RESEARCH REPORT

Alcohol consumption and the incidence of type II diabetes

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Background: This study examines the relation between alcohol and type II diabetes and the possible mediating effects of HDL-cholesterol and serum insulin.

Methods: Prospective study of 5221 men aged 40–59 years with no history of coronary heart disease, diabetes, or stroke drawn from general practices in 18 British towns.

Results: During the mean follow up of 16.8 years there were 198 incident cases of type II diabetes. Occasional drinkers were the reference group. A non-linear relation was seen between alcohol intake and age adjusted risk of diabetes, with risk lowest in light and moderate drinkers and highest in heavy drinkers (quadratic trend p=0.03). Further adjustment for body mass index decreased risk in heavy drinkers. After additional adjustment for physical activity, smoking, and (undiagnosed) pre-existing coronary heart disease, only moderate drinkers showed significantly lower risk than occasional drinkers (RR=0.66 95% CI 0.44 to 0.99). Alcohol intake was inversely associated with serum insulin and positively associated with HDL-cholesterol. Adjustment for these factors reduced the “protective” effect in moderate drinkers (adjusted RR=0.73 95% CI 0.48 to 1.10) but the quadratic trend remained significant (p=0.02).

Conclusion: There is a non-linear relation between alcohol intake and the risk of type II diabetes. Serum insulin and HDL-cholesterol explained a small amount (20%) of the reduction in risk of type II diabetes associated with moderate drinking. The adverse effect of heavy drinking seemed to be partially mediated through its effect on body weight.
Blood lipids
All the blood samples were obtained in the non-fasting state between 0830 and 1830. Detailed information on blood lipid measurements have been published.27 High (raised) HDL-cholesterol and high blood glucose are defined as the concentrations in the top fifth of the distribution in the original 7735 men.

Blood glucose
Glucose was analysed by a glucose oxidase method using an automated analyser (Technicon SMA 12/60). Diurnal variation in glucose levels was modest and no adjustments were made for diurnal variation.28

Serum insulin
Serum insulin concentration was determined by a two site enzyme linked immunosorbent assay (ELISA) using commercially available monoclonal antibodies raised against human insulin (Novo Nordisk A/S: Denmark), which do not cross react with pro-insulin.29 Analyses were performed in the Department of Medicine, University of Newcastle upon Tyne, UK on non-fasting samples that had been stored at −20°C for between 13 and 15 years. In this laboratory no change in insulin levels was detected in repeat assays of 34 samples, stored at −20°C over an eight year period (mean difference 0.19 mU/l, p=0.5). Adjustments were made for the diurnal variation in insulin.29

High (raised) serum insulin is defined as the concentrations in the top fifth of the distribution in the 5221 men.

Serum insulin was adjusted for time of sampling using a simple mathematical approach that makes no assumptions about the form of the association between these variables and time of sampling. The log transformed data on these variables were adjusted for time of sampling, using the mean level of each variable for each hour in which samples were taken and the grand mean. The calculation for serum insulin was as follows: adjusted log insulin=(unadjusted log insulin−the grand mean log insulin for the hour of sampling)+ the grand mean log insulin. The 5221 men were divided into five groups on the basis of their estimated reported weekly intake.22

1 Non-drinkers (n=289).
2 Occasional (<1 unit/week) (n=1212).
3 Light (1–15 units/week). Weekend 1–2/day, 3–6/day, and daily 1–2 (n=1711).
4 Moderate (15–42 units/week). Weekend >6/daily and daily 3–6 (n=1425).
5 Heavy (>42 units/week). Daily >6 drinks/day (n=584).

In the analyses examining the relation between alcohol intake and incidence of diabetes, occasional drinkers are used as the baseline group as non-drinkers are a small and heterogeneous group unsuitable for this purpose.22

Biological measurements
Body mass index (BMI) calculated as weight/height2 was used as an index of relative weight. Obesity is defined as BMI ≥28.0 kg/m², the top fifth of the distribution in the original 7735 men.

Figure 1  Age adjusted event rate/1000 person years for type II diabetes in 5221 men aged 40–59 years over an average follow up of 16.8 years according to alcohol intake.

