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Associations between alcohol consumption and body fat distribution in type 1 diabetes

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ABSTRACT

Aim: To evaluate the associations between alcohol consumption and body fat distribution in type 1 diabetes (T1D).

Methods: DXA assessed the body composition of 548 adults with T1D from the Finnish Diabetic Nephropathy Study. Visceral fat mass (VFM) ≥ 0.7% of body weight for women and ≥ 1.1% for men defined central obesity (CO), whereas body fat mass (BFM) ≥ 40.4% for women and ≥ 31.8% for men defined general obesity (GO). Alcohol consumption data were collected via questionnaires. One standard dose = 12 g of pure alcohol. Participants were classified as abstainers, low-risk, moderate-risk and high-risk alcohol consumers. We used linear and logistic regression models for analyses.

Results: The higher the alcohol consumption the higher the VFM% ($r^2 = 0.23$, $\beta = 0.083$, $p = 0.04$) in both sexes. BFM% presented a similar pattern in men ($r^2 = 0.12$, $\beta = 0.160$, $p = 0.01$), but not in women. One weekly dose increase of alcohol consumption increases the odds of CO by 3% (OR 1.03, $p = 0.037$), but not GO. The odds of CO (OR 7.3, $p = 0.003$) and GO (OR 5.3, $p = 0.007$) increase with high-risk, but not with low- and moderate-risk consumptions.

Conclusions: In adults with T1D, alcohol consumption is linearly associated with VFM% regardless of sex, whereas the association with BFM% is sex-dependent.

1. Introduction

Harmful drinking is one of the world's leading risk factors for premature mortality, causing over 3 million deaths every year, mainly because of injuries, liver cirrhosis, cancers, and cardiovascular disease [1,2]. Excessive alcohol consumption by individuals with type 1 diabetes, has been associated with hypoglycaemia, and glucose variability [3] as well as with diabetic nephropathy and retinopathy [4].

Another major health issue alongside alcohol consumption, obesity, has also kept growing to epidemic proportions. Obesity has nearly tripled in the past 50 years [5] and people with type 1 diabetes have also been gaining weight during the last decades [6]. Nevertheless, it has been recognized that fat distribution has a greater impact on the risk of several chronic diseases than total body fat, explaining the association between central obesity and all-cause mortality [7]. Our group has shown that adults with type 1 diabetes and albuminuria have a higher visceral fat percentage compared to those without albuminuria [8]. Furthermore, we have shown that central obesity has a stronger association with non-alcoholic fatty liver [9], hospitalization or death due to heart failure [10] and severe eye disease in this population [11] than general obesity.

Considering that several factors such as alcohol consumption may be related to body fat distribution [12–15], in the present study, we aimed to explore the associations between alcohol consumption and general obesity or central obesity in adults with type 1 diabetes. In the general population, the literature has generated conflicting results regarding this topic [12,16] and data on people with type 1 diabetes are even more scarce. Therefore, given that both excessive alcohol use and obesity are

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expanding health concerns, we evaluated the association between alcohol consumption and body fat distribution in adults with type 1 diabetes.

2. Methods

2.1. Study design and participants

This is a cross-sectional study aiming to explore the associations between alcohol consumption and body fat distribution in adults with type 1 diabetes. Inclusion criteria: individuals with type 1 diabetes with body composition data assessed by dual-energy X-ray absorptiometry (DXA) and alcohol consumption information from the questionnaires of the Finnish Diabetic Nephropathy (FinnDiane) Study. Exclusion criteria: Individuals that reported to be former alcohol consumers were not included because we did not know for how long they had been without drinking any alcohol. Therefore, the association between alcohol consumption and body fat distribution would be difficult to evaluate. No other exclusion criteria were applied in this study.

Of 5500 participants with type 1 diabetes in the FinnDiane Study cohort, 579 individuals had their body composition assessed by DXA during a regular visit between 2011 and 2019. Of those 579 individuals, 565 had provided information about their alcohol consumption in the FinnDiane questionnaires. We did not include 17 individuals who reported as former drinkers. In the end, 548 individuals were included in the study.

