Coronary Atherosclerosis and Alcohol Consumption

Angiographic and Mortality Data

Romana Femia, Andrea Natali, Antonio L’Abbate, Ele Ferrannini

Objective—Moderate alcohol consumption is associated with reduced cardiovascular disease (CVD) risk. Whether this protection is based on a lesser degree of coronary atherosclerosis has not been established.

Methods and Results—We studied 1676 men and 465 women consecutively undergoing coronary angiography. A score (ATS) was calculated by summing the percent lumen narrowing of all main vessels; alcohol consumption was quantitated by questionnaire. In univariate analysis, ATS was significantly (P=0.001) associated with male sex, age, familial CVD, smoking, diabetes, hypertension, and serum cholesterol levels; alcohol consumption was associated with less frequent diabetes (P<0.001) and lower ATS (P=0.02). By multivariate analysis, alcohol intake was associated with lower ATS (P<0.01) independently of the other risk factors; the estimated effect size was comparable to that associated with a 1-mmol decrement in serum cholesterol. Over a median follow-up of 93 months, 37 women and 194 men died from a cardiac cause. By Cox analysis, positive predictors for cardiac mortality were male sex (hazard ratio [HR], 1.7; 95% confidence interval [CI], 1.1 to 2.6), age (HR, 2.1; 95% CI, 1.8 to 2.5 per decade) and diabetes (HR, 1.7; 95% CI, 1.2 to 2.4), whereas alcohol consumption was the only negative predictor (HR, 0.84; 95% CI, 0.71 to 1.00).

Conclusions—In a selected high-risk population, moderate alcohol consumption was independently associated with less coronary atherosclerosis and lower risk for cardiac mortality. (Arterioscler Thromb Vasc Biol. 2006;26:1607-1612.)

Key Words: alcohol ■ angiography ■ coronary atherosclerosis

Atherosclerotic cardiovascular disease (CVD) still is the leading cause of death and loss of disability-adjusted life years. A large number of epidemiological studies have documented that moderate alcohol use is protective against CVD.1 In a recent analysis of the modifiable risk factors for acute myocardial infarction in 52 countries,2 regular alcohol consumption was independently associated with reduced incident myocardial infarction in both sexes, at all ages and in all regions. A number of mechanisms have been postulated to explain this association, including an increase in serum high-density lipoprotein (HDL) and apolipoprotein-A1 levels,3 anticoagulant effects—by either direct platelet inhibition or inhibition of the fibrinolytic system4–6—a reduction in inflammation,7 and an enhancement of insulin sensitivity.8

The physiological basis for the protective effect of alcohol is, however, less certain. The 6 angiographic studies in the English literature9–14 (Table I, please see http://atvb.ahajournals.org) have included only men (n=1100), are of limited size, have used variable adjustment for relevant CVD risk factors, and mostly (5 of 6) lack follow-up data. This prompted the present study, in which the association of alcohol consumption with angiographically quantified coronary atherosclerosis was analyzed in a large cohort of men and women, in whom information on classical CVD risk factors and clinical outcome was available.

Materials and Methods

Patients

From 1983 to 1992, 2141 of the 4754 patients admitted to our coronary division underwent coronary angiography as part of the clinical work-up of symptoms or signs of heart disease. The indications for angiography were: acute myocardial infarction (n=43), previous myocardial infarction (n=213), cardiomyopathy (n=118), valvular disease (n=105), arrhythmias (n=30), unstable angina (n=34), acute pulmonary edema (n=6); the remainder of the patients had signs or symptoms of myocardial ischemia. Clinically, 52% of men and 56% of women presented with angina on effort, whereas 75% of men and 26% of women presented with resting angina; 56% of the cases presented with both. Since 1983, the relevant clinical data of each patient have been transferred immediately after discharge to a database created for this purpose. Each patient record consisted of the following blocks of information: (1) demographic data (sex, age, height, weight, years of school education); (2) clinical data (history of resting or effort angina, myocardial infarction, acute cerebrovascular events, coronary bypass surgery, and coronary angioplasty); (3) angiographic data. Any stenosis in a main coronary artery was graded in 1 of 4 levels (50%, 75%, 90%, and 100%); stenoses smaller than 50% were defined as irregularities. Clinically significant obstruction is defined as a narrowing by at least 50% with respect to the prestenotic segment, the grade of the narrowest one was considered. These descriptions constituted the basis for the diagnosis of 1-vessel, 2-vessel, or 3-vessel disease.

