

Alcohol Consumption and Risk for Development of Impaired Fasting Glucose or Type 2 Diabetes in Middle-Aged Japanese Men

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OBJECTIVE — To investigate the association between alcohol consumption and risk for development of diabetes.

RESEARCH DESIGN AND METHODS — We examined 2,953 Japanese male office workers aged 35–59 years who did not have impaired fasting glucose (IFG) (a fasting plasma glucose concentration of 6.1–6.9 mmol/l), type 2 diabetes (a fasting plasma glucose concentration of ≥ 7.0 mmol/l or receipt of hypoglycemic medication), medication for hypertension, or a history of cardiovascular disease. Fasting plasma glucose concentrations were measured at periodic annual health examinations from May 1994 through May 2001.

RESULTS — There was a U-shaped association between alcohol consumption and the incidence of IFG or type 2 diabetes during 7 years of follow-up, with the lowest incidence at alcohol intake of 23.0–45.9 g ethanol/day. After controlling for age, family history of diabetes, BMI, cigarette smoking, and physical activity, the relative risk for development of IFG or type 2 diabetes compared with alcohol consumption of 23.0–45.9 g ethanol/day was 1.51 (95% CI, 1.07–2.13), 1.31 (95% CI, 0.93–1.84), 1.18 (95% CI, 0.87–1.61), and 1.43 (95% CI, 1.01–2.02) with alcohol consumption of 0, 0.1–22.9, 46.0–68.9, and ≥ 69.0 g ethanol/day, respectively (P for quadratic trend = 0.016). Analyses by presence or absence of a risk factor revealed that a U-shaped association was more evident in older men, men without a family history of diabetes, and nonsmokers.

CONCLUSIONS — These results indicate that moderate alcohol consumption among apparently healthy Japanese men is associated with reduced risk for development of IFG or type 2 diabetes.

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Type 2 diabetes, which affects ~7 million Japanese individuals, is characterized by impaired insulin secretion and insulin resistance. Aside from obesity, reduced physical activity, and cigarette smoking, there are few other well-established modifiable risk factors for type 2 diabetes (1–6). Alcohol con-

sumption represents a potentially important, modifiable risk factor of type 2 diabetes and may be related to the risk for type 2 diabetes through its effects on insulin secretion and sensitivity (7–10).

Several studies have been performed to study the effect of alcohol consumption on the incidence of type 2 diabetes in men

(1,5,11–17). In some of these studies, the association was U- or J-shaped, with the lowest incidence of type 2 diabetes in subjects with moderate alcohol consumption (5,11–13), whereas others observed overall positive (14,15), null (1,16), or inverse (17) associations. These inconclusive results may have resulted in part from ethnic or lifestyle differences in the study populations but also may have been strongly influenced by different methods used to investigate the association between alcohol intake and type 2 diabetes. In addition, participants with preexisting diabetes were enrolled in some studies and biased populations in others, and these confounders may have influenced the results. Therefore, it is necessary to conduct a longitudinal study in subjects free of diabetes at baseline to clarify the relation between alcohol intake and risk for diabetes.

Using data from serial annual health examinations at the workplace, we carried out a longitudinal population study to examine prospectively the association of alcohol intake with development of impaired fasting glucose (IFG) or type 2 diabetes (as diagnosed with the newly revised criteria of American Diabetes Association in 1997 [18] for epidemiological studies) in normoglycemic middle-aged Japanese men.

RESEARCH DESIGN AND METHODS

Study cohort

Our study is an ongoing cohort investigation, designed to clarify risk factors for major diseases, including hypertension, dyslipidemia, and diabetes among Japanese men who were office workers at one of the biggest building contractors in Japan. The Industrial Safety and Health Law in Japan requires the employer to conduct annual health examinations of all employees, and employees are required by law to participate. A signed self-administered

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Abbreviations: CVD, cardiovascular disease; IFG, impaired fasting glucose.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

questionnaire is a part of this examination, and the employee data, which are anonymous, are available for research with the approval of the employer. To evaluate the association of alcohol intake with development of IFG or type 2 diabetes, a survey of the incidence of IFG or type 2 diabetes was done between 1994 and 2001. All Japanese male office workers aged 35–59 years in May 1994 were invited to take a survey ($n = 3,694$); the participation rate was 99.6% ($n = 3,681$).