The FinnDiane Study is a nationwide, observational, comprehensive multi-center study that started in 1997 and recruitment is still ongoing. The FinnDiane Study intends to identify genetic and environmental risk factors for diabetic complications, with special emphasis on diabetic nephropathy in individuals with type 1 diabetes. This research was approved by the Ethics Committee of Helsinki and Uusimaa Hospital District, and the study was designed according to the Declaration of Helsinki. Written informed consent was obtained from each FinnDiane Study participant.

2.2. Body fat distribution

Body fat distribution was assessed by DXA (GE Healthcare Lunar version 16, Wisconsin, USA), which was part of the regular procedures during the FinnDiane Study visits since 2011. The DXA scan was used according to the manufactureŕs instructions and visceral fat was measured by CoreScan [17]. The total body fat mass percentage (BFM %), visceral fat mass percentage (VFM%), gynoid fat mass percentage (GFM%) and appendicular fat mass percentage (AppFM%) were calculated by dividing the fat mass (grams) by total body weight (grams), multiplied by 100. Appendicular fat mass is related to the fat in both arms and legs. Based on our previous research, we used a VFM% threshold of $\geq$ 0.7 for women and $\geq$ 1.1 for men to define central obesity, and a BFM% $\geq$ 40.4 for women and $\geq$ 31.8 for men to define general obesity [8].

2.3. Alcohol consumption

Data on alcohol consumption were collected from participants via questionnaires during each FinnDiane visit. Participants were requested to report their weekly consumption of different beverage types such as beer (one-third of a liter bottle), wine (glasses), and stronger spirits (deciliters). The amount of alcohol was then converted into standard doses. One standard dose contains 12 g of pure alcohol, which is equivalent to 33 cl of beer, 12 cl of wine, and 4 cl of spirits [18]. Participants who reported to be current alcohol consumers, but drinking alcohol only occasionally, we considered their consumption as 0.1 standard doses per week to differentiate them from the abstainers, since their consumption was less than one dose per week. Then, participants were classified based on their alcohol consumption (dose/week) and the risk of health problems related to alcohol consumption according to the Finnish guidelines: abstainers, low risk (0.1–6.9 doses for women, 0.1–13.9 for men), moderate risk (7–15.9 for women, 14–23.9 for men) and high risk ($\geq$16 for women, $\geq$24 for men) [18]. In the questionnaire, former drinkers were distinguished from abstainers by an inquiry where participants were able to report if they have been previously consuming alcohol but had since stopped. To identify former drinkers, there is an option such as “I do not drink alcohol anymore, but I have used it in the past regularly”. Additionally, they provided information regarding the amount of alcohol they have been consuming in the past. To identify the abstainers, there is another option to indicate such as “I do not drink alcohol at all”.

The participants were additionally divided into groups of beverage types. If more than 50% of the total amount of alcohol consumed in a week was of one type of alcoholic beverage, the individual was classified as a wine drinker, beer drinker or spirit drinker. Otherwise, the individual was considered a mixed drinker. The subgroup analysis regarding beverage type and body fat distribution was conducted on the participants, who had reported drinking regularly (271 participants).

2.4. Diabetic nephropathy (DN) stage

At least two out of three urine samples defined the DN stages according to the urinary albumin excretion rate (UAER) as normoalbuminuria (UAER $<$ 20 μg/min or $<$ 30 mg/24 h), moderate albuminuria (UAER $\geq$ 20 and $<$ 200 μg/min or $\geq$ 30 and $<$ 300 mg/24 h), and severe albuminuria (UAER $\geq$ 200 μg/min or $\geq$ 300 mg/24 h). Individuals with moderate or severe albuminuria were pooled together for the analyses and are referred to throughout the paper as the albuminuric group. Individuals with end-stage renal disease (either on dialysis or having received a kidney transplant) were not included in this study.

2.5. Statistical analyses

Data are presented as means $\pm$ SD for normally distributed values, as median with interquartile range for non-normally distributed values, and percentages for categorical variables. Differences between groups were explored using the analysis of variance (ANOVA) test for normally distributed variables and the Kruskal-Wallis test for variables not normally distributed. Categorical variables were analyzed with the $\chi 2$ test or Fisheŕs test when the expected number was below five.

The associations between the amount of alcohol consumption in doses per week and body fat compartments were evaluated with linear regression models adjusted for sex, age, glycated hemoglobin ($HbA1_c$) and DN stage, when the association between the variables were linear. Men and women were analysed separately, if a sex interaction term was present, otherwise, they were pooled together for the analysis.