Clinical Chart Review

From a detailed review of the clinical charts, the following variables were added. Family history of ischemic heart disease, expressed as...
present or absent according to whether ≥1 first-degree relatives were affected. For the first variable, a score was also derived as the sum of all affected relatives (scored as 1 for parent, 1/n for each affected of n siblings, 0.5 for each second-degree relative; values were multiplied by 5 if the subject was affected before age 60 years). Patients were classified as cigarette smokers, past smokers, or nonsmokers. Cigarette consumption (in pack-years) was estimated by multiplying the number of packets per day by the years of smoking. Patients with either a previous diagnosis of hypertension or diabetes mellitus (n = 12) were excluded from the analysis. Patients were classified as having known diabetes if they reported a previous diagnosis or were receiving antidiabetic treatment. Patients with type 1 diabetes mellitus (n = 12) were excluded from the analysis. Patients with ≥2 consecutive fasting plasma glucose concentrations >7.0 mmol/L and no history of diabetes were classified as having newly diagnosed diabetes. Individual alcoholic beverage consumption was assessed by a standardized questionnaire. Questions about intake of beer, wine, and spirits were included separately. Total alcohol intake was calculated in grams by adding usual intake of alcoholic beverages, assuming the following content: 1 drink of beer, 12.8 g; 1 drink of wine, 11.0 g; and 1 drink of spirits, 14.0 g. From alcoholic beverages, alcohol intake was calculated in grams by adding usual intake of alcohol to the routine chemistry performed on admission, the following data were taken: plasma glucose, total cholesterol, and triglycerides. In addition to grading coronary disease in terms of clinically significant obstruction, the overall atherosclerotic involvement of the coronary vasculature was quantified by a modification of a method proposed previously. In the original descriptions of the angiograms, an index (ATS score) was calculated by summing the percent narrowing of all stenoses, including multiple stenoses along the same main vessel or those on secondary branches (irregularities were taken as 40% stenosis). This ATS score was calculated for each of the main coronary vessels as well as for the entire visible coronary tree.

### Follow-Up
Information concerning death, new acute myocardial infarction, and surgical or transluminal coronary revascularization was collected yearly up to 10 years after discharge from the following sources: outpatient visits, telephone interviews (with the patients, close relatives or the referring physician), questionnaires sent to all patients, and death certificates. Information concerning the events were classified as follows: no event, nonfatal myocardial infarction, cardiac death (which included sudden death, acute left ventricular dysfunction, fatal myocardial infarction and death during or immediately after coronary by-pass surgery), and noncardiac death (which included cancer, accident, stroke, acute pulmonary disease, etc). Of the 2141 patients, 24 subjects were lost to follow-up. Death from all causes was verified in 293 men and 60 women; death from cardiovascular causes was verified in 194 men and 37 women; acute nonfatal myocardial infarction occurred in 144 patients, and an additional 68 patients had a fatal myocardial infarction.

### Methods
Cardiac catheterization was performed according to the Judkins technique. Left ventricular end diastolic and aortic pressures were measured routinely during catheterization. Selective left and right coronary angiography and left ventriculography were performed (in that order), with multiple views (2 for the ventriculography and 6 for the coronaries). The images were recorded on 35-mm film and reviewed by the 2 hemodynamists who had performed the invasive study. Echocardiography was performed as described previously.