Pre-existing undiagnosed coronary heart disease
Men with evidence of CHD (undiagnosed) were defined as those with no recall of a doctor diagnosis of CHD but who had a WHO (Rose) questionnaire response indicating angina or possible myocardial infarction or ECG evidence of definite or possible myocardial ischaemia or myocardial infarction.27

Follow up
All men have been followed up for all cause mortality, cardiovascular morbidity, and development of type II diabetes from
the initial screening in January 1978–July 1980 to December 1995, a mean period of 16.8 years (range 15.5–18.0 years) and follow up has been achieved for 99% of the cohort. Information on death was collected through the established “tagging” procedures provided by the National Health Service registries. Evidence regarding diabetes and non-fatal heart attacks were obtained by reports from general practitioners, by biennial reviews of the patients’ notes through to the end of the study period, and from personal questionnaires to surviving subjects at the fifth year and twelfth year after initial examination. A non-fatal heart attack was diagnosed according to WHO criteria. A diagnosis of diabetes was not accepted on the basis of self completed questionnaire data unless confirmed in the primary care records.

Statistical methods
The Cox proportional hazards model was used to assess the effects of alcohol intake on the risk of type II diabetes incidence and risk of CHD. Alcohol intake was fitted as a categorical variable for the five groups (none, occasional, light, moderate, heavy). In some of the analyses, tests for linear trends across the categories of alcohol were assessed by assigning estimated quantitative median values to each level of alcohol intake (that is, none=0, occasional=0.5/week, light=7/week, moderate=29/week, heavy=56/week) and treating alcohol as a continuous ordinal variable. In addition, we considered non-linear trends by adding a quadratic alcohol term (square of the ordinal alcohol variable) to the model. In other analyses, we considered non-linear trends by treating alcohol as a continuous ordinal variable. In addition, tests for linear trends across the categories of alcohol were assessed by assigning estimated quantitative median values to each level of alcohol intake (that is, none=0, occasional=0.5/week, light=7/week, moderate=29/week, heavy=56/week) and treating alcohol as a continuous ordinal variable. In addition, we considered non-linear trends by adding a quadratic alcohol term (square of the ordinal alcohol variable) to the model. In other analyses, we considered non-linear trends by treating alcohol as a continuous ordinal variable.

RESULTS
During the mean follow up period of 16.8 years there were 198 incident cases of type II diabetes (2.6/1000 person years) in the 5221 men with data on alcohol intake and free of diagnosed CHD, stroke, or diabetes. Figure 1 shows the relation between alcohol intake and age adjusted rates for type II diabetes. A non-linear relation was seen between alcohol intake and diabetes after age adjustment with the lowest rates in moderate drinkers (quadratic trend p=0.01). Heavy drinkers showed the highest rates followed by non-drinkers. Heavy drinkers showed significantly higher risk than moderate drinkers (RR=1.71 p<0.05). Adjustment for BMI diminished the increased risk in heavy drinkers (table 1), which now did not differ from occasional or non-drinkers but the quadratic trend remained significant (p=0.03). Further adjustment for potential confounders—that is, social class, physical activity, smoking and CHD (undiagnosed)—reduced the risk in heavy drinkers slightly but the quadratic trend remained significant (p=0.03) and moderate drinkers showed significantly lower risk than occasional drinkers (p=0.04) (table 1).

Alcohol intake and metabolic risk factors
Table 2 shows the relation between alcohol intake and serum HDL-cholesterol and serum insulin concentrations. These relations have been adjusted for lifestyle characteristics and follow up time in a time varying analysis. Direct standardisation was used to obtain age adjusted rates per 1000 person years using the study population as the standard. To determine the possible mediating factors, serum insulin and HDL-cholesterol were fitted in turn to the multivariate model that included age, BMI, smoking, social class, alcohol intake, and pre-existing CHD (undiagnosed). Logistic regression was used to assess the odds of having increased levels of serum insulin and HDL-cholesterol. Analysis of covariance was used to obtain adjusted mean HDL-cholesterol and serum insulin by the five groups of alcohol. Tests for linear trends were obtained by treating alcohol as a continuous ordinal variable.