Of 3,681 potential participants, 663 (18.0%) were excluded: 175 (4.8%) had IFG, 282 (7.7%) had type 2 diabetes, 253 (6.9%) were taking antihypertensive medication, and 32 (0.9%) had a history of either coronary heart disease or stroke. Thus, the baseline population consisted of 3,018 men. We also excluded 65 men who did not participate in consecutive annual health examinations during follow-up. The final study population for analysis therefore consisted of 2,953 men. Men in whom IFG or type 2 diabetes was found during repeated surveys through May 2001 were classified as having IFG or type 2 diabetes. To determine the incidence of type 2 diabetes, incidental cases of IFG were followed and considered type 2 diabetes if they reached that end point. Altogether, 39 participants who started taking medication for diabetes during the observation period were considered to have type 2 diabetes. Owing to the age range of the study population, all cases of IFG or type 2 diabetes were diagnosed after 35 years of age.

Study design

Fasting plasma glucose concentrations were measured at annual health examinations in May from 1994 to 2001. The participants were asked to fast for at least 8 h and to avoid smoking and heavy physical activity for more than 2 h before the examinations. Blood samples were drawn from an antecubital vein. Glucose was measured using hexokinase-glucose dehydrogenase method (Shino-Test, Tokyo, Japan) with Olympus AU-5000 in 1994 and Olympus AU-5200 in 1995 to 2001 (Olympus Japan, Tokyo, Japan) at FALCO Biosystems Tokyo (Tokyo, Japan). Quality control of the laboratory was done internally, and the coefficients of variation between and within assays for plasma glucose were no more than 3% from 1994 to 2001. Normal fasting glucose, IFG, and type 2 diabetes were de-

termined using the criteria of the American Diabetes Association (18). Normal fasting glucose was defined as fasting plasma glucose concentration of <6.1 mmol/l. IFG was defined as fasting plasma glucose concentration of 6.1–6.9 mmol/l. Type 2 diabetes was defined as fasting plasma glucose concentration of ≥ 7.0 mmol/l or receipt of hypoglycemic medications, because an oral glucose tolerance test was not done for every subject.

Annual health examinations at study entry included medical history, physical examination, anthropometric measurements, biochemical measurements, and a questionnaire on health-related behaviors, such as alcohol consumption, smoking, and physical activity. Medical history and history of use of prescription drugs were assessed by the examining physicians. After a 5-min rest in a quiet room, systolic and diastolic blood pressures were measured in the right arm using a standard mercury sphygmomanometer. Serum total cholesterol was determined by the Olympus AU-5000 using the enzymatic method with a commercial reagents kit (Wako, Osaka, Japan), and HDL cholesterol was assayed using the same enzymatic method after precipitation by polyethylene glycol. Enzyme activities for γ -glutamyltransferase, aspartate aminotransferase, and alanine aminotransferase were measured by the Olympus AU-5000 using commercial reagents (International Reagents, Kobe, Japan) based on the principles recommended by the Japan Society of Clinical Chemistry (19).