### Statistical Analysis
Values are given as means±SD. Between group mean differences were tested by the unpaired t test (or ANOVA) or the χ² test, for continuous and nominal variables, respectively. Serum triglycerides, cigarette and alcohol consumption, and the ATS score are given as median and [interquartile range] and were logistically transformed for statistical analysis. Multiple regression and logistic analyses were performed by standard methods; results are given as regression coefficient (mean±SE) and odds ratio (point estimate and 95% confidence interval [CI]), respectively. Multivariate analysis of mortality was performed using Cox proportional hazards analysis; results are expressed as hazard ratio (HR) and 95%CI.

### Results
Table 1 shows the clinical characteristics of the study cohort stratified by sex and presence (ATS+) or absence (ATS−) of any angiographically documented coronary narrowing. In comparison with ATS− subjects, ATS+ men and women were older, more often diabetic or hypertensive, or smokers

<table>
<thead>
<tr>
<th>Table 1. Clinical Characteristics by Sex and Presence (ATS+) or Absence (ATS−) of Coronary Atherosclerosis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Men</strong></td>
</tr>
<tr>
<td><strong>ATS−</strong></td>
</tr>
<tr>
<td>-----------</td>
</tr>
<tr>
<td>Age, y</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
</tr>
<tr>
<td>Familial IHD, %</td>
</tr>
<tr>
<td>Diabtes, %</td>
</tr>
<tr>
<td>Hypertesion, %</td>
</tr>
<tr>
<td>Smoking, n/ex/c, in %</td>
</tr>
<tr>
<td>Cigarette consumption, pack-years</td>
</tr>
<tr>
<td>Systolic BP, mm Hg</td>
</tr>
<tr>
<td>Diastolic BP, mm Hg</td>
</tr>
<tr>
<td>Total cholesterol, mmol/L</td>
</tr>
<tr>
<td>Triglycerides, mmol/L</td>
</tr>
</tbody>
</table>

*P<0.05 or less for the comparison ATS+ vs ATS− by χ² analysis or ANOVA (for nominal and continuous variables, respectively).

§Significantly (P<0.05 or less) different between women and men by 2-way ANOVA.
or dyslipidemic (higher serum total cholesterol or triglyceride concentrations). A family history of ischemic heart disease was more prevalent in ATS+ men but not in ATS+ women.

Among men, 24% were nondrinkers as compared with 59% of women (P<0.0001); however, average alcohol consumption was comparable to that of previous studies (Table 1), and very few subjects habitually consumed >500 g of alcohol per week and men generally consumed more alcohol than women. By using the sex-specific median alcohol consumption (231 g/wk in men and 154 g/wk in women), we stratified subjects into nondrinkers, light drinkers (below the median) and moderate drinkers (above the median); their clinical characteristics are shown in Table 2. Alcohol consumption was associated with significantly less frequent diabetes in both women and men, and with less frequent hypertension (and lower systolic and diastolic blood pressure values) in women only. Current smoking was more prevalent among moderately drinking men. Alcohol consumption was associated with higher serum total cholesterol concentrations. Serum triglycerides, however, tended to increase in men and decrease in women across drinking status (P=0.06 for the interaction). The ATS score was smaller in women than men and, in either sex, decreased across drinking category. The percentage of subjects with clinically significant obstruction in a major coronary vessel (single vessel or multivessel disease) generally decreased across drinking status (χ²=22.3, P=0.0002); this effect fell short of statistical significance when sexes were separated separately.

