Alcohol Intake Among Women and Its Relationship to Diabetes Incidence and All-Cause Mortality

The 32-year follow-up of a population study of women in Gothenburg, Sweden

Leif Lapidus, md[1,2]
Calle Bengtsson[2]
Elisabet Bergfors, md[2]
Cecilia Björkelund[2]
Fredrik Spak[3]
Lauren Lissner[2,4]

OBJECTIVE — The purpose of the study was to explore the predictive value of women's alcohol habits in relation to incidence of diabetes and all-cause mortality. Special attention was paid to potential confounding factors such as age, heredity, education, socioeconomic group, physical inactivity, smoking, blood pressure, serum lipids, and, in particular, obesity.

RESEARCH DESIGN AND METHODS — A longitudinal population study consisting of a representative sample of 1,462 women aged 38–60 started in Göteborg, Sweden, in 1968–1969 monitoring for diabetes and mortality over 32 years.

RESULTS — Alcohol intake, expressed as intake of wine, hard liquor, or total grams of alcohol, was significantly negatively associated to 32-year diabetes incidence independent of age. However, the apparently protective effect of the alcohol variables was attenuated when BMI was included as a covariate. The inverse relationship between wine intake and diabetes did not remain after adjustment for physical activity or socioeconomic group. Beer and wine intake were significantly negatively associated to mortality. Increase of alcohol intake between the examination in 1968–1969 and 1980–1981 was significantly inversely related to the mortality between 1980–1981 and 2000–2001 and independent of all covariates. No relationship was observed between an increase in alcohol intake and diabetes incidence. However, after adjustment for age, family history, and basal alcohol consumption altogether, a significant inverse relationship was observed between increase of alcohol and diabetes incidence.

CONCLUSIONS — The initially significant inverse associations observed between alcohol and diabetes as well as mortality were dependent on a number of confounding factors, of which BMI seems to be the most important.

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Several studies have shown the benefits of moderate alcohol consumption on cardiovascular disease among both men and women as reviewed by Howard et al. (1). Alcohol consumption has been reported to correlate with improved insulin sensitivity (2). In prospective studies of men, high alcohol consumption was shown to be associated with increased risk of diabetes in one population (3), but with reduced risk in another cohort (4). In female populations, an apparent protective effect of alcohol could be explained by less drinking among obese women, although there is no evidence. In a twin cohort study, high alcohol consumption was associated with an increased incidence of diabetes in lean but not in overweight women (5). In a large Japanese cohort of men (6) the protective effect was only present in men with BMI $\geq$22.1 kg/m^2. A nonlinear association has also been observed whereby the lowest risk was observed among those who consume light to moderate amounts of alcohol (7). The confounding effect of central obesity has rarely been addressed (1).

We have previously reported on longitudinal changes and secular trends in women's alcohol habits in a prospective population study in Göteborg (8), showing a gradually distinct increase of wine and hard liquor intake among the examinations in 1968–1969, 1980–1981, and 1992–1993. The same population has also been extensively studied with regard to health risks of different types of obesity and for this purpose have been thoroughly examined by a series of anthropometric parameters. In this article, we relate alcohol intake to incidence of diabetes and mortality from all causes during 32 years of follow-up, with specific attention to confounding by BMI and central obesity expressed as waist-to-hip ratio (WHR).

From the [1]Department of Medicine, Sahlgrenska University Hospital, Göteborg, Sweden; the [2]Department of Primary Health Care, Göteborg University, Göteborg, Sweden; and the [3]Department of Social Medicine, Göteborg University, Göteborg, Sweden; and the [4]Nordic School of Public Health, Göteborg, Sweden.

Address correspondence and reprint requests to Dr. Leif Lapidus, Department of Primary Health Care, Sahlgrenska Academy, Göteborg University, Box 454 S-405 30 Göteborg, Sweden. E-mail: leif.lapidus@swipnet.se.

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Abbreviations: WHR, waist-to-hip ratio.