For health-related behaviors, the questions about alcohol intake included items about frequency of alcohol consumption per week, type of alcoholic beverage, and usual amount consumed daily in units of “go” (a traditional Japanese unit of measurement, by volume, corresponding to 23 g ethanol). Weekly alcohol intake was calculated and then converted to daily alcohol consumption using standard Japanese tables. One go is 180 ml of sake, and it corresponds to one bottle (663 ml) of beer, two single shots (75 ml) of whiskey, or two glasses (180 ml) of wine. Subjects were classified as non-drinkers or current drinkers who averaged 0.1–22.9, 23.0–45.9, 46.0–68.9, or ≥ 69.0 g ethanol/day. The questionnaires were also used to ask about smoking habits (never, past, or current smoker); past or current smokers were asked about the number of cigarettes

smoked per day and the duration of smoking in years. Participants were asked about the type and weekly frequency of leisure-time physical activity. Physical exercise was defined as participation in any physical activity, such as jogging, bicycling, swimming, or tennis, that was performed long enough to sweat.

Statistical analyses

The χ^2 test and one-way ANOVA were used to analyze the statistical differences among characteristics of the study participants at enrollment according to alcohol intake. For each participant, person-years of follow-up were calculated from the date of enrollment to the date of the development of IFG or type 2 diabetes or the date of last follow-up, whichever came first. The follow-up rate was 92.6% of the total potential person-years of follow-up. Cox's proportional hazards models were used to evaluate the association between alcohol intake and development of IFG or type 2 diabetes. Data were adjusted first for age alone and then for the following multiple covariables: age, family history of diabetes, BMI, cigarette smoking, and physical activity. Potential confounding factors were treated as categorical variables: age and BMI (graded from 1 through 5 [first through fifth quintiles]); family history of diabetes (no or yes); cigarette smoking (graded as 1 [none] or as quartile 1 [grade of 2] to quartile 4 [grade of 5] for current smokers); and regular physical exercise (graded from 1 to 3 [hardly ever, once a week, or twice or more a week]). To assess nonlinearity between alcohol intake and the risk for IFG or type 2 diabetes, the estimated quantitative median value for each category of alcohol intake was included as a linear and quadratic term in the model.

Data were analyzed using the SPSS/PC statistical package (SPSS, Chicago, IL). All reported P values are two-tailed, and those <0.05 were considered to be statistically significant.

RESULTS — The baseline characteristics of the study sample according to alcohol intake are shown in Table 1. Tests for differences in baseline characteristics across drinking groups were significant except for family history of diabetes and fasting plasma glucose. Current cigarette smoking, regular physical activity, systolic and diastolic blood pressures, HDL cholesterol, and γ -glutamyltransferase

Table 1—Baseline characteristics of 2,953 Japanese male office workers, according to alcohol intake

Characteristics	Alcohol intake (g ethanol/day)				
	0	0.1–22.9	23.0–45.9	46.0–68.9	≥69.0
n	421	534	698	881	419
Age (years)	47.4 ± 6.3	45.7 ± 6.2	46.0 ± 6.3	46.6 ± 5.9	47.1 ± 5.3
Family history of diabetes (%)	6.7	9.2	7.7	7.4	9.8
BMI (kg/m ²)	23.3 ± 2.8	23.2 ± 2.7	23.1 ± 2.4	23.3 ± 2.6	23.7 ± 2.6
Current smoker (%)	45.4	42.5	46.4	54.6	63.5
Regular physical activity at least once a week (%)	42.0	52.4	51.6	56.8	54.7
Systolic blood pressure (mmHg)	124.9 ± 14.9	124.4 ± 14.5	124.5 ± 13.5	128.5 ± 13.8	130.6 ± 14.3
Diastolic blood pressure (mmHg)	75.0 ± 11.3	76.2 ± 10.5	76.2 ± 10.4	78.5 ± 10.5	80.1 ± 10.5
Fasting plasma glucose (mmol/l)	4.99 ± 0.37	4.98 ± 0.35	4.97 ± 0.34	5.00 ± 0.35	4.99 ± 0.36
Total cholesterol (mmol/l)	5.03 ± 0.82	5.01 ± 0.82	4.92 ± 0.79	4.99 ± 0.83	5.05 ± 0.81
HDL cholesterol (mmol/l)	1.29 ± 0.30	1.35 ± 0.30	1.40 ± 0.29	1.45 ± 0.35	1.48 ± 0.36
γ-Glutamyltransferase (units/l)	25.6 ± 30.1	27.4 ± 27.1	30.5 ± 28.9	41.4 ± 33.4	61.3 ± 56.3
Aspartate aminotransferase (units/l)	22.1 ± 9.6	21.2 ± 8.3	21.6 ± 7.4	22.5 ± 7.5	27.2 ± 15.5
Alanine aminotransferase (units/l)	23.8 ± 16.9	24.0 ± 16.1	23.6 ± 14.1	23.3 ± 12.9	27.1 ± 15.8