Table 2  Alcohol intake and unadjusted (SD) and adjusted mean levels of serum HDL-cholesterol (mmol/l) and serum insulin (mU/l) (*geometric mean) in men with no diagnosed CHD, stroke, or diabetes. Adjusted for age, BMI, social class, smoking, physical activity, and pre-existing CHD (undiagnosed)

<table>
<thead>
<tr>
<th>Alcohol intake</th>
<th>Non</th>
<th>Occ</th>
<th>Light</th>
<th>Mod</th>
<th>Heavy</th>
<th>p&lt;0.0001</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adjusted HDL-C</td>
<td>1.04</td>
<td>1.05</td>
<td>1.11</td>
<td>1.20</td>
<td>1.30</td>
<td>p&lt;0.0001</td>
</tr>
<tr>
<td>Adjusted insulin*</td>
<td>12.6</td>
<td>12.8</td>
<td>11.9</td>
<td>11.1</td>
<td>10.7</td>
<td>p&lt;0.0001</td>
</tr>
<tr>
<td>Top fifth %</td>
<td>9.3</td>
<td>9.7</td>
<td>20.0</td>
<td>25.4</td>
<td>37.0</td>
<td></td>
</tr>
<tr>
<td>Unadjusted HDL-C</td>
<td>(0.21)</td>
<td>(0.24)</td>
<td>(0.25)</td>
<td>(0.28)</td>
<td>(0.34)</td>
<td></td>
</tr>
<tr>
<td>Unadjusted insulin*</td>
<td>(7.7–22.1)</td>
<td>(7.9–21.3)</td>
<td>(7.4–20.0)</td>
<td>(6.9–18.5)</td>
<td>(6.7–18.0)</td>
<td></td>
</tr>
</tbody>
</table>

Top fifth % = percentage of men in alcohol intake category with concentrations in the top fifth of the distribution in 5221 men.
pre-existing CHD (undiagnosed). Alcohol intake was positively associated with HDL-cholesterol concentration and inversely associated with insulin concentrations. Figure 2 shows the odds of having raised levels of these factors in the 5221 men after adjustments as in table 2. Heavy drinking was associated with an eightfold increase in having high HDL-cholesterol and a 40% reduction in risk of having hyperinsulinaemia compared with occasional drinkers.

Alcohol intake, metabolic factors, and risk of type II diabetes
To assess the possible protective role of HDL-cholesterol and insulin on the relation between alcohol intake and type II diabetes we have examined the effects of additional adjustments for these factors (table 3). Adjustment slightly reduced the protective effects seen in moderate drinkers. Insulin and HDL-cholesterol singly or combined accounted for only about 20% of the reduction seen and although the reduced risk in moderate drinkers compared with occasional drinkers was no longer statistically significant (p=0.13), the quadratic trend remained significant (p=0.04) suggesting a non-linear trend between alcohol intake and risk of diabetes.

Alcohol intake, high risk subjects, and diabetes
We have also examined the relation between alcohol intake and risk of diabetes in men at low and high risk of diabetes (table 4). As the non-drinkers and occasional drinkers showed similar relative risks of diabetes they have been combined to achieve reasonable numbers. Because of the smaller number involved when stratifying we are primarily concerned with whether the associations differ between the levels of these risk factors rather than evaluating the statistical significance within each subgroup. The protective effect of moderate drinking was seen in both men with and without pre-existing CHD (undiagnosed). The protective effect was more evident in men with increased risk of diabetes—that is, heavier men, current smokers, men with low HDL-cholesterol, higher insulin levels, and higher glucose levels. On the other hand, the protective effect was more evident in physically active men. No protective effect was seen in men at low risk—that is, with

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**Table 3** Relative risk of type II diabetes (confidence intervals in parentheses) according to alcohol intake, in men free of doctor-diagnosed CHD, stroke, or diabetes adjusting for HDL-cholesterol and serum insulin