Logistic regression models adjusted for confounders were used to evaluate the associations between the independent variables and central obesity or general obesity. The amount of alcohol consumption in doses per week (continuous variable), the groups of alcohol consumption according to the risk for health problems (nominal variable) and the groups of beverage type (nominal variable) were considered the independent variables, whereas central obesity and general obesity were the dependent variables in separated models. The analyses using logistic regression models were adjusted for age, glycated hemoglobin ($HbA1_c$) and DN stage, but sex was not included in the models because the dependent variables were built based on sex thresholds.

Additionally, we conducted sensitivity analyses including 386 individuals that had answered the questionnaires regarding their leisure-time physical activity (LTPA). The total LTPA was converted into the metabolic equivalent of task (MET) and additionally included in the models as a covariate with age, HbA1c and DN stage.

Abstainers were used as the reference group in the logistic regression model when the groups of alcohol consumption were considered as the independent variable. The wine drinkers were used as the reference

group when the groups of beverage type were considered as the independent variable. Results are presented as odds ratios (OR) with 95% confidential intervals (95% CI). The analyses were performed using IBM SPSS statistics version 27 (IBM Corporation, Armonk, NY, USA).

3. Results

Of a total of 548 individuals, 30.1% presented with general obesity and 46.5% with central obesity. The median and interquartile range (IQR) of age was 42 (34, 53) years, the duration of diabetes was 25.4 (19.6, 37.5) years and 40% of the individuals were men. High-risk consumers had a median alcohol intake of 28 (24, 36), moderate-risk 12 [8,19] and low-risk 0.1 (0.1, 4.0) doses per week. High-risk consumers had the highest VFM% [2.19 (0.93–2.48), p = 0.002] and the lowest AppFM% [12.2 (10.1–15.1), p = 0.04]. Conversely, BFM% did not differ (p = 0.40) between the groups (Table 1). Furthermore, the group of high-risk consumers had the highest proportion of individuals with central obesity [79%, p = 0.03] and general obesity [63%, p = 0.003] (Fig. 1). Of note, the higher consumption of alcohol the higher the proportion of individuals with central obesity, although this pattern was not observed concerning general obesity (Fig. 1). Detailed clinical characteristics according to the groups of alcohol consumption are depicted in Table 1.

Due to significant interaction term regarding sex (p < 0.001), the associations between alcohol consumption in doses per week and BFM %, GFM% and AppFM% were analysed separately by sex. However, for the association with VFM%, men and women were pooled together, since there were no interactions between sex (p = 0.66). Including all individuals, the higher consumption of alcohol, the higher the VFM% in the unadjusted ($r^2 = 0.05$, $\beta = 0.217$, $p < 0.001$) and after adjusting for sex, age, HbA1c and DN stage ($r^2 = 0.23$, $\beta = 0.083$, $p = 0.041$). The linear regressions between alcohol consumption in doses per week and VFM% in men and women are shown in Fig. 2. The relationship between alcohol consumption and BFM%, GFM% and AppFM% was linear only in men. The higher the alcohol consumption the higher BFM% ($r^2 = 0.12$, $\beta = 0.160$, $p = 0.01$) and GFM% ($r^2 = 0.08$, $\beta = 0.152$, $p = 0.02$) was observed in the fully adjusted model. Despite not achieving statistical significance, the association with AppFM% ($r^2 = 0.05$, $\beta = 0.127$, $p = 0.059$) was in the same direction. In women, no linear association was found between alcohol consumption and BFM%, GFM% or AppFM%, and no difference between groups of alcohol consumption was observed regarding BFM% and AppFM% (Supplementary Fig. 1). Of note, women with a low-risk consumption presented with the highest GFM% (p = 0.009) and a trend of decreasing GFM% according to the increase in alcohol consumption was observed (Supplementary Fig. 1).

Using a binary logistic regression model adjusted for confounders and the amount of alcohol consumption as a continuous variable, we found that each one dose increase per week of alcohol consumption was associated with 3% higher odds of having central obesity [OR 1.03, 95% CI (1.002–1.055), p = 0.037]. However, this association was not seen for general obesity [OR 1.02, 95% CI (0.99–1.04), p = 0.17], possibly due to a non-linear relationship between alcohol consumption and general obesity as shown in Fig. 1. Using a nominal logistic regression model adjusted for confounders and the groups of alcohol consumption as the independent variable, high-risk consumption was associated with increased odds of having central obesity [OR 7.3, 95%CI (1.8–27.6), p = 0.003] and general obesity [OR 5.3, 95%CI (1.6–18.0), p = 0.007]. However, low- and moderate-risk consumptions were not.