In univariate analysis, the ATS score was significantly (P≤0.001) associated with the classical CVD risk factors (male sex, age, familial ischemic heart disease, smoking, presence of diabetes or hypertension and serum total cholesterol levels); alcohol consumption was associated with a lower ATS (P=0.02). In multivariate analysis (Table 3), moderate alcohol consumption was associated with a lower ATS score independently of the other CVD risk factors. In this model, the interaction between sex and alcohol consumption was not significant (F=1.62, P=0.2). The predicted effect size was comparable to that associated with a 1-nmol decrement in serum total cholesterol concentrations. When the analysis was performed by using presence or absence of angiographically documented coronary atherosclerosis (ATS+ or ATS−) as the dependent variable, the odds ratio for moderate alcohol consumption was 0.75 (95% CI, 0.56 to

### TABLE 2. Clinical Characteristics by Sex and Drinking Status

<table>
<thead>
<tr>
<th></th>
<th>ND</th>
<th>LD</th>
<th>MD</th>
<th>ND</th>
<th>LD</th>
<th>MD</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>398</td>
<td>592</td>
<td>686</td>
<td>274</td>
<td>71</td>
<td>120</td>
</tr>
<tr>
<td>Age, y</td>
<td>54±10</td>
<td>55±10</td>
<td>54±10</td>
<td>57±11</td>
<td>59±9</td>
<td>57±9§</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>26.6±3.4</td>
<td>25.9±2.9</td>
<td>26.4±3.0</td>
<td>26.5±3.7</td>
<td>26.6±3.8</td>
<td>26.0±4.0</td>
</tr>
<tr>
<td>Familial IHD, %</td>
<td>42</td>
<td>41</td>
<td>39</td>
<td>45</td>
<td>46</td>
<td>51</td>
</tr>
<tr>
<td>Diabetes, %</td>
<td>15</td>
<td>11</td>
<td>7</td>
<td>20</td>
<td>13</td>
<td>8</td>
</tr>
<tr>
<td>Hypertension, %</td>
<td>40</td>
<td>39</td>
<td>38</td>
<td>43</td>
<td>54</td>
<td>33**</td>
</tr>
<tr>
<td>Smoking, n/ex/c; in %</td>
<td>19/59/22</td>
<td>17/62/21</td>
<td>10/61/29**</td>
<td>72/18/9</td>
<td>69/15/15</td>
<td>65/19/16</td>
</tr>
<tr>
<td>Systolic BP, mm Hg</td>
<td>128±14</td>
<td>129±14</td>
<td>128±14</td>
<td>128±15</td>
<td>131±17</td>
<td>124±14**</td>
</tr>
<tr>
<td>Diastolic BP, mm Hg</td>
<td>81±8</td>
<td>81±8</td>
<td>81±8</td>
<td>79±8</td>
<td>80±9</td>
<td>77±7**</td>
</tr>
<tr>
<td>Total cholesterol, mmol/L*</td>
<td>5.2±1.2</td>
<td>5.4±1.5</td>
<td>5.6±1.3</td>
<td>5.6±1.4</td>
<td>5.6±1.4</td>
<td>5.7±1.3§</td>
</tr>
<tr>
<td>Triglycerides, mmol/L*</td>
<td>1.66 [0.98]</td>
<td>1.65 [0.93]</td>
<td>1.72 [1.02]</td>
<td>1.44 [0.91]</td>
<td>1.38 [0.72]</td>
<td>1.29 [0.69]§</td>
</tr>
<tr>
<td>Alcohol consumption,* g/wk</td>
<td>0</td>
<td>154 [64]</td>
<td>308 [216]</td>
<td>0</td>
<td>77 [0]</td>
<td>154 [41]§</td>
</tr>
<tr>
<td>ATS, score units*</td>
<td>255±207</td>
<td>273±217</td>
<td>234±197</td>
<td>143±205</td>
<td>115±181</td>
<td>115±178§</td>
</tr>
<tr>
<td>N° diseased vessels, 0/1/2</td>
<td>25/32/43</td>
<td>23/34/43</td>
<td>24/38/39</td>
<td>56/15/29</td>
<td>67/18/15</td>
<td>61/14/26</td>
</tr>
</tbody>
</table>

* Regression coefficients (±SE) and their statistical significance in a multivariate analysis of the dependence of ATS score (log-transformed) on risk factors.