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

Table 1—*Characteristics in the population study of women in Gothenburg in 1968–1969, incidence of diabetes and all-cause mortality during 32 years*

Age (years)	38	46	50	54	60	All women
Year of birth	1930	1922	1918	1914	1908	—
n	372	431	398	180	81	1,462
Participation rate 1968–1969 (%)	91.4	90.0	91.3	88.7	83.5	90.1
Smokers (%)	46.6	43.9	36.9	38.3	19.8	40.7
Beer (%)*	53.0	48.7	46.6	49.4	39.5	48.8
Wine (%)*	16.4	21.1	21.2	17.8	14.8	19.2
Hard liquor (%)*	3.2	6.5	6.3	9.4	6.2	6.0
Alcohol (g/month)	134	139	144	155	108	139
BMI	23.4	23.5	24.8	24.9	25.1	24.1
Diabetes incidence (%)	7.8	10.7	8.2	7.9	6.5	8.7
Mortality (%)	15.3	29.4	39.3	61.2	82.8	34.6

*At least once per week.

RESEARCH DESIGN AND METHODS

— In 1968–1969 a prospective study including 1,462 women aged 38, 46, 50, 54, and 60 was initiated in Gothenburg, Sweden (9). A systematic sampling method, based on date of birth, was chosen to get a representative cross section of the women in the community of the ages studied. The participation rate was high (90.1%). Table 1 shows the number of participants in each age group at the initial examination. The participants were offered reexamination on four occasions, in 1974–1975, 1980–1981, 1992–1993, and 2000–2001 (10–13). Nonparticipants in the most recent examination, initiated in 2000, were offered home visits. A total participation rate of 71% of those who participated in 1968–1969 and who were still alive in 2000–2001 was obtained.

Clinical examination

Alcohol intake. Information about alcohol intake was obtained during a standardized interview with an examining physician. Each subject was asked to answer three separate questions describing her usual intake of beer, wine, and strong liquor at the baseline examination in 1968–1969 as well as at the examination in 1980–1981. Possible answers included "never," "formerly," "monthly," "weekly," "several times in a week," or "daily." Alcohol exposure was evaluated as a risk factor in four ways: the intakes of beer, wine, and strong spirits were studied as three separate categorical variables. In addition, the total alcohol intake per month, expressed in grams, was calculated by converting the alcohol intake into approximate grams. Although the question about intake of beer did not distinguish different alcohol contents, according to observations from earlier studies we assumed that the average serving of beer contained 33 cl with an average alcohol content of 3%, which is equal to 7.8 g of alcohol. Similarly, an ordinary wine serving was assumed to be 1.5 glasses containing 14 cl with an average alcohol content of 13%, and a serving of strong liquor was assumed to be 1.5 glasses containing 4 cl of 40% alcohol content. We further assumed that "monthly" on average means twice a month, "weekly" twice a week, and "several times in a week" four times every week. The estimated alcohol intakes (grams) for different categories in the questionnaire are shown in the APPENDIX.

The change in alcohol habits between 1968–1969 and 1980–1981 was defined as the difference of alcohol intake, expressed as grams per month according to the definition above, between the intake reported at the examination in 1980–1981 and in 1968–1969.

Covariates

Heredity. Family history for diabetes was defined as having at least one parent or sibling with diabetes.

Physical activity. Subjects were interviewed about physical activity during their leisure time. According to the degree of physical activity, the women were classified into four groups (I–IV) using a classification that has been validated in a similar population study of men (14) and found to be a strong predictor of mortality in the cohort (15). Activity in group I includes reading or watching television, in group II activity includes walking or cycling for 30 min per day, in group III regular activity includes running, tennis, or swimming, and for group IV activity includes heavy activity or competitive sports.

Social data. Information about socioeconomic status was obtained by a questionnaire. The women reported their own occupations and, if married, their husbands' occupations, according to Carlsson's standard occupations grouping system (16). The analyses reported are based on three groups, using the woman's occupational group if available, and the husband's occupation otherwise.

Blood analyses. Blood samples were taken after overnight fasting from all participants. Serum cholesterol concentration was determined according to the method described by Levine and Zak (17), and serum triglyceride concentration was determined according to the method of Lofland (18).

