Blood lipids: the relationship with alcohol intake, smoking, and body weight

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Abstract

Study objective—The aim was to assess the interrelationship between alcohol intake, cigarette smoking, body weight, and blood lipid concentrations.

Design—This was the cross sectional (screening) phase of a prospective study. The main outcome measure was the blood lipids (serum total cholesterol, HDL cholesterol, and triglycerides).

Setting—General practices in 24 towns (The British Regional Heart Study).

Subjects—Subjects were 7735 men aged 40–59 years, selected at random from the age-sex registers of one group practice in each of the 24 towns.

Results—Univariate analysis showed little association between alcohol intake and total cholesterol, a strong positive relation with HDL cholesterol, and a significant increase in triglycerides in heavy drinkers. A strong positive association between alcohol intake and body weight was present in non-smokers but not in moderate/heavy smokers. With the exception of HDL cholesterol, the relationships between alcohol intake and serum lipids were significantly different in smokers and non-smokers, apparently due to the opposing effect of smoking on blood lipids and body weight. Total cholesterol and triglycerides were significantly and positively associated with alcohol intake in non-smokers, the cholesterol association being largely mediated by the influence of alcohol on body weight. In smokers, no such association was seen: current smokers who were heavy drinkers or non-drinkers had the lowest mean cholesterol levels.

Conclusions—The associations between alcohol intake and body weight and alcohol intake and blood lipids are strongly conditioned by cigarette smoking. Simple standardisation for smoking in multivariate analyses may obscure the independent relationship with alcohol. These findings are of importance in studies seeking to relate alcohol intake, body weight, or cigarette smoking to blood lipid concentrations, or blood lipid concentration to morbidity or mortality.

The consistent finding of a U shaped or inverse relationship between alcohol and cardiovascular disease has led researchers to explore the mechanisms by which alcohol may exert this apparently protective effect. Particular interest has focused on the relationship between alcohol and blood lipid concentrations. Alcohol is known to be an important factor influencing lipid metabolism, and observational and clinical studies have reported strong positive relationships between alcohol and high density lipoprotein (HDL) cholesterol, and to a lesser extent serum triglycerides. The association with total cholesterol has been less consistent. Some studies have reported positive associations between alcohol and serum total cholesterol while others have found no association. The association between alcohol and blood lipids is complicated by the strong interrelationship between alcohol and smoking, which has also been shown to have an influence on blood lipids. Smoking has been shown to be associated with lower HDL cholesterol. Several of the studies that have examined the alcohol-blood lipid relationship have not considered potential confounding factors. Where this has been done, the independence of the relationship with alcohol has been examined by standardising for smoking in multivariate analyses. Few studies have examined the simultaneous effects of smoking and alcohol on blood lipids. Because of the strong interrelationship between alcohol and smoking and the known association of both with body weight, which itself influences blood lipids, it is of importance to assess the interrelationships which alcohol, smoking, and body weight may have with blood lipids. This will help to determine the independence of such relationships.

Methods

The British Regional Heart Study (BRHS) is a large prospective study of cardiovascular disease involving 7735 men aged 40–59 years selected from the age-sex registers of one group general practice in each of 24 towns in England, Wales, and Scotland. The criteria for selecting the town, the general practice, and the subjects, as well as the methods of data collection, have been reported. Research nurses administered to each man a standard questionnaire which included questions on smoking habits, alcohol intake, and medical history. A number of physical measurements were made and blood was taken for measurement of biochemical variables and haematology.

ALCOHOL INTAKE

Alcohol consumption was recorded using questions on frequency, quantity, and type, similar to those used in the General Household Survey. The men were asked to indicate their category of

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Accepted for publication July 1991
current alcohol intake as none, on special occasions only, once/twice a week, or daily/most days. They were also asked how much they usually took per day when they drank—one to two drinks, three to six drinks, or more than six drinks. A drink was defined as a half pint of beer, one glass of wine or sherry, or a single tot of spirits. The men were classified into five groups according to their weekly alcohol intake: none, occasional (<1 drink per week), light (1–15 drinks per week), moderate (16–42), and heavy (>42). Heavy drinkers consisted of men who drank more than six drinks daily or most days.