ND indicates light drinker (<231 g/wk in men, <154 g/wk in women); LD, moderate drinker (≥231 g/wk in men, ≥154 g/wk in women); ND, nondrinker.

*P<0.05 for the comparison across category of drinking habit.
§Significantly (P<0.05) different between women and men by 2-way ANOVA.
**P≤0.05 in women or men only.
0.99) (Figure 1); adding diabetes treatment had no effect whereas adding antihypertensive treatment yielded an additional independent risk (OR, 1.60; 95% CI, 1.25 to 2.04) without weakening the effect of alcohol use.

Over a median follow-up period of 93 months, a total of 60 women and 289 men died from all causes; significant predictors of total mortality were male sex (HR, 1.5; 95% CI, 1.0 to 2.1), age (HR, 2.0; 95% CI, 1.7 to 2.3 per decade), diabetes (HR, 1.6; 95% CI, 1.2 to 2.1), and smoking (HR, 1.4; 95% CI, 1.3 to 1.9). When considering cardiac mortality alone (37 women and 194 men), presence of any degree of coronary atherosclerosis was a strong predictor in both men and women (P<0.001). When cardiac mortality was related to the number of vessels (0, 1, or ≥2) with clinically significant stenosis, the protective effect of an alcohol consumption of ≥200 g per week was only evident in the patients with multivessel disease (Figure 2). By a Cox proportional hazards analysis that included indications for angiography, male sex (HR, 1.7; 95% CI, 1.1 to 2.6), age (HR, 2.1; 95% CI, 1.8 to 2.5 per decade), and diabetes (HR, 1.7; 95% CI, 1.2 to 2.4) were significant positive predictors, whereas alcohol consumption (as a continuous variable) was a significant negative predictor (HR, 0.84; 95% CI, 0.71 to 1.00) (Figure 3). In this model, the sex–alcohol consumption interaction was not significant (χ²=0.07, P=0.79). In addition, introducing ATS into this model reduced the predictivity of alcohol consumption (HR, 0.86; 95% CI, 0.73 to 1.02).

**Figure 1.** Logistic regression analysis of factors associated with presence or absence of coronary atherosclerosis (any narrowing on visible vessels by quantitative angiography).

**Figure 2.** Cumulative cardiac survival in patients with 0 vessel, 1 vessel, or ≥2 vessel disease by drinking habits (cutoff of alcohol consumption: 200 g/wk).

**Figure 3.** Predictors of cardiac mortality by Cox proportional hazards analysis.

### Discussion

The main result of the present analysis is that, in a referral population of subjects at high risk for CVD alcohol consumption was associated with less coronary atherosclerosis at angiography and reduced cardiac mortality at follow-up. Specifically, after accounting for all other major CVD risk factors moderate alcohol consumption (in our cohort, <231 g/wk in men, <154 g/wk in women) was associated with a reduction in the ATS score comparable to that predicted by a 1-mmol decrement in serum total cholesterol concentrations. Although the data suggest that lighter alcohol consumption may not carry such benefit, a clear threshold or J-shaped relationship could not be identified because our cohort included too few women (who drank less than men) and very few heavy drinkers. Also, the ATS score decreased across drinking category somewhat, but the change was more evident in women than in men, ie, in the smaller group. Accordingly, when the multivariate analysis of ATS predictors was performed separately in men and women, there was a suggestion that in women the protective effect of alcohol (OR, 0.67) was stronger than in men (OR, 0.87), but this difference was not fully supported statistically. Finally, the effect of alcohol consumption on coronary artery disease was seen both when using ATS, ie, the total extent of lumen narrowing in the visible vasculature and by using the criterion of clinically significant obstruction, ie, the tightest of all visible stenoses along a major branch. Of note is that the effect of alcohol was independent of the confounding influence exerted by much stronger determinants such as age, smoking, and diabetes.