Anthropometry. Body weight was measured to the nearest 0.1 kg with a balance scale. Subjects wore only briefs when being weighed. Body height without shoes was measured to the nearest 0.5 cm. BMI was calculated as weight in kilograms divided by the square of height in meters. Waist and hip circumferences were measured with a steel tape, and WHR was calculated.

Blood pressure. Blood pressure was measured with a mercury manometer after ~5 min of rest with the subject in the seated position.

Smoking. Smoking habits at the initial study in 1968–1969 are presented in Table 1. Subjects who currently smoked one or more cigarettes per day were defined as smokers.

End points during follow up

Diabetes. A subject was defined as having diabetes if she was receiving antidiabetic therapy (insulin or tablets) or if two fasting blood samples showed glucose concentrations meeting the current World Health Organization definition of diabetes, i.e., plasma glucose concentration ≥7.0 mmol/l (2000–2001) or if the diagnosis had been made by her physician. Diabetes was also accepted as a diagnosis if it was written on the death certificate. The date of

diagnosis was confined to the year the diabetes was diagnosed.

Mortality. Confirmation of whether subjects were alive or dead at the end of the 32 years of follow-up was obtained from the National Person Registry for all of the initial 1,462 participants. Death certificates were obtained for 99.3% of the 555 women who died during the 32-year follow-up period, and the exact date of death from these certificates was used in the statistical analyses.

Statistical methods

Age-adjusted proportional hazards regression was performed in which end points during years of follow-up were related to exposure to alcohol at the baseline examination in 1968–1969 or 12-year change. For diabetes incidence, censored patients were defined as women in whom diabetes was not diagnosed. Censored patients were followed until the last examination in which they participated except when the diagnosis of diabetes was written on the death certificate.

Multivariate adjustment was performed to adjust for confounding by specified risk factors measured at baseline. Stepwise analyses were performed according to the model of hazard regression with backward elimination. Risk ratios and 95% confidence intervals for alcohol intake in 1968–1969 or alcohol intake difference 1980–1981 minus 1968–1969 were both based on 100-g intervals. All models for 12-year change of exposure of alcohol were adjusted for baseline consumption levels. Women who were diagnosed with diabetes between the two examinations, 1968–1969 and 1980–1981, were excluded from the 12-year change analyses.

RESULTS

Alcohol intake in the initial study

Table 1 shows the intake of each beverage type and average intake expressed in grams per month in the different age strata at the baseline examination in 1968–1969. Additionally, it was observed that 24% of women never consumed any kind of alcohol whereas 11% consumed at least one of the alcoholic beverage types daily. Average amounts per month were 108 g for 60-year-old women and 134 g for those who were aged 38 in 1968–1969. Intraindividual correlations between total intake of alcohol in 1968–1969 versus 1980–1981 were significant ($P < 0.001$), indicating that this variable was a relatively stable characteristic at the individual level. The average age-standardized consumption in obese women (BMI >30 kg/m^2) was 75 g/week compared with 145 g/week in nonobese women ($P = 0.0002$).

Initial prevalence and 32-year incidence of diabetes

Eleven women (0.8% of the total number studied) had manifest diabetes at the first examination in 1968–1969 and were thus excluded from the statistical analyses of 32-year incidence. Four women were receiving diet therapy only, three used antidiabetic tablets, three were receiving insulin treatment, and one had previously unknown diabetes. Altogether 126 subjects developed diabetes that was not present at the baseline examination during the 32-year follow-up period. One woman with a high glucose value at the 2000–2001 examination died shortly thereafter and was excluded from the statistical analyses because of lack of confirmatory diagnoses of blood glucose. Another woman with an elevated fasting plasma glucose value (8.3 mmol/l) was also excluded from the analysis because the glucose value was not confirmed. Of all the patients with diabetes in this study only one had type 1 diabetes, but she was excluded from the present analyses (along with all prevalent cases at baseline) because she had already been diagnosed in 1968.

