000)</td>
<td>0 1</td>
<td>0 1</td>
<td>Prospective 1828</td>
<td>CHD mortality</td>
<td>B</td>
<td></td>
</tr>
<tr>
<td>Bobak et al 44 (2000)</td>
<td>&lt;71 1</td>
<td>&lt;71 1</td>
<td>Case-control 202/735</td>
<td>MI</td>
<td>B</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations and explanations as in Table 1. Data from Renaud et al 41 were kindly provided by Dr S. Renaud because of a printing mistake in the original publication.
show any significant relationship between different amounts of beer intake and vascular risk (Figure 5) when considering either all studies or subgroups. The apparent continuously decreasing risk with increasing beer consumption was not statistically significant at any amount of beer, eg, at 750 mL of daily beer intake, the predicted reduction of risk was 0.87 (95% CI, 0.57 to 1.33).

**Discussion**

Epidemiological studies have suggested that mortality and CHD are lower for people who drink low-to-moderate amounts of alcohol than for those who do not drink or who drink heavily. Evidence obtained from this meta-analysis indicates an average significant reduction of 32% of overall vascular risk associated with drinking wine. Not only were nonfatal vascular end points significantly reduced in wine drinkers, but so was cardiovascular mortality. In studies with only men, the protection offered by wine was surprisingly small (13%) and not significant, in contrast with studies enrolling both sexes (47%). Whether women are more susceptible to the benefit of wine or if they are more likely to drink lower amounts, thus taking its maximal advantage, remains to be established. In relation to the reported association between moderate alcohol consumption and increased risk of breast cancer, our finding suggests that the overall effect of moderate wine intake on women’s health may actually be favorable. In agreement with the relation between alcohol intake and all-cause mortality previously reported, we observed a J-shaped relationship between wine intake and vascular risk suggesting that light-to-moderate wine drinkers have lower vascular risk than either heavier drinkers or nondrinkers.

Beer drinking was also associated with a reduced risk of vascular events, although at an extent lower than that observed with wine. A significant inverse association was still apparent when only CHD was considered but, unlike with wine, it did not reach statistical significance when CVD events or cardiovascular mortality were separately evaluated, likely due to the small number of available studies. Risk reduction connected with beer drinking was smaller but, unlike wine, still significant in studies in which only men were included. This suggests that women might be particularly responsive to alcohol itself rather than to the nonalco-
holic components\textsuperscript{11} of these beverages. The most important difference between wine and beer consumption was observed in the meta-analysis of studies reporting trend analysis. In contrast with wine, the fitted models failed to show any significant relationship between different amounts of beer intake and vascular risk, even when different subgroups were analyzed. Thus, the inverse association between beer consumption and vascular risk observed in the drinkers versus nondrinkers meta-analysis should be interpreted with caution.

**Figure 4.** Odds ratios for vascular disease comparing beer intake versus no beer intake. Styled as Figure 1.

**Figure 5.** RRs or odds ratios for different categories of beer intake (dose-response curves). The black line indicates the predicted model using data from all studies. Considering all the studies, the best-fitting model was not statistically significant ($R^2=0.64$) including a negative linear term ($\beta_1=-1.8\pm2.9\times10^{-4}; P=0.54$); the inclusion of a quadratic term did not improve the fit. In prospective studies $\beta_1=-1.0\pm4.3\times10^{-4}$ ($P=0.83$).
Strength and Potential Limitations of This Meta-Analysis

The results of any meta-analysis, especially in nonexperimental epidemiology, may be invalid due to publication bias or confounding effect. This was not the case here, because no sample size bias could be shown by funnel plot, nor were the results affected by any of the adjustments considered. Self-reported wine or beer consumption is thought to be inaccurate. Underreporting on wine or beer drinkers would, however, result in a tendency for RRs to be biased toward the null hypothesis, whereas our meta-analysis showed significant associations. Errors in reporting beer intake might have contributed to our failure to draw any statistically significant dose-response curve from studies on this beverage.

Irregular (binge) drinkers may be frequent in cohorts of beer drinkers and might have obscured a possible dose-dependent risk reduction in regular beer drinkers. The choice of nondrinkers as a reference group has been questioned because this group may include ex-drinkers who have quit because of health problems. We performed a subanalysis restricted to studies that excluded either ex-drinkers or very light or occasional drinkers from the reference group, but the estimated overall risk still seemed to be significantly reduced. The relative 10% overall difference between the RR of wine versus beer drinkers was unchanged in studies that assessed both wine and beer drinking versus the same reference group. However, the potential confounding effect of the combined drinking of different types of alcoholic beverages in the same population was excluded by pooling data from studies that had taken this issue into consideration. Uncontrolled confounding by other known risk factors can also be reasonably excluded because the great majority of studies were adjusted for these variables; in particular, the overall results were confirmed by analyzing subgroups from studies that also adjusted for indicators of social class.

Conclusions

Association does not prove cause and effect, but the presence of a dose-response relation in wine meta-analysis, although of a complex, J-shaped type, is of importance. Definite proof could only be obtained by large long-term intervention trials. Such trials seem to be unfeasible for several reasons, including ethical concerns. The evidence for the benefit connected with wine consumption should, therefore, critically include molecular and cell biology studies, animal and observational epidemiological studies, and their meta-analyses. On this basis, what should cardiologists advise their patients regarding wine or beer consumption? First, patients and their relatives should be informed of what lifestyle changes might be beneficial to them. Besides insisting on the control of risk factors, abstainers should be informed that in the absence of contraindications and in the context of healthy eating and lifestyle, low-to-moderate wine consumption may contribute to better health. People who are already regular light-to-moderate wine consumers should be encouraged to continue. The hazards of excess drinking should always be highlighted, and heavy drinkers should be pushed to cut their consumption to a moderate level.

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