Data are means ± SD unless indicated otherwise.

showed a linear trend related to alcohol intake.

Altogether 281 and 168 men developed IFG and type 2 diabetes during the 18,214 and 18,673 person-years of follow-up, respectively. There was a U-shaped association of alcohol intake with the incidence of both IFG and type 2 diabetes, with the lowest incidence at alcohol intake of 23.0–45.9 g ethanol/day (Fig. 1). IFG and type 2 diabetes were combined in this study, because we assumed a similar mechanism for the development of IFG and type 2 diabetes.

During 7 years of follow-up representing 17,871 person-years, 370 men developed IFG or type 2 diabetes (Table 2). The age-adjusted relative risk for development of IFG or type 2 diabetes compared with alcohol consumption of 23.0–45.9 g ethanol/day was 1.58 (95% CI, 1.12–2.23), 1.34 (95% CI, 0.95–1.88), 1.27 (95% CI, 0.93–1.72), and 1.71 (95% CI, 1.22–2.41) with alcohol consumption of 0, 0.1–22.9, 46.0–68.9, and ≥69.0 g ethanol/day, respectively (*P* for quadratic trend = 0.001). The multivariate adjustment for age, family history of diabetes, BMI, cigarette smoking, and regular physical activity reduced the risk in those who drank ≥69.0 g ethanol/day substantially, but the quadratic trend remained significant (*P* = 0.016).

Results of stratified analyses by presence or absence of a risk factor are shown in Table 3. A U-shaped association of alcohol consumption with risk for development of IFG or type 2 diabetes was

observed, after adjustment for various potential confounders for diabetes. The protective effect of moderate alcohol consumption was more evident in older men, men without a family history of diabetes, and nonsmokers. Tests for interaction between alcohol consumption and these risk factors on risk for development of IFG or type 2 diabetes were not statistically significant.

CONCLUSIONS — We found a non-linear relation between alcohol consumption and risk for development of IFG or type 2 diabetes even after adjustment for

potential confounders for diabetes. The risk for development of IFG or type 2 diabetes decreased progressively up to levels of moderate drinking (23.0–45.9 g ethanol/day) and increased in heavy drinkers (≥69.0 g ethanol/day). From stratified analyses by presence or absence of a risk factor, the protective effect of moderate consumption seemed to be more evident in older men, men without a family history of diabetes, and nonsmokers. Younger men, men with a family history of diabetes, and current smokers who drank ≥46.0 g ethanol/day had the lower relative risk for development of IFG or