SMOKING

The men were classified according to their current smoking status into six groups: those who had never smoked, ex-cigarette smokers, and four groups of current smokers (1–19, 20–21, 21–39, and ≥40 cigarettes/day, equivalent to <1 pack, 1 pack, 1–2, and ≥2 packs/day). Ex-cigarette smokers who currently smoked pipe/cigars are grouped as ex-cigarette smokers. Those men who only ever smoked pipe/cigars are included with "never smoked". In some of the analyses the term "non-smokers" is used to refer both to ex-smokers and men who had never smoked.

BODY MASS INDEX

Body mass index, calculated as weight/height² (kg/m²), was used as an index of relative weight. Each man was weighed in trousers and socks to the nearest 0.1 kg on an MSp110 field survey scale (beam balance) and height was measured without shoes to the nearest millimetre with a Harpenden stadiometer with digital meter. Body mass index was not available for three men. In some of the analyses the men have been grouped into five groups on the basis of equal intervals.

SERUM LIPIDS

All the blood samples were obtained in the non-fasting state between 0830 and 1830 hours. The estimations of serum total cholesterol and HDL cholesterol were carried out in the Wolfson Research Laboratories, Birmingham. Serum total cholesterol was measured by a modified Liebermann-Burchard method on a Technicon SMA 12/60 analyser. HDL cholesterol was measured after precipitation by magnesium/phosphotungstate, by the Liebermann-Burchard or by enzymic procedures. The triglyceride estimations were carried out at the Royal Free Hospital School of Medicine using an enzymic method. Triglyceride estimations were introduced after the survey had been completed in six towns and results are therefore available for 5675 men from the last 18 towns. Adjustments have been made for the marked diurnal variation in triglyceride measurement. Triglyceride concentrations have a highly skewed distribution, so log transformation and geometric means have been used. The distributions and interrelationships between these lipids have been published.

STATISTICAL METHODS

The analysis of variance was used to assess the difference in mean serum lipids and body mass index between the five alcohol categories and the smoking categories. Linear regression was used to assess the association between body mass index and the serum lipids. Tests for trend were assessed by assigning quantitative values to each level of alcohol intake (ie, none = 0, occasional = 1, light = 2, moderate = 3, heavy = 4). Tests for interactions were carried out using multiple regression analyses, fitting a smoking-alcohol interaction term in the appropriate multiple regression model with alcohol fitted continuously for total cholesterol and HDL cholesterol and as a 0,1 variable (heavy drinking = the rest) for triglycerides and smoking status as three categories. Adjusted means in table III were obtained using similar multiple regression models with a smoking-alcohol interaction term, fitting alcohol as five categories.

Results

Figure I shows the relationship between alcohol intake and the serum lipids. There was a tendency for total cholesterol to increase with increasing intake up to levels of moderate drinking but there was no significant difference overall in cholesterol concentrations between the five alcohol groups. The proportion of men with high cholesterol concentrations (≥7.2 mmol/litre) was greater in all drinkers than in non-drinkers and occasional drinkers. There was a significant positive association between alcohol and HDL cholesterol (p < 0.0001) with a 17% increase in heavy drinkers compared to non-drinkers. Light and moderate drinkers showed a 5% and a 10% increase respectively. The proportion of men with low concentrations of HDL cholesterol, a finding associated with an increased risk of ischaemic heart disease, decreased significantly with increasing alcohol intake. There was a tendency for triglycerides to increase with increasing alcohol intake (p < 0.01), although levels were only significantly raised in heavy drinkers.

![Figure 1](http://jehc.bmj.com/) Mean serum total cholesterol, HDL cholesterol, and triglycerides (±geometric) by alcohol intake with 95% confidence intervals and percentage of subjects in extreme fifth of distribution.
SMOKING, BODY MASS INDEX, AND BLOOD LIPIDS

Table I shows the relationship between smoking status and the serum lipids, and between body mass index and the serum lipids. Cigarette smoking had little influence on total cholesterol. There was a significant inverse association between cigarette smoking and HDL cholesterol. Men who had never smoked had significantly lower mean levels of triglycerides than ex-smokers and current smokers. There was little difference in triglycerides between ex-smokers and all current smokers. Body mass index showed a strong positive relation with cholesterol and triglycerides and a strong inverese association with HDL cholesterol.