<table>
<thead>
<tr>
<th>Adjusted relative risk</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetics</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>1.10 (0.61 to 2.00)</td>
<td>1.12 (0.62 to 2.03)</td>
<td>1.11 (0.60 to 2.01)</td>
<td>1.12 (0.62 to 2.04)</td>
</tr>
<tr>
<td>Occ</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Light</td>
<td>0.81 (0.55 to 1.20)</td>
<td>0.84 (0.57 to 1.23)</td>
<td>0.86 (0.59 to 1.27)</td>
<td>0.87 (0.59 to 1.29)</td>
</tr>
<tr>
<td>Mod</td>
<td>0.66 (0.44 to 0.99)</td>
<td>0.71 (0.47 to 1.06)</td>
<td>0.72 (0.47 to 1.10)</td>
<td>0.74 (0.48 to 1.12)</td>
</tr>
<tr>
<td>Heavy</td>
<td>0.96 (0.60 to 1.52)</td>
<td>1.06 (0.66 to 1.68)</td>
<td>1.22 (0.76 to 1.97)</td>
<td>1.24 (0.77 to 1.99)</td>
</tr>
<tr>
<td>Linear trend</td>
<td>p=0.50</td>
<td>p=0.81</td>
<td>p=0.75</td>
<td>p=0.71</td>
</tr>
<tr>
<td>quadratic trend*</td>
<td>p=0.03</td>
<td>p=0.03</td>
<td>p=0.02</td>
<td>p=0.02</td>
</tr>
</tbody>
</table>

A=adjusted for age, smoking, physical activity, social class, pre-existing CHD, and BMI. B=adjusted for A+insulin only. C=adjusted for A+HDL-C only. D=adjusted for A+insulin and HDL-C. *Quadratic trend was obtained from a model that included a linear and quadratic term.

**Table 4** Incidence rate (per 1000 person years) and adjusted relative risk for diabetes according to risk factors for type II diabetes by alcohol intake. Number of cases/number of men in parentheses. Relative risks adjusted for age, smoking, physical activity, social class, pre-existing CHD (undiagnosed), and BMI

<table>
<thead>
<tr>
<th>Rate /1000 p-y</th>
<th>Non/Occ</th>
<th>Light</th>
<th>Mod</th>
<th>Heavy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-existing CHD (undiagnosed)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>(141/4113)</td>
<td>2.3</td>
<td>1.00</td>
<td>0.75</td>
</tr>
<tr>
<td>Yes</td>
<td>(57/1108)</td>
<td>3.6</td>
<td>1.00</td>
<td>0.89</td>
</tr>
<tr>
<td>BMI</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;25</td>
<td>(43/2427)</td>
<td>1.2</td>
<td>1.00</td>
<td>0.77</td>
</tr>
<tr>
<td>25–28</td>
<td>(69/1814)</td>
<td>2.5</td>
<td>1.00</td>
<td>0.67</td>
</tr>
<tr>
<td>&gt;28</td>
<td>(86/980)</td>
<td>6.1</td>
<td>1.00</td>
<td>0.81</td>
</tr>
<tr>
<td>Current smoking</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>(113/3015)</td>
<td>2.4</td>
<td>1.00</td>
<td>0.97</td>
</tr>
<tr>
<td>Yes</td>
<td>(83/2200)</td>
<td>2.7</td>
<td>1.00</td>
<td>0.60</td>
</tr>
<tr>
<td>Physical activity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not active</td>
<td>(14/632)</td>
<td>3.0</td>
<td>1.00</td>
<td>0.91</td>
</tr>
<tr>
<td>Active</td>
<td>(50/1917)</td>
<td>1.7</td>
<td>1.00</td>
<td>0.57</td>
</tr>
<tr>
<td>Glucose (tertiles mmol/l)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 &lt;5.1</td>
<td>(40/1848)</td>
<td>1.4</td>
<td>1.00</td>
<td>0.86</td>
</tr>
<tr>
<td>2 5.1–9.49</td>
<td>(43/1560)</td>
<td>1.8</td>
<td>1.00</td>
<td>0.60</td>
</tr>
<tr>
<td>3 9.5–14.48</td>
<td>(115/1813)</td>
<td>4.3</td>
<td>1.00</td>
<td>0.86</td>
</tr>
<tr>
<td>Insulin (tertiles U/l)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 &lt;8.6</td>
<td>(27/1748)</td>
<td>1.0</td>
<td>1.00</td>
<td>1.93</td>
</tr>
<tr>
<td>2 8.6–13.4</td>
<td>(59/1735)</td>
<td>2.3</td>
<td>1.00</td>
<td>0.42*</td>
</tr>
<tr>
<td>3 13.5–24.8</td>
<td>(112/1734)</td>
<td>4.4</td>
<td>1.00</td>
<td>0.92</td>
</tr>
<tr>
<td>HDL-C (tertiles mmol/l)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 &lt;1.01</td>
<td>(94/1725)</td>
<td>3.7</td>
<td>1.00</td>
<td>0.69</td>
</tr>
<tr>
<td>2 1.01–1.29</td>
<td>(58/1697)</td>
<td>2.3</td>
<td>1.00</td>
<td>1.08</td>
</tr>
<tr>
<td>3 1.30–1.52</td>
<td>(42/1714)</td>
<td>1.6</td>
<td>1.00</td>
<td>1.04</td>
</tr>
</tbody>
</table>