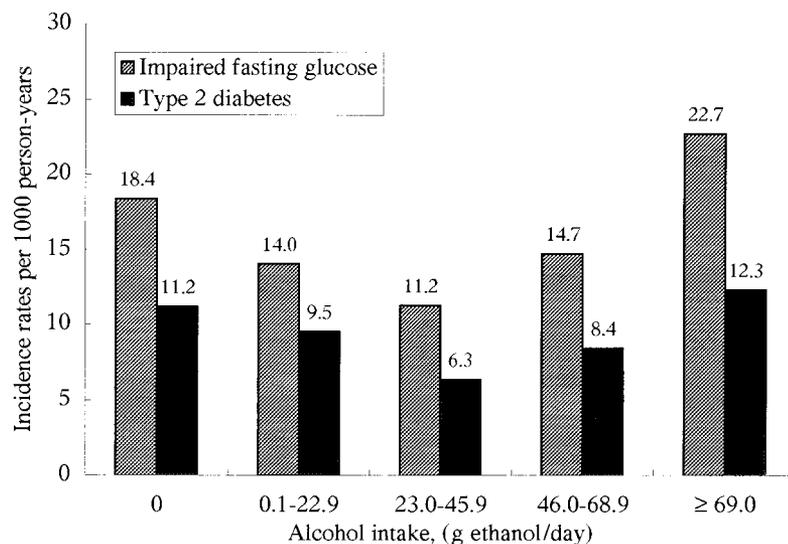


Figure 1—Incidence rates per 1,000 person-years for IFG and type 2 diabetes according to alcohol intake among 2,953 Japanese male office workers during 7 years of follow-up.

Table 2—The risk for incidence of impaired fasting glucose or type 2 diabetes by alcohol intake

	Alcohol intake (g ethanol/day)					P value*
	0	0.1–22.9	23.0–45.9	46.0–68.9	≥69.0	
Participants (n)	421	534	698	881	419	
Cases (n)	63	67	66	107	67	
Total person-years	2491	3251	4282	5386	2461	
Rate per 1,000 person-years	25.3	20.6	15.4	19.9	27.2	
Age-adjusted relative risk (95% CI)	1.58 (1.12–2.23)	1.34 (0.95–1.88)	1.00 (Referent)	1.27 (0.93–1.72)	1.71 (1.22–2.41)	0.001
Multivariate-adjusted relative risk (95% CI)†	1.51 (1.07–2.13)	1.31 (0.93–1.84)	1.00 (Referent)	1.18 (0.87–1.61)	1.43 (1.01–2.02)	0.016

*For quadratic trend. †Adjusted for age, family history of diabetes, BMI, cigarette smoking, and regular physical activity at study entry.

type 2 diabetes than older men, men without a family history of diabetes, and nonsmokers, respectively.

However, the lower relative risk for development of IFG or type 2 diabetes associated with moderate alcohol intake in men with a family history of diabetes and in current smokers does not really mean that genetic predisposition and cigarette smoking improve the risk for development of IFG or type 2 diabetes in moderate drinkers. As shown in Table 3, men with a family history of diabetes and current smokers had higher incidence rates of IFG or type 2 diabetes than those without a family history of diabetes and nonsmokers, respectively. Furthermore,

in moderate drinkers (23.0–45.9 g ethanol/day), these differences were more pronounced (incidence rate of IFG or type 2 diabetes: 13.9 and 32.9 per 1,000 person-years for those without and with family history of diabetes, respectively, and 9.0 and 23.2 per 1,000 person-years for nonsmokers and current smokers, respectively). Thus, the influence of alcohol on development of IFG or type 2 diabetes might be obscured by genetic predisposition and cigarette smoking.

Our results are consistent with those of other studies (5,11–13) and indicate that moderate alcohol consumption is associated with a reduced risk for IFG or type 2 diabetes. Although the mechanism

of how moderate drinking decreases the risk for diabetes remains to be elucidated, the findings that light-to-moderate alcohol intake is associated with a lower risk of diabetes and high alcohol intake is associated with a high risk are biologically plausible. Several studies have suggested that moderate alcohol consumption may increase insulin sensitivity and lower insulin resistance (7–10). Increased insulin secretory responses and enhanced glucose disposal rates have been observed after moderate alcohol ingestion in subjects without diabetes as well as in subjects with type 2 diabetes (20,21). This increased secretory response may be a result of the alcohol-induced stimulation of in-

Table 3—The risk for incidence of impaired fasting glucose or type 2 diabetes by alcohol intake within subgroups