Regarding the groups of beverage types, beer was the most common beverage among our study population, whereas only eight individuals were classified as spirit drinkers (Table 1). Due to the small sample size of the spirit drinkers' group, it was not possible to perform a logistic regression for the association between the groups of beverage types and central or general obesity.

Table 1
Clinical characteristics according to the groups of alcohol consumption.

Variable	All	Abstainers	Low-risk	Moderate-risk	High-risk	p-value
n	548	35	443	51	19	
Men (%)	218 (39.8)	11 (31.4)	171 (38.6)	22 (43.1)	14 (73.7)	0.014
Age, years	42 (34–53)	55 (38–63)	41 (33–51)	51 (41–59)	48 (39–55)	<0.001
Age at onset of diabetes, years	14.1 (9.4–22.0)	15.6 (11.1–26.0)	13.6 (8.8–21.4)	15.7 (11.7–24.9)	19.8 (12.5–29.0)	0.013
Duration of diabetes, years	25.4 (19.6–37.5)	34.0 (20.8–44.3)	24.6 (19.3–36.9)	29.7 (20.6–41.3)	24.7 (20.2–36.9)	0.028
Systolic blood pressure, mmHg	132 (121–144)	137 (123–149)	130 (120–143)	139 (129–156)	145 (133–159)	<0.001
Diastolic blood pressure, mmHg	76 ± 8.87	73 ± 8.97	76 ± 9.03	79 ± 7.16	81 ± 6.39	0.007
HbA1c, %	7.8 (7.1–8.6)	8.2 (7.0–9.3)	7.8 (7.1–8.6)	8.1 (7.4–8.9)	8.1 (7.5–8.6)	0.22
HbA1c, mmol/mol	62.0 (54.0–71.0)	66.0 (53.0–78.0)	62.0 (54.0–71.0)	65.0 (57.0–74.0)	65.0 (58.0–70.0)	0.22
Total cholesterol, mmol/l	4.48 (3.98–5.06)	4.31 (3.84–4.87)	4.45 (3.97–4.99)	4.73 (4.20–5.40)	5.06 (4.35–6.30)	<0.001
HDL-cholesterol, mmol/l	1.55 (1.27–1.84)	1.45 (1.18–1.7)	1.53 (1.26–1.82)	1.77 (1.48–2.17)	1.52 (1.25–2.07)	0.004
Triglycerides, mmol/l	0.90 (0.69–1.34)	0.92 (0.75–1.3)	0.90 (0.69–1.30)	0.86 (0.66–1.45)	1.34 (0.90–2.33)	0.018
BMI, kg/m2	25.9 (23.2–28.7)	24.1 (21.3–28.1)	25.9 (23.2–28.7)	25.5 (23.2–27.9)	29.6 (26.3–32.0)	0.007
WHtR	0.50 (0.46–0.57)	0.49 (0.44–0.57)	0.50 (0.46–0.56)	0.50 (0.46–0.56)	0.57 (0.52–0.61)	0.008
BFM, %	32.3 ± 8.69	30.8 ± 8.94	32.5 ± 8.91	30.9 ± 6.84	33.3 ± 7.34	0.40
VFM, %	0.77 (0.36–1.47)	0.72 (0.37–1.12)	0.73 (0.36–1.40)	0.89 (0.34–1.57)	2.19 (0.93–2.48)	0.002
GFM, %	5.41 (4.20–6.73)	5.04 (3.86–6.41)	5.57 (4.25–6.82)	5.31 (4.20–6.03)	4.76 (3.93–5.53)	0.12
AppFM, %	14.2 (11.0–17.4)	14.5 (11.0–17.1)	14.3 (11.1–17.9)	13.2 (10.5–16.2)	12.2 (10.1–15.1)	0.04
Diabetic nephropathy stages						0.06
Normoalbuminuria (%)	426 (77.7)	22 (62.9)	352 (79.5)	40 (78.4)	12 (63.2)	
Albuminuria (%)	122 (22.3)	13 (37.1)	91 (20.5)	11 (21.6)	7 (36.8)	
Leisure-time physical activity, MET*	15.92 (6.64–31.37)	16.4 (3.26–34.2)	16.2 (6.84–31.54)	13.8 (7.1–30.8)	12.1 (6.52–27.6)	0.96
Alcohol consumption, standard drinks	0.1 (0.1–6)	0	0.1 (0.1–4)	12 (8–19)	28 (24–36)	<0.0001
Beverage consumption**						0.36
Beer drinker (%)	140 (51.7)		103 (51.2)	26 (51.0)	11 (57.9)	
Wine drinker (%)	74 (27.3)		58 (28.9)	14 (27.5)	2 (10.5)	
Spirit drinker (%)	8 (3.0)		5 (2.5)	1 (2.0)	2 (10.5)	
Mixed drinker (%)	49 (18.1)		35 (17.4)	10 (19.6)	4 (21.1)	