ALCOHOL, SMOKING, AND BODY MASS INDEX

Alcohol intake showed a strong positive relation with body mass index (fig 2) which increased significantly from light to heavy alcohol intake (p < 0.0001). There was little difference between non-drinkers, occasional drinkers, and light drinkers. Previous studies have shown strong associations between alcohol and smoking, but although cigarette smokers tended to be heavier drinkers they had significantly lower body mass index than non-smokers (table I). Among current smokers there was no dose-response relationship with body mass index. Table II shows the association between cigarette smoking and the five alcohol intake groups. The proportion of current smoking increased with increasing alcohol intake and this was most apparent for moderate and heavy smoking. Because of the strong association between alcohol and smoking and the apparently opposing effects that alcohol and smoking exert on body weight, we have examined the relationship between alcohol and body mass index separately by smoking categories to assess the simultaneous effects of smoking and alcohol (fig 3). Current smokers (light and moderate/heavy) had a lower body mass index than non-smokers at almost all levels of alcohol intake. The strongest influence of alcohol on body weight was seen in non-smokers. Among light current smokers there was a tendency for body mass index to increase with increasing intake, although heavy drinkers did not have a particularly high mean body mass index. Among moderate/heavy smokers, alcohol had virtually no influence on body mass index, reflecting the stronger influence of smoking on body weight.

Table I: Mean serum lipids and body mass index (BMI) by smoking status and mean serum lipids by body mass index

<table>
<thead>
<tr>
<th>Smoking status</th>
<th>n</th>
<th>Total chol</th>
<th>HDL-C</th>
<th>Triglyceride</th>
<th>BMI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Never</td>
<td>1819</td>
<td>6.25</td>
<td>1.17</td>
<td>1.64</td>
<td>25.6</td>
</tr>
<tr>
<td>Ex</td>
<td>2715</td>
<td>6.35</td>
<td>1.16</td>
<td>1.78</td>
<td>26.0</td>
</tr>
<tr>
<td>1-19</td>
<td>1188</td>
<td>6.28</td>
<td>1.18</td>
<td>1.75</td>
<td>25.0</td>
</tr>
<tr>
<td>20</td>
<td>865</td>
<td>6.12</td>
<td>1.12</td>
<td>1.75</td>
<td>24.9</td>
</tr>
<tr>
<td>21-39</td>
<td>286</td>
<td>6.28</td>
<td>1.14</td>
<td>1.73</td>
<td>25.2</td>
</tr>
<tr>
<td>≥40</td>
<td>316</td>
<td>6.33</td>
<td>1.11</td>
<td>1.80</td>
<td>24.6</td>
</tr>
<tr>
<td>Test for difference between groups: NS</td>
<td>*</td>
<td>NS</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table II: Percentage distribution of smoking status categories within each drinking group

<table>
<thead>
<tr>
<th>Smoking status</th>
<th>Alcohol intake</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>None</td>
</tr>
<tr>
<td>Never</td>
<td>32 2</td>
</tr>
<tr>
<td>Ex</td>
<td>30 9</td>
</tr>
<tr>
<td>Light (&gt;1/9/d)</td>
<td>16 5</td>
</tr>
<tr>
<td>Moderate (&gt;2/0/d)</td>
<td>8 8</td>
</tr>
<tr>
<td>Heavy (&gt;2/1/d)</td>
<td>12 2</td>
</tr>
<tr>
<td>Current smoking</td>
<td>36 9</td>
</tr>
<tr>
<td>All men</td>
<td>100 100 100</td>
</tr>
</tbody>
</table>

Figure 2: Alcohol intake and mean body mass index (BMI) with 95% confidence intervals and percentage obese (BMI ≥ 28 kg/m²; top fifth of the BMI distribution)

Figure 3: Alcohol intake and mean body mass index (BMI) in men who had never smoked, ex-cigarette smokers, and current cigarette smokers (1-19 and ≥ 20/day)

ALCOHOL, SMOKING, AND SERUM LIPIDS

Because of the strong interrelationship between alcohol intake, smoking, and serum lipids we have examined the relationship between alcohol and serum lipids separately by smoking status (fig 4).

Total cholesterol

Among non-smokers (never smoked and ex-smokers) there was a positive association between alcohol intake and total cholesterol which reflects the body mass index pattern (p < 0.05). Among current smokers, heavy drinkers and non-drinkers had the lowest cholesterol levels. There was no difference between occasional, light, or moderate drinkers. A formal test for interaction
showed a significant difference in the alcohol-total cholesterol relationship with cigarette smoking (p < 0.05).