*p<0.05 with non/occasional as comparison group.
higher HDL-cholesterol, low insulin levels, or low glucose levels. However, tests for interaction between alcohol and these risk factors on risk of diabetes were not statistically significant.

DISCUSSION

A non-linear relation was observed between alcohol and incidence of type II diabetes even after adjustment for potential confounders. Risk decreased progressively up to levels of moderate drinking (3–6 drinks/day) and increased in heavy drinkers (daily>6 drinks/day). Serum HDL-cholesterol and serum insulin accounted for only a small proportion of the reduced risk seen in moderate drinkers. The protective effect of moderate drinking seemed to be more evident in men at increased risk of diabetes—that is, heavier men, current smokers, men with low HDL-cholesterol, higher insulin levels, and higher glucose levels.

Other studies

The findings in this study are consistent with several previous prospective studies. In the earlier US Nurses Health Study of 85,000 women aged 35–59 years followed up for four years, relative risk of diabetes decreased progressively from 1.0 in non-drinkers to 0.3 in their heaviest drinking category (≥15.0 g/day). Additional adjustment for BMI attenuated the protective effect and the possible protective effect of alcohol was considered to be attributable to residual confounding by obesity and its biological consequences. In a later extended follow-up study, light drinking (5–10 g/day) was associated with significantly lower risk than in non-drinkers and this was seen in both lean (<25 kg/m²) and obese (≥30 kg/m²) women. In the US Health Professionals’ Study comprising some 42,000 men aged 40–75 years followed up for six years, men who drank 30–49.9 g of alcohol showed a 40% reduction in risk of diabetes after full adjustment, compared with abstainers and risk increased in the higher intake group (≥50 g/day). In the Osaka Health Survey of over 6000 Japanese men aged 35–61 years followed up for 10 years, a non-linear relation was seen with the lowest risk in those drinking 29.1–50 g/day (“moderate drinking”). As in this study, their heavier drinkers (>50 g/day) showed the highest risk, but this was attenuated after adjustment that included BMI. Similarly in The Cooper Clinic Study (Texas, USA) of over 8600 men aged 30–79 years followed up for six years a non-linear relation was observed with the lowest risk in those drinking 61.9–122.7 g/week (about 1–2 US drinks/day). Heavy drinkers (>277 g/week) showed a more than twofold increase in risk compared with these men. In the US Physicians Health Study of 21,000 men aged 40–84 years followed up for 12 years, those drinking 2–4 US drinks/week, 5–6 drinks/week, and ≥1 drink/day showed significantly lower risk than those who rarely/never drank with the lowest risk (RR=0.57) in the ≥1 drink/day group. As this was their highest drinking category, it was not possible to assess the effects of heavy drinking in this study. Although the level of drinking associated with reduction in risk of developing type II diabetes varied between these prospective studies, in the majority, light and moderate drinkers have consistently shown lower risk of developing diabetes than heavy drinkers or non-drinkers.

Alcohol and serum insulin concentrations

An inverse relation was observed between alcohol intake and non-fasting insulin concentrations even after adjustment for potential confounders. The findings suggest that part of the “protective” effect of alcohol on both type II diabetes and CHD may be mediated via effects of alcohol on insulin sensitivity. The finding of lower insulin concentrations in drinkers compared with non-drinkers is consistent with data from previous smaller studies that have examined associations between fasting and post-load insulin and alcohol. Few studies have examined the relation between alcohol and type II diabetes adjusting for HDL-cholesterol and we are not aware of any prospective study that has examined the influence of insulin on the alcohol-diabetes or CHD relation. In the Cooper Clinic Study the non-linear relation between alcohol intake and diabetes persisted even after adjustment for HDL-cholesterol. In the present study, HDL-cholesterol and insulin seemed to account for only a small proportion of the reduced risk seen in moderate drinkers in multivariate analysis, although the difference between moderate drinking and occasional drinking (baseline) was no longer statistically significant. The lack of statistical significance may be attributable to the lack of power in categorical analysis when comparing group data. When alcohol was fitted continuously the quadratic trend remained significant after adjustment for insulin and HDL-cholesterol suggesting a non-linear relation between alcohol and diabetes.