Risk factors	Rate per 1,000 person-years	Alcohol intake (g ethanol/day)					P value*
		0	0.1–22.9	23.0–45.9	46.0–68.9	≥69.0	
Age (median, 46.4 years)							
Below median	17.4	1.54 (0.93–2.54)	1.11 (0.69–1.79)	1.00 (Referent)	0.99 (0.64–1.55)	1.12 (0.67–1.87)	0.153
At or above median	24.4	1.56 (0.96–2.53)	1.52 (0.94–2.47)	1.00 (Referent)	1.39 (0.90–2.14)	1.78 (1.11–2.88)	0.038
Family history of diabetes							
No	19.8	1.53 (1.06–2.21)	1.32 (0.91–1.90)	1.00 (Referent)	1.23 (0.89–1.71)	1.50 (1.04–2.17)	0.015
Yes	30.9	1.64 (0.58–4.65)	1.29 (0.53–3.14)	1.00 (Referent)	0.85 (0.34–2.12)	1.05 (0.39–2.82)	0.457
BMI (median, 23.2 kg/m ²)							
Below median	16.7	1.90 (1.12–3.24)	1.51 (0.89–2.56)	1.00 (Referent)	1.27 (0.78–2.03)	1.44 (0.82–2.53)	0.056
At or above median	24.9	1.35 (0.85–2.13)	1.21 (0.78–1.89)	1.00 (Referent)	1.14 (0.76–1.70)	1.48 (0.95–2.29)	0.069
Current smoking							
No	16.7	2.20 (1.26–3.86)	1.86 (1.07–3.22)	1.00 (Referent)	1.74 (1.03–2.94)	2.64 (1.48–4.74)	0.002
Yes	24.8	1.20 (0.77–1.89)	1.05 (0.68–1.64)	1.00 (Referent)	0.95 (0.65–1.40)	1.06 (0.70–1.62)	0.405
Physical exercise (times/week)							
Hardly any	21.1	1.53 (0.96–2.45)	1.31 (0.81–2.12)	1.00 (Referent)	1.09 (0.69–1.72)	1.44 (0.88–2.37)	0.055
Once or more	20.3	1.52 (0.90–2.55)	1.31 (0.81–2.13)	1.00 (Referent)	1.29 (0.84–1.96)	1.39 (0.86–2.26)	0.153

Data are relative risk (95% CI) unless indicated otherwise. Relative risks were adjusted for age, family history of diabetes, BMI, cigarette smoking, and regular physical activity at study entry. *For quadratic trend.

sulin secretion by gastrointestinal secretagogues or an insulinogenic effect of corticotropin peptides present in cells of the gastroentero-pancreatic system (22–25). On the other hand, diabetogenic effects of high alcohol intake include its contribution to excess caloric intake and obesity, induction of pancreatitis, disturbance of carbohydrate and glucose metabolism, and impairment of liver function (7,26). Large amounts of alcohol decrease insulin-mediated glucose uptake, and alcoholics have decreased glucose tolerance (25,27,28). In vitro studies indicate that exposure of β -cells to alcohol is associated with decreased insulin secretion (29,30) and that metabolites of alcohol such as ethanol, 2,3-butanediol, and 1,2-propanediol inhibit basal and insulin-stimulated adipocyte metabolism (31). In an experimental study, atrophy of β -cells and reduction of absolute pancreatic islet volume have been observed in ethanol-treated rats (32). In addition, gross ultrastructural alterations are seen in most β -cells of ethanol-treated rats: irregularity of the nuclear envelope with deep invagination, margination of heterochromatin, and many empty granules or granules without clear electron-dense crystals of insulin. These conditions may be attributable to a direct toxic effect of alcohol on pancreatic islet cells or to inhibition of insulin secretion and increase in insulin resistance, and large amounts of alcohol may be relevant to the pathogenesis of a high incidence of type 2 diabetes.

