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# Higher plain water intake is associated with lower type 2 diabetes risk: A cross-sectional study in humans

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## Abbreviations

BMI; body mass index

CI; confidence interval

IPAQ; International Physical Activity Questionnaire

FFQ: Food Frequency Questionnaire

MET: Metabolic equivalent of task

PA; physical activity

SED; standard error of difference

SEM; standard error of the mean

SSB; sugar-sweetened beverage

T2D; type 2 diabetes

# Higher plain water intake is associated with lower type 2 diabetes risk: A cross-sectional study in humans

*Abstract*

The aim of this study was to investigate the relationship between plain water intake and type 2 diabetes (T2D) risk. It was hypothesized that higher plain water intake would be associated with a lower T2D risk score. 138 adults from Southwest and Southeast England answered a cross-sectional online survey assessing T2D risk (using the Diabetes UK risk assessment), physical activity (using the short International Physical Activity Questionnaire), and consumption of fruits, vegetables and beverages (using an adapted version of the Cambridge European Prospective Investigation into Cancer and Nutrition Food Frequency Questionnaire). There was a trend for differences in mean plain water intake between those stratified as having low, increased, moderate or high risk of T2D but these did not achieve significance ($P$=0·084). However, plain water intake was significantly negatively correlated with T2D risk score (τ=-0·180, $P$=0·005), and for every 240mL cup of water consumed per day, T2D risk score was reduced by 0·72 points (range 0-47) (B=-0·003, 95% CI=-0·006, -0·001, $P$=0·014). The current study has provided preliminary results which are supported by theory; mechanisms need to be explored further to determine the true effect of plain water intake on disease risk. As increasing plain water intake is a simple and cost effective dietary modification, its impact on T2D risk is important to investigate further in a randomized controlled trial. Overall, this study found that plain water intake had a significant negative

correlation with T2D risk score and regression analysis suggested that water may have a role in reducing T2D risk.

**Key words:** Cross-sectional studies; Diabetes Mellitus, Type 2; Humans; Life style; Risk assessment; Water

*1. Introduction*

Type 2 diabetes (T2D) is a complex metabolic condition characterized by hyperglycaemia [1]. In England, T2D costs the National Health Service approximately 10% of its annual budget [2] - a figure which has been predicted to rise to 17% by 2035/36 [3], making it a significant public health concern. Several factors affect T2D risk, with the two main modifiable risk factors being diet and physical activity (PA), which combined have been shown to reduce risk by up to 58% [4,5]. Many dietary factors affecting risk have been extensively studied, such as sugar-sweetened beverages (SSBs) [6]. One dietary factor that has not been comprehensively studied in relation to T2D is plain water intake.

The importance of water intake to maintain life and normal metabolic function is well established [7-9]. Nevertheless, water is often ignored in dietary recommendations [10] despite it being a simple and inexpensive dietary modification. Further to this, there are discrepancies regarding the amount of water reported to be needed per day for general health [7,8,11], meaning recommendations are difficult to make. This is due to many factors, such as some foods (for example, fruits and vegetables) having a high water content which has been shown to affect water intake [12]. Some research has found that plain water intake may aid weight loss (associated with reduced T2D risk), with the main proposed mechanism being its role in increasing feelings of satiety [11,13]. However, these studies focused on replacing caloric drinks with non-caloric drinks, thus do not necessarily show the independent effect of plain water intake.

Currently, only two studies have been identified that have directly investigated the relationship between T2D and plain water intake, and their results were conflicting. Pan *et al*. found that water intake was not significantly associated with T2D risk in 82,902 females from the Nurses' Health Study II [14]. Conversely, in the D.E.S.I.R. cohort of 3,615 participants, Roussel *et al.* found that participants who were drinking between 0.5-1 liters per day and those drinking >1 liter per day were at lowest risk of developing hyperglycemia over a 9-year period (by 36 and 27%, respectively), compared to those drinking <0.5 liters per day [15]. Further to these studies, de Koning *et al*. analysed data from the Health Professionals Follow Up study, focusing on the effects of sugar- and artificially-sweetened beverages on T2D risk. Within this analysis, water was associated with a significant 3% increase in risk of T2D, however the authors stated that these results were possibly due to residual confounding factors associated with T2D, as water intake was not the primary focus of their study [16]. Investigating the relationship between plain water intake and T2D risk is therefore important due to the paucity of research in this area along with the relative simplicity and cost-effectiveness of this dietary change.