Unmeasured and residual confounding

Studies of this nature will always carry uncertainty arising from unmeasured and residual confounding. For example, we have no measurements of dietary intake or any measures of central obesity, which are both areas of possible confounding. However, the Clinic Cooper study found the non-linear alcohol-diabetes association to be independent of waist circumference and BMI. The Nurses Health Study found the lower risk associated with light drinking to be independent of dietary factors including trans fatty acids, glycaemic load, fibre, and polyunsaturated fat. Although we did not separate lifelong teetotallers from ex-drinkers, we excluded a substantial number of people with ill health who may have modified their drinking habits. There is also the possibility of residual confounding arising from imprecise measurement of those variables that have been entered in the adjustment processes.

Bias

It can be argued that more precise measurements of insulin might have attenuated the risk further. The use of non-fasting insulin measurements adjusted for time of sampling has almost certainly increased the amount of random error or “noise” in the data compared with the use of insulin measured under more rigorous conditions. Despite this constraint, previous reports from the British Regional Heart Study have shown increased serum insulin to be associated with increased risk of both type II diabetes and CHD consistent with findings from other insulin-CHD/diabetes studies. The associations between non-fasting insulin and cardiovascular risk factors, such as BMI, blood lipids, and blood pressure reported in this cohort are consistent with those reported with fasting and post-load insulin in other studies. Correlation coefficients between insulin and biological risk factors in the BRHS have been shown to be virtually identical to those
Heavy drinking

Heavy drinking has been implicated as a risk factor for type II diabetes. In the majority of prospective studies, heavy drinkers have higher risk than light/moderate drinkers and in many studies heavy drinkers have the highest risk. In the Paris Prospective Study, persons with diabetes had a higher risk of death by cirrhosis, which was strongly associated with alcohol consumption. In the present study despite the favourable effects of alcohol on HDL-cholesterol and on serum insulin concentrations, heavy drinking was associated with increased risk of diabetes. It has been suggested that the mechanism may be attributable to a direct toxic effect of alcohol on the pancreatic islet cells or may reflect a truncal fat pattern associated with alcohol intake. In this study, part of the effect of heavy drinking on risk of diabetes seemed to be mediated through its effect on BMI.

Clinical implications

Despite the consistent association between light to moderate alcohol intake and reduced risk of major coronary heart disease events, virtually all authorities recommend that those who do not drink should not take up drinking and that occasional drinkers should not increase their intake. Moreover, as light to moderate alcohol intake does not have the same "protective" effect on non-cardiovascular or all cause mortality as it does on CHD, there is little justification for persuading those who do not drink regularly to do so. The same arguments apply to the observation that light to moderate alcohol intake is associated with a reduction in the risk of diabetes. Most recent studies have focused on the increased risk of diabetes in men with high alcohol intake and suggested that drinking less might reduce the risk in these subjects. We need further clinical/physiological studies to explore the complex interrelations between alcohol intake, body weight and obesity, insulin resistance, and lipid metabolism. Present knowledge regarding the modifiable factors involved in the development of diabetes indicate that overweight/obesity, physical activity, and cigarette smoking should be the key components of any public health programme for the prevention of type II diabetes.

Conclusion

A non-linear relation was seen between alcohol intake and the incidence of type II diabetes. With occasional drinkers as the baseline, risk was lowest in light to moderate drinkers and increased in the heavy drinkers. HDL-cholesterol and serum insulin seemed to explain only some of the reduction in risk of type II diabetes associated with moderate drinking. The adverse effect of heavy drinking on risk of diabetes seemed to be partially mediated through its effect on BMI. Our understanding of the mechanisms underlying the association between light to moderate drinking and the reduced risk of diabetes is limited. The relations between alcohol consumption, obesity, insulin concentrations, and blood lipids need to be explored.

REFERENCES


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