**HDL cholesterol**
Alcohol was positively associated with HDL cholesterol within all smoking categories. At each level of alcohol intake smokers had lower levels of HDL cholesterol than non-smokers (except in the heavy drinkers) which suggests that the relationship between alcohol and HDL cholesterol may be stronger than with cigarette smoking. The association between heavy drinking and HDL cholesterol appeared to be somewhat stronger in current smokers and a test for interaction showed a significant difference in the slope of the alcohol relationship with smoking status.

**Triglyceride**
Heavy drinking was associated with a significant increase in triglycerides (fig 1) but this was only apparent in non-smokers (fig 2). In current smokers there was no association between alcohol and triglycerides. A test for the difference in association with heavy drinking by smoking was marginally significant (p = 0.05).

**ADJUSTMENTS FOR BODY MASS INDEX**
The association between alcohol intake and serum lipids, particularly that seen for total cholesterol and triglycerides among non-smokers, may be mediated by its influence on body weight. In order to determine whether there is an independent association between alcohol and serum lipids we have examined the alcohol-blood lipid relationship by smoking, adjusted for body mass index. To illustrate the effects of adjustment and for simplification we have presented the adjusted and unadjusted mean levels of the serum lipids for occasional and heavy drinkers only, and the unadjusted and adjusted percentage difference in means between occasional and heavy drinkers (table III). The difference in total cholesterol levels between heavy and occasional drinkers was substantially reduced after adjustment for body mass index in non-smokers (never smoked and ex-smokers), but there was still a significant positive association. Since body mass index is inversely associated with HDL cholesterol, adjusting for it has strengthened the association between HDL cholesterol and alcohol intake in all smoking categories. The positive association between heavy drinking and triglycerides in non-smokers was substantially reduced after taking body mass index into account, but still remained significant.

**SOCIAL CLASS AS A POTENTIAL CONFOUNDING FACTOR**
An earlier report from this study has shown manual workers to have significantly lower serum cholesterol than non-manual workers (6.39 ± 6.23 mmol/litre, p < 0.0001). No significant association with social class was seen for HDL cholesterol or triglyceride. Since smoking and drinking are strongly associated with social class, we examined the alcohol-smoking-cholesterol relationships (fig 4) separately by the two main social classes. Within both manual and non-manual workers, the alcohol-smoking-cholesterol relationship was similar (data not shown). Adjusting for social class made a very small and non-significant difference to the relationship between alcohol and blood cholesterol within the smoking categories.

### Table III Unadjusted and body mass index (BMI) adjusted (in brackets) mean levels of blood lipids in occasional and heavy drinkers by smoking status: never smokers, ex-cigarette smokers, and current cigarette smokers

<table>
<thead>
<tr>
<th>Drinking</th>
<th>Never smoked</th>
<th>Ex-smokers</th>
<th>Current smokers</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mean (adj)</td>
<td>mean (adj)</td>
<td>mean (adj)</td>
</tr>
<tr>
<td><strong>Cholesterol</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>occasional</td>
<td>6.23 (6.23)</td>
<td>6.27 (6.25)</td>
<td>6.31 (6.33)</td>
</tr>
<tr>
<td>heavy</td>
<td>6.39 (6.31)</td>
<td>6.45 (6.36)</td>
<td>6.15 (6.17)</td>
</tr>
<tr>
<td>% difference</td>
<td>2.6% (1.3%)</td>
<td>2.9% (1.8%)</td>
<td>-2.5% (-2.5%)</td>
</tr>
<tr>
<td><strong>HDL Cholesterol</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>occasional</td>
<td>1.11 (1.11)</td>
<td>1.10 (1.10)</td>
<td>1.03 (1.02)</td>
</tr>
<tr>
<td>heavy</td>
<td>1.27 (1.31)</td>
<td>1.24 (1.27)</td>
<td>1.28 (1.27)</td>
</tr>
<tr>
<td>% difference</td>
<td>14% (18%)</td>
<td>13% (15%)</td>
<td>24% (25%)</td>
</tr>
<tr>
<td><strong>Triglyceride</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>occasional</td>
<td>1.62 (1.60)</td>
<td>1.74 (1.72)</td>
<td>1.79 (1.84)</td>
</tr>
<tr>
<td>heavy</td>
<td>1.97 (1.85)</td>
<td>2.03 (1.85)</td>
<td>1.72 (1.79)</td>
</tr>
<tr>
<td>% difference</td>
<td>22% (12.5%)</td>
<td>16% (7.6%)</td>
<td>-4% (-3%)</td>
</tr>
</tbody>
</table>