The aim of the current exploratory study was to contribute to the limited evidence base by investigating the link between plain water intake and T2D risk in adults in the UK. We hypothesized that higher plain water intake would be associated with a lower T2D risk score. To test this hypothesis, the study objectives were to examine the association of plain water intake with T2D risk score, while taking into account key dietary and lifestyle factors associated with plain water intake.

## *2. Methods and materials*

### 2.1 Study design and sample

The study used cross-sectional data collected via an online survey tool. A convenience sample was used which targeted a selection of companies across London (*n*=2), the southeast (*n*=4) and the southwest (*n*=2) of England as well as students and staff at a major university in the southwest. The survey was disseminated via an email to participating companies, which gave details about the nature of the survey and a direct hyperlink. They were then asked to forward the survey to their employees/students. Exclusion criteria were: being <18 years old or having any type of diabetes or a known glucose disorder. No incentive was given to participate in the survey.

The study was conducted according to the guidelines laid down in the Declaration of Helsinki and all procedures involving human subjects/patients were approved by the University of Bristol Centre of Exercise, Nutrition and Health Sciences Ethics Committee. Informed consent was obtained from all participants prior to completing the survey, which terminated automatically if participants did not consent.

The research received no specific grant from any funding agency in the public, commercial or not-for-profit sectors.

## 2.2 Measurements

The survey comprised of four sections, assessing risk of T2D, PA, fruit and vegetable intake and beverage intake. The T2D risk score was calculated using the Diabetes UK risk assessment tool [17], which has been validated for use in the UK [18]. This measures seven key factors related to T2D risk: age, sex, body mass index (BMI), waist circumference, ethnicity, first degree family history of diabetes and antihypertensive medication use/history of hypertension. Each answer is scored with a set amount of points according to how strongly that factor is correlated with T2D risk, based on the response to an oral glucose tolerance test. These points are then summed to calculate a risk score of 0-47 points. From this score, participants are stratified into low (0-6 points), moderate (7-15 points), increased (16-24 points) and high (25-47 points) risk groups [17].

Physical activity was assessed using the short International Physical Activity Questionnaire (IPAQ) [19]. The IPAQ has been validated to assess low, moderate and high intensity PA levels via self-report [20]. Respondents are asked on how many days, and for how long, they engaged in specific activities of certain intensity. Each activity is assigned a set metabolic equivalent of task (MET) value, depending on its intensity, which is multiplied by the number

of minutes and number of days reported. The values for each type of activity are summed to give the total MET-minutes per week [19].

Dietary variables were assessed using a modified Cambridge European Prospective Investigation into Cancer and Nutrition Food Frequency Questionnaire (FFQ) [21]. The FFQ was modified to ask participants about their consumption trends over the last seven days only (as opposed to the last year). In terms of diet, only fruit and vegetables were measured due to their inclusion as the only dietary variable on Lindström and Tuemilehto's T2D risk inventory in Finland [22], as well as being a marker of a healthier lifestyle and influencing plain water intake. A list of 38 fruits and vegetables were presented and participants were asked to state their average intake over the last seven days ('never', 'once', '2-4 times', '5-6 times', 'once per day', '2-3 per day', '4-5 per day', or '6+ times per day'). As plain water was not included in the original FFQ, it was added to the survey in the same format used for other beverages. Plain water was measured in glasses and an information tab stated that one glass was equal to 240mL (standard measure). Participants were asked to state their average intake for a range of beverages (including, but not exclusive of, alcoholic, caffeinated, sugar-sweetened and artificially sweetened beverages) over the last seven days, with a range from 'never' to '6+ times per day' (as above). Thus, the FFQ led to the assessment of average daily plain water intake, average total beverage intake (sum of all beverages, including plain water) and average total fruit and vegetable intake (servings/day).

2.3 Statistical analyses

Statistical analyses were conducted using Statistical Package for Social Sciences version 19 (SPSS, IBM, Armonk, New York, USA), with the significance level defined as $P \leq 0{\cdot}05$ (two-tailed). Data are presented as means and standard error of the means (SEM), standard errors of difference (SED), or, where applicable, 95% confidence intervals (CI). There were three outcome variables: total beverages (sum of all beverages consumed per day on average), plain water intake (average plain water intake per day) and diabetes risk score, which were all assessed for normality. Non-normally distributed data with a positive skew were transformed using a Log10 transformation, and non-parametric tests were used if this did not correct the skewness. To establish significant differences in plain water intake between sexes, ethnic groups, those with and without a diabetic relative and those with and without hypertension, the Mann-Whitney U test was used, whilst an independent samples T-test was used for examining differences in total beverage intake between these different subgroups.