* Leisure time physical activity data are available from 386 individuals. Percentages reported as valid percentages.
** Beverage consumption data are available from 271 individuals. Percentages reported as valid percentages.
Data are shown as means ± SD for normally distributed values, as medians (IQR) for non-normally distributed values and as percentages for categorical variables. Comparisons between groups were performed with analysis of variance (ANOVA), Kruskal–Wallis test or $\chi 2$ test, respectively. HbA1c, glycated hemoglobin; HDL, high-density lipoprotein; BMI, body mass index; WHtR, waist-to-height ratio; BFM, body fat mass; VFM, visceral fat mass; GFM, gynoid fat mass; AppFM, appendicular fat mass; MET, metabolic equivalent of task.

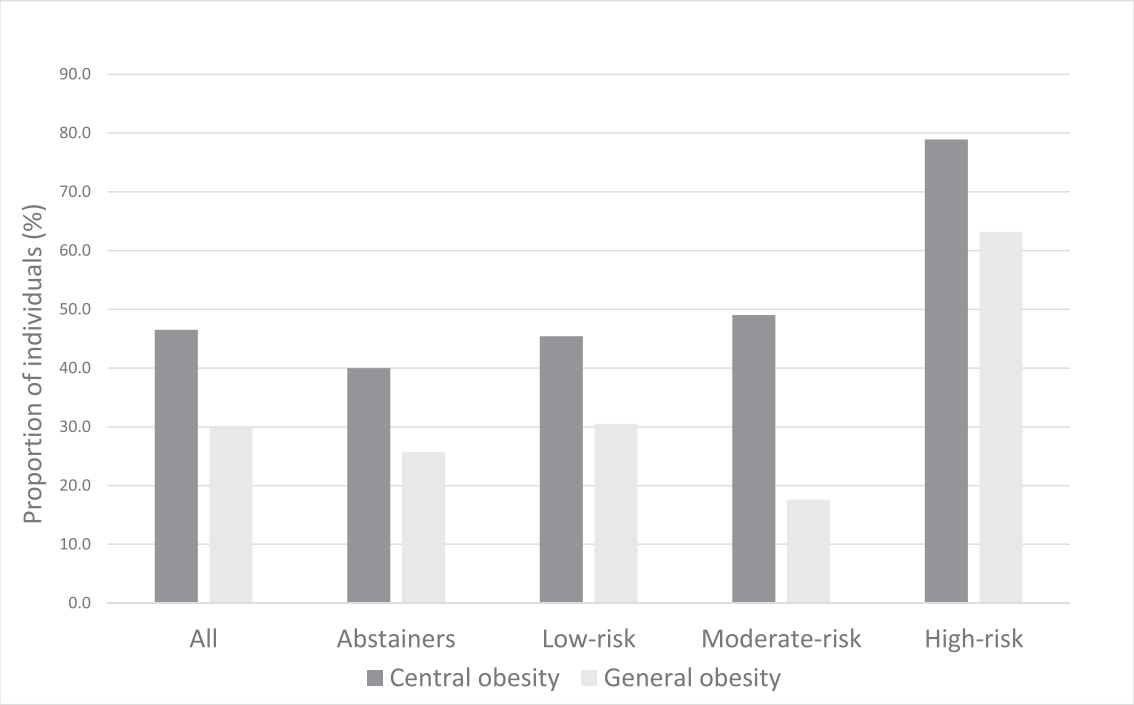

Fig. 1. The proportion of individuals with general and central obesity according to the groups of alcohol consumption. The group of high-risk consumption have the highest proportion of individuals with general obesity ($p = 0.003$) and central obesity ($p = 0.03$). Comparisons between groups were done with the $\chi 2$ test or Fisheŕs test when the expected number is below five.