**Figure 4** Alcohol intake and mean total cholesterol, HDL cholesterol and triglycerides (*geometric*) in men who had never smoked, ex-cigarette smokers, and current cigarette smokers. Occ = occasional; mod = moderate

**ALCOHOL, HDL CHOLESTEROL, AND TRIGLYCERIDES**
An earlier report from this study has shown a strong inverse relationship between HDL cholesterol and triglycerides (r = 0.48, p < 0.0001). Since alcohol is a strong determinant of HDL cholesterol and high HDL cholesterol is associated with lower triglycerides, the association between
Alcohol and triglyceride will be counterbalanced by the strong association with HDL cholesterol. To assess the extent to which the relationship is associated with HDL cholesterol, we have examined the alcohol-triglyceride relation adjusting in addition for HDL cholesterol. After adjustment alcohol showed a stronger positive association with triglycerides in all smoking categories (table III). Heavy drinking was associated with a 25–35% rise in triglycerides compared to occasional drinking, the association being greatest in those who had never smoked.

Discussion
In the univariate results there was little association between alcohol and total cholesterol, a strong dose-response relationship between alcohol and HDL cholesterol, and a significant rise in triglycerides in heavy drinkers. These findings are similar to those of the US lipoprotein study which took no account of potential confounding variables. Because of the strong interrelationships between alcohol, smoking and body weight, it is difficult to interpret the association between alcohol intake and blood lipids in univariate analyses.

Alcohol, smoking, and body weight
The increase in body weight with heavier drinking seen in the present study (fig 2) is consistent with findings from several other studies. This was most evident in non-smokers and to a lesser extent in light smokers but was not seen in heavier smokers. These findings are similar to those of the Framingham study. The lack of association between alcohol intake and body weight seen in moderate/heavy smokers is likely to reflect both the opposing metabolic effect of smoking and possibly a different pattern of diet among heavy drinkers who smoke.

Alcohol, smoking, and serum lipids
Total cholesterol
The association between alcohol and total cholesterol has not been consistent, several studies showing no association while others show a small positive association. In the Poland–USA collaborative study a positive association between alcohol and total cholesterol was reported in Polish rural men but not in urban men, possibly due to differences in diet and smoking. When the alcohol-total cholesterol relationship was examined by smoking there was a small but significant positive association between alcohol and total cholesterol in men who are not currently smoking. The lowest total cholesterol concentrations are seen in current smokers who are heavy drinkers or non-drinkers. This is of interest as attention has been focused recently on the relationship between low blood cholesterol and increased mortality. It is well established that heavy drinking and cigarette smoking increase all cause mortality, and we have previously shown that middle aged British non-drinkers are predominantly ex-drinkers and have a high burden of cardiovascular and other disorders. Thus it is possible that the reported association between low blood cholesterol levels and increased mortality may be reflecting the burden of chronic disease encountered in heavy drinkers who are cigarette smokers and in ex-drinkers, particularly those who are current smokers. Though social class is strongly associated with both alcohol and smoking, and manual workers have significantly lower cholesterol concentrations, social class appeared to have little effect on the patterns of relationship observed. In the Framingham study a positive association between alcohol and total cholesterol in non-smokers but not in smokers was also observed but not commented on. The association in the present study seen in non-smokers, although significant, was very small and appeared to be partly due to the confounding effects of body weight.

Triglycerides
Some studies have shown a moderate increase in triglycerides in heavy drinkers, but others have not found any such association. These studies differ in their population structures and in their definitions of heavy drinking and they have usually controlled for confounding factors by standardisation in multivariate analyses. In the present study triglycerides were raised only in heavy drinkers who did not smoke, reflecting the alcohol-body weight relation with smoking status. Clinical studies have also shown a triglyceride response to alcohol only in obese heavy drinkers. After adjusting for body weight, the triglyceride increase in heavy drinkers in the present study was substantially reduced, but still remained significant.

In this study a strong inverse association has been observed between HDL cholesterol and triglycerides. In other studies this association is not often mentioned. Since most studies tend to show a stronger association between alcohol and HDL cholesterol than with triglycerides, the triglyceride response to alcohol will be counterbalanced by the associated rise in HDL cholesterol with alcohol. This was clearly seen in our study when, after adjusting for HDL cholesterol, heavy drinking was associated with a 25–35% rise in triglycerides in all smoking categories. The degree of independent association between alcohol and triglyceride is thus likely to be underestimated due to the opposing relationship with HDL cholesterol. Because obesity is associated with a lowering of HDL cholesterol and a rise in triglyceride levels, the triglyceride response to alcohol may be more apparent in obese people.