Alcohol and end points

Alcohol habits in relation to diabetes incidence. Table 2 shows the relationship between alcohol intake and diabetes incidence. Intakes of wine and hard liquor and total alcohol intake per month expressed in grams were all significantly inversely related to the diabetes incidence over 32 years, independent of age. The protective effect of initial alcohol intake (wine, hard liquor, or total grams per month) did, however, not remain when BMI was included in the multivariate analyses (Table 2). Furthermore, the inverse relationship between intake of wine and diabetes did not remain after inclusion of physical activity or socioeconomic group, respectively. No relationship was observed between beer intake and diabetes incidence in any of the models studied (Table 2).

When similar analyses were performed separately in women with BMI >25 kg/m^2 ($n = 477$) and in women with BMI ≤25 kg/m^2 ($n = 969$), there was a significant inverse relationship between total alcohol intake expressed in grams and diabetes incidence ($P < 0.05$) in the group of women with BMI >25 kg/m^2. This relationship did not remain when controlling for heredity, BMI, systolic blood pressure, physical activity, or social group. No significant associations were observed between intake of beer, wine, or hard liquor and diabetes incidence among women with BMI >25 kg/m^2. In the group of lean women no significant relationships between alcohol intake and diabetes incidence were observed.

Stepwise analyses with backward elimination including alcohol intake and all covariates studied (Table 2) showed that only four of the studied variables were statistically and independently associated to diabetes incidence: heredity, BMI, WHR, and physical inactivity; all were positively related to diabetes.

Alcohol habits and 32-year mortality. Table 3 shows the association between alcohol intake and overall mortality. Women who at the initial examination reported a high intake of beer or wine had a significantly lower mortality during the 32-year follow-up when controlling for age. These observations did not remain when family history, BMI, WHR, systolic blood pressure, serum triglyceride levels, education, physical activity, or socioeconomic group was added separately in the multivariate analysis. Furthermore, the inverse relationship between intake of wine and early death did not remain when serum cholesterol level was added to the multivariate analysis. Amount of alcohol intake expressed in grams of alcohol and intake of hard liquor did not relate to overall mortality (Table 3).

When the analyses were performed separately in women with BMI >25 kg/m^2 and women with BMI ≤25 kg/m^2, there was a significant inverse relationship between intake of beer and wine and mortality ($P < 0.05$) in the group of women with BMI >25 kg/m^2. The covariates had an effect on this association similar to that observed for the total population sample. In the group of lean women no significant relationships were observed.

Table 2—*Relative risks (RRs) and 95% CIs from proportional hazards model describing the relation between alcohol intake and type 2 diabetes incidence over 32 years*

	Variables							
	Beer		Wine		Hard liquor		Alcohol (100 g/month)	
Covariates	RR	95% CI	RR	95% CI	RR	95% CI	RR	95% CI
Age	0.94	0.86–1.02	0.88*	0.80–0.98	0.84*	0.72–0.96	0.83†	0.72–0.95
Age + heredity	0.93	0.85–1.02	0.88*	0.78–1.00	0.82*	0.68–0.98	0.78†	0.65–0.94
Age + BMI	1.00	0.92–1.08	0.92	0.84–1.02	0.89	0.78–1.03	0.90	0.79–1.02
Age + WHR	0.93	0.86–1.01	0.88*	0.79–0.98	0.82†	0.70–0.95	0.80†	0.69–0.92
Age + smoking	0.94	0.86–1.02	0.88*	0.80–0.97	0.82†	0.71–0.95	0.82†	0.72–0.94
Age + systolic blood pressure	0.95	0.88–1.03	0.90*	0.82–1.00	0.86*	0.75–1.00	0.85*	0.74–0.97
Age + triglycerides	0.95	0.88–1.03	0.90*	0.81–0.99	0.85*	0.73–0.98	0.84*	0.74–0.97
Age + cholesterol	0.94	0.87–1.02	0.89*	0.80–0.98	0.84*	0.73–0.97	0.83†	0.73–0.95
Age + education	0.95	0.87–1.03	0.90*	0.81–0.99	0.85*	0.73–0.98	0.84*	0.73–0.97
Age + physical activity	0.95	0.88–1.03	0.91	0.82–1.00	0.83*	0.72–0.96	0.85*	0.74–0.97
Age + social group	0.96	0.88–1.04	0.92	0.83–1.03	0.86*	0.74–0.99	0.86*	0.75–0.99

Stepwise model: independent risk factors for diabetes incidence

	P value	Maximum likelihood estimate	SE of parameter estimate
Heredity	0.04	0.47	0.23
BMI	<0.0001	0.16	0.03
WHR	<0.0001	9.34	2.01
Physical inactivity	0.02	0.50	0.22

Independent risk factors for diabetes incidence according to the stepwise model. *$P < 0.05$; †$P < 0.01$.