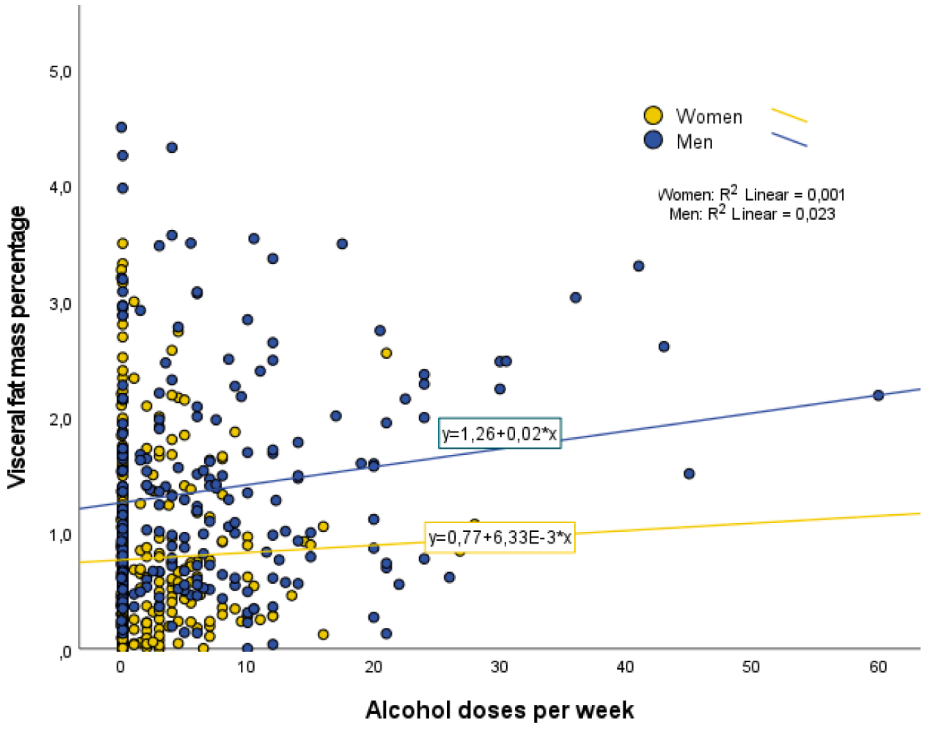

Fig. 2. Linear regression between alcohol consumption in doses per week and visceral fat mass percentage in men and women. Blue dots represent men and yellow dots represent women.

There was no difference between the groups regarding LTPA (Table 1). Out of 386 individuals that reported LTPA, 145 were men (66.5% of the men) and 241 were women (73% of the women). Out of 145 men who reported LTPA, 8.3% reported no LTPA whereas 6.6% out of 241 women reported no LTPA, which means that individuals who reported LTPA were mostly active people. In the sensitivity analysis including LTPA as a covariate beyond age, DN stage and $HbA1_c$, the amount of alcohol consumption did not yield any significant results regarding central obesity ($p = 0.23$) or general obesity ($p = 0.34$).

4. Discussion

This study brings important and novel information on alcohol consumption's relationship with body fat distribution measured by DXA in people living with type 1 diabetes. We found that alcohol consumption is linearly associated with VFM% and increases the odds of having central obesity by 3% per each weekly dose increase of consumed alcohol. Notably, the associations between alcohol consumption and other fat compartments such as BFM%, GFM% and AppFM% vary according to sex. Furthermore, a high-risk consumption of alcohol is associated with both central and general obesity, but low- and moderate-risk consumption is not.