Diet as a potential confounding variable
Studies on alcoholics show that they have nutritional deficiencies and it is generally believed that heavy drinkers and heavy smokers tend to eat less than other subjects. Several studies have reported that in lighter drinkers alcohol is added to the diet while in heavy drinkers alcohol displaces other sources of energy intake. This is reflected by total cholesterol increasing up to moderate levels of drinking and then declining in heavy drinkers. This decline was marked in heavy drinkers who smoke. Indeed, in heavy drinkers who smoke, total cholesterol concentrations were lower than in lighter drinkers. Another possibility, already referred to, is that the lower total cholesterol reflects an increased burden of chronic disease in heavy drinkers who smoke. Total cholesterol and triglycerides are strongly determined by dietary intake and body weight and the differences in the response of these lipids to alcohol between smokers and non-smokers may be attributed, at least in part, to dietary differences.
ALCOHOL, SMOKING, AND HDL CHOLESTEROL

In the present study there was a strong dose-response relationship between alcohol and HDL cholesterol, evident in both smokers and non-smokers, although the magnitude of the increase was small. HDL cholesterol is not generally considered to be associated with diet.\(^2\) This strong association between alcohol and HDL cholesterol independent of smoking has been seen in the majority of studies. Though smoking is reported to lower HDL cholesterol, the effect was not seen in heavy drinkers, suggesting that drinking is the more influential factor on HDL cholesterol.

IMPLICATIONS

In epidemiological studies in which blood lipids are measured, simple standardisation for smoking status and or body weight is used to allow for their influence on the blood lipids. It is evident from the findings in this study that cigarette smoking has a profound effect upon the relationship between alcohol and body weight, so that a clear view of this relationship is only made possible by separately examining the several categories of smoking (fig 3). Similarly, cigarette smoking has a considerable effect on the relationship between alcohol intake and blood lipids, so that a proper understanding of the relationship is only possible when examined by the separate smoking categories (fig 4). Body weight, which has well established and strong relationships with all the blood lipids (table I), is also profoundly affected by cigarette smoking and this too must be taken into account when assessing the association between alcohol and blood lipids in the different smoking groups (table III). Given these complex interrelationships, simple standardisation of blood lipid levels in population groups for cigarette smoking status and/or body weight is not likely to provide a clear and detailed picture of the association between alcohol and blood lipids. It could lead to misinterpretation of the data and to invalid conclusions regarding the effects of alcohol. Such misinterpretations could well account for the inconsistent findings regarding alcohol and blood lipids and alcohol and body weight found in population studies. For individuals, failure to take account of the alcohol intake, smoking status, and body weight in assessing and monitoring blood lipid concentrations could also lead to misinterpretation and invalid conclusions. This is particularly true of lipid screening programmes in which the major concern is often with the blood lipid measurements alone rather than with the overall risk of coronary heart disease.

While HDL cholesterol concentrations are affected similarly by alcohol in all smoking categories, total cholesterol and triglycerides may be relatively low in heavy drinkers who are also current smokers. Thus "acceptable" levels of these lipid fractions may be recorded in individuals who for smoking reasons alone may be at increased risk of cardiovascular disease. Monitoring of changes in blood lipids in individuals regarded as having "unacceptable" levels also requires a knowledge not only of their changes in diet but also of changes in their smoking, alcohol intake, and body weight. Decisions made using the standard cut off points currently provided by many organisations may be inappropriate if due allowance has not been made for the simultaneous effects of these other factors.

The British Regional Heart Study is a British Heart Foundation research group and is also supported by the Department of Health and the Chest, Heart and Stroke Association. Goya Wannamethee has been supported by the Institute of Alcohol Studies. Serum total cholesterol and high density lipoprotein cholesterol concentrations were determined in the Wolfson Research Laboratories, Birmingham (Professor T P Whitehead). Triglyceride concentrations were determined in the Royal Free Hospital School of Medicine, by Mrs Ila Patel and Dr D G Cramp. We are grateful to Dr Andrew Phillips for his helpful advice in preparing this paper and to other members of the department for their comments.

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*J Epidemiol Community Health* 1992 46: 197-202
doi: 10.1136/jech.46.3.197

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