Stepwise analyses with backward elimination including alcohol intake and all covariates studied showed six variables that were statistically and independently associated to mortality: age, WHR, smoking, systolic blood pressure, triglyceride levels, and physical inactivity; all were positively related to mortality (Table 3).

Increase of alcohol intake during 1968–1969 through 1980–1981 in relation to 20-year incidence of diabetes and mortality

Diabetes incidence. There was no relationship between increase of alcohol intake expressed in grams per month between the first examination in 1968–1969 and the examination in 1980–1981 and diabetes incidence between 1980–1981 and 2000–2001 when age and alcohol intake in 1968 were included as covariates (Table 4). Further covariate adjustment did not change this result, except for the model including heredity, which displayed a significant inverse relationship between increase in alcohol intake and diabetes incidence.

Mortality. Increase in alcohol intake between the two examinations was significantly inversely related to mortality when age and baseline alcohol intake were included as covariates. This inverse significant relationship remained after adjustment for all other covariates studied (Table 4).

CONCLUSIONS — Whether moderate alcohol consumption conveys a protective effect for mortality and cardiovascular disease has been a topic of intensive research and debate. Strong evidence of the cardiovascular benefits of moderate

Table 3—*RRs and 95% CIs from proportional hazards model for the association between alcohol intake and all-cause mortality during 32 years*

	Variables							
	Beer		Wine		Hard liquor		Alcohol (100 g/month)	
Covariates	RR	95% CI	RR	95% CI	RR	95% CI	RR	95% CI
Age	0.96*	0.92–1.00	0.95*	0.91–0.99	1.03	0.97–1.08	0.99	0.94–1.04

Stepwise model: independent risk factors for all-cause mortality

	P value	Maximum likelihood estimate	SE of parameter estimate
Age	<0.0001	0.11	0.01
WHR	0.02	2.69	1.14
Smoking	<0.0001	0.78	0.12
Systolic blood pressure	0.0003	0.009	0.002
Triglycerides	0.001	0.004	0.001
Physical inactivity	0.03	0.24	0.11

Independent risk factors for all-cause mortality according to the stepwise model. *$P < 0.05$; †$P < 0.01$.

Table 4—*RRs and 95% CIs from proportional hazards model for the association between change of alcohol intake between 1968–69 and 1980–81 on type 2 diabetes incidence and all-cause mortality during 20 years*

	Alcohol increase (100 g/month)			
	Diabetes		Mortality	
Covariates	RR	95% CI	RR	95% CI
Age	0.88	0.72–1.06	0.88†	0.80–0.97
Age + heredity	0.75*	0.56–0.99	0.87*	0.77–0.98
Age + BMI	0.94	0.78–1.13	0.88†	0.80–0.97
Age + WHR	0.90	0.73–1.10	0.91*	0.83–0.99
Age + smoking	0.87	0.71–1.05	0.86†	0.78–0.94
Age + systolic blood pressure	0.89	0.73–1.08	0.89*	0.81–0.97
Age + triglycerides	0.88	0.72–1.07	0.88†	0.80–0.97
Age + cholesterol	0.88	0.72–1.06	0.88†	0.80–0.97
Age + education	0.88	0.72–1.06	0.89*	0.81–0.97
Age + physical activity	0.88	0.73–1.06	0.88†	0.80–0.96
Age + social group	0.88	0.73–1.07	0.89*	0.91–0.98

All models adjusted for baseline consumption levels. *$P < 0.05$; †$P < 0.01$.