In our cohort, cardiac mortality was predicted by the expected set of risk factors (Figure 3) and was clearly related to the severity of coronary atherosclerosis in men as well as in women. Furthermore, there was a graded increase in cardiac deaths in subjects with ≥1 vessel involvement on the angiogram. This finding implies that, although cardiac mortality can be caused by a variety of causes (myocardial infarction, arrhythmias, and heart failure), the underlying atherosclerotic disease of the coronary vasculature is a common soil for any such cause. By analogy, the observed protection by alcohol consumption against cardiac mortality can be thought to be causally related to the less extensive atherosclerotic disease of the coronary arteries. In fact, when ATS was included among the predictors of cardiac death, the protective effect of alcohol was attenuated. Of note, reduced
cardiac mortality with an alcohol consumption of ≥200 g per week was clearly seen in patients with multivessel disease, whereas this effect was not evident in patients with single vessel disease. Although in general any effect is more readily demonstrable in higher-risk than in low-risk conditions, this finding may also suggest that the mechanism by which alcohol exerts its anti-atherogenic action relates to the progression of the disease rather than its initiation. With regard to this, the only serial angiographic study relevant to this issue, performed in a small cohort of Swedish women hospitalized for an acute coronary syndrome, has documented a slower progression of coronary atherosclerosis in women drinking moderate amounts of alcohol (>35 g/wk) as compared with both lighter drinkers and abstainers.

Collectively, our findings provide strong support for the notion that moderate alcohol consumption is associated with a degree of protection of the coronary vasculature from atherosclerotic involvement, and that this effect translates into a modest reduction in risk of cardiac death over a period of several years. Clearly, this conclusion cannot be readily extrapolated from a Coronary Care Unit referral population to the general population, in which quantitative assessment of coronary atherosclerosis is not feasible (at least by invasive methods). The finding, however, that regular alcohol consumption is independently associated with fewer clinical events (incident myocardial infarction) in both sexes, at all ages and in all regions does imply that reduced coronary atherosclerosis may be the underlying reason also in the general population.

It is of interest that in our cohort there was relatively little difference in the clinical phenotype between drinkers and nondrinkers (Table 3). Thus, age, adiposity, familial ischemic heart disease, and smoking habits were similar across drinking status in both men and women. The only clinically relevant phenotypic differences were the lower prevalence of known diabetes in drinkers versus nondrinkers (in men and women) and the lower prevalence of hypertension (in women only). With regard to this, there is ample evidence that moderate alcohol consumption reduces the risk of both prevalent and incident diabetes. Furthermore, moderate alcohol consumption reduces CVD risk in diabetic patients to the same extent than in nondiabetic subjects. Among the mechanisms invoked to explain this protection, enhanced insulin sensitivity and attenuated low-grade inflammation—both predictors of incident diabetes—have been proposed to mediate the effect of alcohol on glucose homeostasis. Notably, the favorable effect of moderate alcohol consumption on insulin action has been particularly well-documented (using a prospective experimental protocol) in nondiabetic postmenopausal women. However, moderate alcohol consumption has been reported to be associated with less hypertension in women. This finding has not been confirmed in other studies, and alcohol consumption is associated with obesity, which itself a risk factor for hypertension.

With regard to other potential mediators of the effects of moderate drinking, the serum lipid profile has received much attention. Most epidemiological studies concur to show that alcohol consumption is associated with higher serum concentrations of both HDL cholesterol and triglycerides. Likewise, a meta-analysis of short-term alcohol administration protocols shows that alcohol intake raises both HDL cholesterol and triglycerides within a period of 1 to 9 weeks. This dual effect may result from an alcohol-induced increase in post-prandial triglyceride-rich lipoprotein flux and removal, which secondarily raises HDL cholesterol and reverse cholesterol transport. In our cohort, serum HDL cholesterol levels were unfortunately missing in many cases, but the increasing pattern of total cholesterol concentrations across drinking status may reflect increasing HDL cholesterol levels. Serum triglycerides, however, increased across category of drinking status in men but not in women, in whom the inverse trend was observed (Table 3). The reasons for the sex difference in the association between alcohol intake and serum triglycerides are unclear and may simply be caused by confounding. However, differences in frequency of alcohol intake, levels of sex hormone-binding globulin, and genetic polymorphism in the apolipoprotein CIII, apolipoprotein E, and other factors controlling lipid metabolism may be involved.