Some studies have reported that the volume of consumed alcohol, especially heavy drinking is associated with different measures of adiposity [15,16,19–21]. On the other hand, similar to our results, other studies have shown that light and moderate alcohol consumption have not been linked to obesity, defined by BMI [16,21]. Different from previous studies that used anthropometric measurements, we used DXA to assess body fat compartments which is a more accurate method and enables a better understanding of the association between alcohol consumption and metabolically different types of fat tissue, such as peripheral fat (GFM%, AppFM%) which is considered a metabolically good fat and visceral fat (VFM%), considered a bad fat [22]. In our study, we observed increased odds of central and general obesity with high-risk consumption but not with low- and moderate-risk consumption, which is aligned with a recent publication that showed light alcohol consumption is associated with a minimal increase in cardiovascular risk and heavier consumption is associated with an exponential risk increase [2].

Interestingly, we observed that low- and moderate-risk groups presented a lower prevalence of albuminuria than the abstainers and high-risk consumers. A similar finding has been reported in the EURODIAB study [23] in which the association between alcohol consumption and diabetic microvascular complications (including nephropathy) has a U-shape and moderate consumers have the lowest risk. In another study regarding alcohol consumption and the risk of microvascular complications in type 1 diabetes [4] it was also observed that lifelong abstainers have a higher risk of nephropathy compared with light consumers. The reason why abstainers have a higher prevalence of nephropathy compared to moderate consumers is not well understood. However, important factors not measured in this study such as behavioral, social, demographic and genetics [24] may possibly explain the differences. Independently of groups of consumers, most importantly, is the increased linear risk of VFM% accumulation according to the increase in alcohol consumption.

A common explanation of how alcohol consumption influences weight gain is usually related to its high-energy content (7.1 kcal/g) added to the total daily caloric intake [12], or that alcohol may promote food intake by inhibiting hormones linked to satiety such as leptin and glucagon-like peptide-1 [16]. However, an increase in total caloric intake does not fully explain the association between alcohol consumption and fat distribution. A Korean study found a positive association between alcohol consumption and increased visceral adipose tissue (VAT), whereas a negative association with subcutaneous adipose tissue (SAT) [19]. In our study, we also found a positive association between alcohol consumption and VFM% independent of sex, however, the association with peripheral fat (GFM%) pointed towards different directions in men and women. Differences in the relationship between alcohol consumption and SAT according to sex were previously observed in a cross-sectional Framingham Heart Study [25], in which a lower SAT at the abdominal area measured by computed tomography was seen in women consuming more than seven doses of alcohol per week compared to those who consumed less, whereas in men, there was no change in SAT according to alcohol consumption [25]. A cross-over randomized controlled trial (RCT) including 34 men showed no differences in subcutaneous and abdominal fat measured by ultrasound after moderate alcohol intake for four weeks [26]. Of note, this RCT included only men, a short period of alcohol exposure and had a small sample size. So far, the mechanism by which alcohol affects SAT/peripheral fat compartments such as GFM% has not been elucidated in previous studies and we cannot explain it either with this current observational study setup. However, we can speculate that alcohol may interfere with body fat distribution driven by sex hormones, which could explain the differences in peripheral fat accumulation between men and women.

Regarding alcohol and VFM%, alcohol intake enhances cortisol secretion and in that manner [25] may lead to an increase in abdominal fat as well as steatosis [27]. Alcohol consumption is also a well-known risk factor for non-alcoholic steatohepatitis [28] which is strongly related to VAT [29]. Although the association between alcohol consumption and abdominal fat is not new, the main novelty of the present study is the sex-independent linear association between alcohol consumption and visceral fat, especially in individuals with type 1 diabetes, a population that we showed central obesity increases the risk of several diabetes complications.

The lack of significant changes in BFM% in women when alcohol consumption increases might be explained by the trend of decreasing GFM% combined with the increase of VFM%. On the other side, in men, we saw an increase in BFM% that may be a consequence of the increase in VFM% and GFM% along with the increase in alcohol intake. Another possible explanation for the difference between BFM% accumulation according to sex is the small number of women in the high-risk consumption group. The majority of women were in the group of low- and moderate-risk consumption. Therefore, the amount of consumed alcohol was possibly not high enough to impact the total body fat, although it was important to influence peripheral fat accumulation. It seems that low and moderate alcohol consumption by women may interfere with peripheral body fat accumulation, but a higher amount of alcohol consumption would be necessary to affect total body fat. Most importantly, in the current study, we showed that alcohol consumption is linearly associated with visceral fat (the bad fat) independently of sex.