alcohol consumption among men and women must be weighed against the likelihood of excess all-cause mortality occurring at high consumption levels, together with a small but well-documented risk of excess of breast cancer among female drinkers and the broader social costs of heavy alcohol use (19–21). Whether alcohol intake itself is protective for diabetes or whether it is a marker for other health-related lifestyle characteristics remains uncertain. For example, both smoking and obesity are potential confounding factors which may be related both to the exposure (alcohol) as well as to diabetes. In the case of smoking, we found that the association between alcohol and diabetes was stable after adjustment for smoking habits. On the other hand, the BMI-adjusted analyses suggested that obesity is the most powerful explanatory factor for an apparent protective effect of alcohol on diabetes, simply because alcohol-consuming women were less likely to be obese at baseline, a relationship that has been observed in other studies. Previous observations by other investigators that the association is independent of BMI may be explained by differences in measurements (e.g., self-reported versus measured body weight), larger sample sizes, or different underlying alcohol-obesity-social class relationships in different populations. In a stepwise model, including alcohol intake and all covariates studied, only four variables (heredity, BMI, WHR, and physical activity) were independently related to diabetes.

In Sweden there has been a gradual shift of alcohol consumption among social classes over the latter decades. Whereas for women today, alcohol consumption is fairly similar among lower and higher social layers, the distribution was different in the 1960s. At that time both total alcohol consumption and binge drinking were more common among wealthier women, presumably as alcohol consumption follows the "luxury index." Because BMI is negatively correlated with higher social status, it is likely that leaner subjects would drink more, due to socioeconomical confounding. Another possible contributing factor is that leaner subjects often have a higher water-to-fat ratio in their bodies. This might imply that leaner subjects drink relatively more to achieve a given alcohol concentration. It is further likely that this principle has more impact on subjects with small or moderate alcohol intake, as the effect rationally would taper off once a high alcohol concentration is reached. Most women actually drink small or moderate amounts of alcohol, and this finding was even more pronounced in 1968, when the initial examination in the present population study was carried out.

Inconsistencies between this and other research must also be considered in the context of biases such as errors in self-reported alcohol intake. The relative validity of our interview about alcohol habits has been assessed previously and judged satisfactory by comparisons with alcohol values obtained by a separate dietary interview (8). However, a number of limitations to our exposure data should be pointed out. For instance, this method is unable to capture episodic ("binge") drinking patterns. Also, beer may not perform as well as other alcohol sources in this analysis because the category combined lower alcohol content varieties of beer traditionally consumed at meals with less frequently consumed higher-alcohol varieties served on other occasions. Moreover, in view of the large number of women reporting to be lifetime teetotalers (21%) it seems possible that alcohol intake may have been underreported due to social desirability and/or recall biases, which could potentially distort associations with end points and affect our results if the end point is not uniform across categories. Another methodological limitation of this and much previous work is the fact that alcohol measures were only taken at a single point. Therefore, we attempted to study effects of increasing alcohol intake during the first 12 years of observation on end points occurring over the following 20 years. These results were unexpected in that they were inconsistent with those from the simple baseline model. Specifically, increases in alcohol intake did not have the same protective effect on subsequent diabetes, but the same increases of alcohol intake significantly reduced all-cause mortality. Given the well-documented increase in alcohol consumption by Swedish women in recent decades, the observation may have public health implications. Furthermore, some of the covariates in the present article are based on self-report such as heredity, education, smoking, and physical activity and may thus be methodological limitations.

Thus, this analysis of 38- to 60-year-old Swedish women followed for 32 years demonstrated that the inverse association between intake of alcohol expressed in grams as well as intake of hard liquor and incident diabetes could be largely explained by confounding due to BMI. The most robust association seemed to be between the increase in alcohol intake and longevity. Our results indicate that the often-expressed concern about moderate alcohol consumption may be unjustified.

In summary, our study lends support to previous research suggesting a protective effect of alcohol on diabetes but suggests that

the effect will be exaggerated if results are not adjusted for relative weight.

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APPENDIX

Conversions to grams of alcohol monthly for beer, wine, and hard liquor according to different categories in the questionnaire

	Beer (g)	Wine (g)	Hard liquor (g)
"Never"	0	0	0
"Monthly"	16	43	38
"Weekly"	62	172	152
"Several times a week"	124	344	304
"Daily"	234	645	570

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