In conclusion, in a selected high-risk population, moderate alcohol consumption was independently associated with less coronary atherosclerosis and reduced cardiac mortality.

Disclosures

None.

References


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Table I. Studies of the association between alcohol consumption and coronary atherosclerosis

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<tr>
<th>Reference</th>
<th>Subjects</th>
<th>Mean age (years)</th>
<th>Alcohol consumption</th>
<th>Quantitation of atherosclerosis</th>
<th>Follow-up</th>
<th>Confounders</th>
<th>Conclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Barboriak JJ et al. [9]</td>
<td>718 men</td>
<td>52</td>
<td>Not specified</td>
<td>Quantitative coronary angiography using a score ranging from 0 (no occlusion) to 300 (occlusion all main branches)</td>
<td>none</td>
<td>Age, Smoking, Blood pressure, Plasma cholesterol, Plasma triglycerides, HDL cholesterol</td>
<td>Less coronary occlusion among moderate drinkers than abstainers.</td>
</tr>
<tr>
<td>Gruchow HW et al. [10]</td>
<td>526 men</td>
<td>54</td>
<td>Non-drinkers</td>
<td>Quantitative coronary angiography using a score ranging from 0 (no occlusion) to 300 (occlusion all main branches)</td>
<td>none</td>
<td>Age, BMI, Smoking Blood pressure, Plasma cholesterol, Plasma triglycerides, HDL cholesterol</td>
<td>Higher levels of occlusion found among non-drinkers, occasional drinkers, and regular drinkers with variable intake; significantly lower levels of occlusion in regular drinkers with consistent amounts.</td>
</tr>
<tr>
<td>Handa K et al. [11]</td>
<td>212 men</td>
<td>58</td>
<td>None Light (1-100 ml/week) Moderate (101-300) Heavy (≥ 300)</td>
<td>Quantitative coronary angiography (Gensini’s severity score)</td>
<td>none</td>
<td>Age, Smoking, Blood pressure, Diabetes Plasma cholesterol, Plasma triglycerides, HDL cholesterol, Uric acid Fibrinogen</td>
<td>Moderate alcohol consumption protective against severe coronary atherosclerosis.</td>
</tr>
<tr>
<td>Ducimetiere P et al. [13]</td>
<td>484 men</td>
<td>52</td>
<td>8 classes: from 0 to 150 ml/day</td>
<td>Quantitative coronary angiography using the Gravity score (range 0-181) originally used in CASS study</td>
<td>none</td>
<td>Age, Smoking, Blood pressure, Diabetes Plasma cholesterol, Plasma triglycerides, HDL and LDL cholesterol</td>
<td>Less severe extent of coronary atherosclerosis in alcohol consumers. Biphasic relationship between alcohol consumption and coronary stenosis, with a nadir at 101-125 ml/day.</td>
</tr>
<tr>
<td>Janszky I. et al. [14]</td>
<td>103 women</td>
<td>65</td>
<td>abstainers light (0.1-5 g/day) moderate (&gt; 5 g/day)</td>
<td>Quantitative coronary angiography</td>
<td>3 years</td>
<td>Age, BMI, Smoking CHD family history Diabetes, Hypertension, Hyperlipidemia, Menopausal status, Physical activity</td>
<td>Moderate alcohol consumption inversely associated with progression of coronary atherosclerosis</td>
</tr>
</tbody>
</table>