Physical activity is a significant confounder in studies regarding obesity and body composition since it promotes fat mass reduction and muscle mass increase [30]. Furthermore, a higher physical activity level is inversely associated with VAT and SAT [25]. In our sensitivity analysis, the association between alcohol consumption and central or general obesity was no more significant after adjusting for LTPA. Of note, around 30% of the individuals did not answer the LTPA questionnaire and those who answered were mostly active individuals, which highlights the relevant impact of physical activity on body composition.

Nevertheless, beyond the association between alcohol consumption and body fat distribution, it is important to consider that alcohol consumption has a major impact on other health-related outcomes such as intoxication and injuries, alcohol-related liver disease, cancer, and alcohol dependency [18].

Our study has some limitations to be considered. This is a cross-sectional study, therefore, it is not possible to infer any causality regarding alcohol's effect on fat distribution. Second, alcohol consumption was based on self-reported questionnaires, therefore, we have to take into consideration that participants may under- or overestimate their alcohol consumption. A small number of individuals in high-risk consumption group may impact the analysis by groups of consumers as a nominal variable. However, the most important finding of this study is the linear association between alcohol consumption, as a continuous variable, and VFM% which is unaffected by group numbers. We also had missing data on LTPA, but we tried to cover this limitation by performing a sensitivity analysis.

Our study has also several strengths, and the major one is the assessment of body composition by DXA instead of anthropometric measurements, which have been widely used in previous studies. Furthermore, with fat compartment measures, we could better evaluate the association between alcohol consumption and different types of body fat tissue such as central fatness (VFM%) which is considered a

metabolically bad fat and peripheral fatness (GFM%, AppFM%) which is considered a metabolically good fat, as well as total body fat (BFM%) [22]. Second, we have a complete clinical characterization of the participants and a detailed alcohol consumption questionnaire that allowed the participants to report the amount of consumed alcohol. Another strength of our questionnaire is the differentiation between abstainers from former drinkers, who usually quit drinking due to health-related problems which may impact the analyses. The detailed questionnaire on LTPA allowed us to calculate the MET, which is an important variable for body fat distribution.

Our observational results motivate future studies to explore the mechanisms involved in the relationship between alcohol consumption and body fat distribution, considering not only the effect of the amount of alcohol consumed but also behavioral, demographic, social and genetic factors. Moreover, given that alcohol consumption is associated with increased odds of central obesity, which has been linked to diabetes complications in type 1 diabetes, longitudinal studies are necessary to evaluate the impact of alcohol consumption on diabetes complications adjusted for central obesity. Future studies are needed to answer the question of whether increasing VFM% with a trend of decreasing GFM% (metabolically good fat) will lead to worse cardiovascular outcomes in women compared to men with similar alcohol consumption.

In conclusion, our study showed in adults with type 1 diabetes that VFM% increases linearly with the increase of alcohol consumption independently of sex, whereas the association with BFM% and GFM% is sex-dependent. Furthermore, each dose increase of alcohol consumption per week increases the odds of central obesity by 3%. However, consumption of $\geq$ 24 standard doses of alcohol per week for men and $\geq$ 16 standard doses per week for women, is associated with both central and general obesity.

Declaration of Competing Interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: [P-H.G. reports receiving lecture honorariums from Astellas, AstraZeneca, Bayer, Boehringer Ingelheim, Eli Lilly, Elo Water, Medscape, MSD, Mundipharma, Novo Nordisk, PeerVoice, Sanofi, Sciarc, and being an advisory board member of AbbVie, Astellas, AstraZeneca, Bayer, Boehringer Ingelheim, Eli Lilly, Medscape, MSD, Mundipharma, Nestlè, Novo Nordisk, and Sanofi. EBP reports receiving lecture honorariums from Eli Lilly, Abbott, Astra Zeneca, Sanofi, and Boehringer Ingelheim. All other authors (I.L. and V.H) report no conflicts of interest.

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Author Contributions

I.L. was responsible for data collection, statistical analyses, interpreting the results and writing the manuscript. E.B.P was responsible for the concept and study design, interpreting the results, supervising the study analyses, and writing the manuscript. V.H. was responsible for acquisition of the clinical data and critical revision of the manuscript. P-H.G. supervised the study and is the guarantor of this work, had full access to all the data and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Appendix A. Supplementary material

Supplementary data to this article can be found online at https://doi.org/10.1016/j.diabres.2023.110891.

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