As for the association between alcohol consumption and mortality, previous studies reveal a J- or U-shaped relationship between alcohol consumption and total mortality in men (33–36). The reduction in total mortality at light-to-moderate levels is due to a reduction in cardiovascular disease (CVD), without significant increases in other causes of mortality. Biological mechanisms that might explain the association between light-to-moderate alcohol consumption and mortality might be increased insulin sensitivity and decreased insulin resistance (7–10) as well as the increase in HDL cholesterol, decreased platelet aggregation, and enhanced fibrinolysis (37–39).

Our study had several limitations. First, we estimated alcohol intake from a questionnaire of self-reported drinking habits, and misreporting of alcohol consumption could be sources of bias. In this

study, there was a clear dose-response relation between alcohol intake and biological markers of alcohol consumption such as HDL cholesterol and γ -glutamyl transpeptidase, suggesting that little misclassification of alcohol intake had occurred.

Second, bias in case-finding could have occurred. Specifically, heavy drinkers are more likely to visit a doctor for reasons other than diabetes; thus diabetes could have been found by chance. However, because all incidental cases were found by periodic annual screening in our study, such bias is unlikely to have occurred. Furthermore, participants in our normoglycemic cohort, particularly those in the older age-groups, might not be typical of the general population. Those whose plasma glucose concentration was already above borderline values or who reported taking drugs for hypertension or having a history of CVD during the initial examination were excluded. Because hypertension is a recognized risk factor for diabetes (40,41), exclusion of hypertensive persons would bias the study toward a particularly healthy study population at a low risk for diabetes. The selection of men with rigorously normal fasting plasma glucose concentration at study entry could have had an effect on the observations.

Third, an assessment of IFG or type 2 diabetes entirely dependent on fasting plasma glucose concentration may underestimate overt longer-term diabetes. HbA_{1c} was measured using high-performance liquid chromatography (Sekisui Chemical, Osaka, Japan) with Olympus AU-5000 in 1994 and Olympus AU-5200 in 1995 to 2001. From the distribution of fasting plasma glucose and HbA_{1c}, the cut-off points of 6.1 and 7.0 mmol/l for fasting plasma glucose were compatible with those of 5.9% and 6.5% for HbA_{1c}, respectively. Among 3,140 men who had HbA_{1c} <5.9%, were not taking medication for diabetes or hypertension, and had no history of CVD at study entry, a U-shaped association was found between alcohol consumption and development of the elevated HbA_{1c} of both 5.9–6.4% and \geq 6.5% over a 7-year period, with the lowest incidence at alcohol intake of 23.0–45.9 g ethanol/day. The multivariate adjusted relative risk for development of the elevated HbA_{1c} level of \geq 5.9% compared with alcohol consumption of 23.0–45.9 g ethanol/day

was 1.70 (95% CI, 1.21–2.38), 1.55 (95% CI, 1.12–2.15), 1.32 (95% CI, 0.98–1.79), and 1.67 (95% CI, 1.20–2.32) with alcohol consumption of 0, 0.1–22.9, 46.0–68.9, and \geq 69.0 g ethanol/day, respectively (*P* for quadratic trend <0.001). These results are consistent with those of the analyses using fasting plasma glucose, and the rise in fasting plasma glucose observed in this normoglycemic cohort may express the development of diabetes during the follow-up period.

Finally, we could not include several confounding variables in this study, such as visceral adiposity (waist-to-hip ratio) and fasting insulin level. The central pattern of fat distribution, with its increased waist-to-hip ratio, is associated with more insulin resistance than is the peripheral pattern of distribution (42,43). Individuals with the central pattern are more likely to have glucose intolerance and hyperinsulinemia resulting from insulin resistance (44,45). Therefore, visceral adiposity and fasting insulin level should be included in future studies.

Despite these potential limitations, our findings, which were obtained from a cohort of middle-aged Japanese men, support the conclusion that moderate alcohol consumption is associated with a lower risk for development of IFG or type 2 diabetes. As moderate alcohol consumption might lower the risks for CVD and diabetes, it may well be that the effects of moderate alcohol consumption on CVD are related to the changes in insulin and glucose metabolism.

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