A Kruskal-Wallis test for non-normal data and a one-way ANOVA for normal data were used to establish significant differences in plain water intake and total beverage intake, respectively, between age groups, BMI categories, and waist circumference categories. Post-hoc pair-wise comparisons with Dunn-Bonferroni or Bonferroni correction were conducted on variables with significant differences for non-normally distributed and normally distributed data, respectively.

Pearson's correlation coefficient or Kendall's tau was used to examine correlations between T2D risk score and beverage intake on parametric and non-parametric data, respectively. Kendall's tau was favoured over Spearman's rho for non-normal data, as this test yields more accurate estimations of the value which would have been obtained from the population, thus it can draw more accurate generalizations [23].

In order to determine the relationship between T2D risk factors and plain water intake, two linear regression analyses were run. Bivariate analyses were conducted on all variables to find significant correlations for use in the regression analyses. The first regression had T2D risk score as the outcome variable with variables that had a significant correlation with T2D risk score as predictor variables, added in stepwise. The second linear regression analysis used water intake as the outcome variable with the seven characteristics of the T2D risk score used as predictor variables in step one and variables with significant correlations to water intake as predictor variables in step two.

*3. Results*

A total of 138 participants completed the online survey. Participant characteristics for the seven selected factors which contribute to the T2D risk score are presented in Table 1. Total beverage intake did not differ significantly between any participant characteristic groups. However, significantly more plain water was consumed among those aged <50 years, compared to 60-69 years ($P$=0·030), those with a waist circumference <90cm compared to

those reporting a waist circumference of both 90-99·9cm ($P$=0·035) and 100-109·9cm ($P$=0·023) (after Dunn-Bonferroni correction, the significance of these results diminished to $P$=0·211 and $P$=0·140, respectively), and those reporting hypertension, compared to those reporting normotension ($P$=0·003).

Plain water intake was negatively correlated with the T2D risk score ($\tau$=-0·180, $P$=0·005) (Table 1), confirming our hypothesis that higher plain water intake would be associated with a lower T2D risk score. Differences in water intake between risk stratified groupings failed to achieve significance ($H_{(3)}$=6·646, $P$=0·084) (Table 2). These differences in water intake between T2D risk score groups approached significance at the 0.05 level when moderate and high risk groups were collapsed to account for the small number of participants classified as high risk ($H_{(2)}$=5·920, $P$=0·052). Despite not reaching significance, there was a clear trend of lower water consumption in higher T2D risk score groups (Figure 1). Total beverage intake was not associated with the T2D risk score (r=0·024, $P$=0·780), and there were no significant differences in total beverage intake between risk groups ($F_{(3)}$=0·302, $P$=0·824) (Table 2).

The first linear regression had T2D risk score as the outcome variable and caffeinated beverage intake (model 1), fruit juice intake (model 2), full-fat milk intake (model 3) and plain water intake (model 4) as the predictor variables, as these were found to be significantly correlated with the T2D risk score. In the final model, the explanation of variance in T2D risk score was 16·9% ($P$<0·001). The analysis showed that each cup (240mL) of plain water drunk per day was associated with a significant 0·72 points (range 0-47 points) decrease in T2D risk score (B=-0·003, 95% CI=-0·006, -0·001, $P$=0·014) (Table 3).

The second regression analysis (with plain water intake as the outcome variable) showed that only combined fruit and vegetable consumption significantly predicted water intake; an increase of one fruit or vegetable per day was associated with an increase in plain water intake of nearly 22mL (B=21·928, 95% CI=0·672, 43·183, *P*=0·043) (Table 4). Bivariate correlations between all variables showed that fruit and vegetable intake was negatively correlated with SSBs (r=-0·173, *P*=0·043) and was positively correlated with PA (r=0·388, *P*<0·001).

*4. Discussion*

The aim of this exploratory study was to investigate the association between plain water intake and T2D risk status. The results showed that the mean differences in plain water intake between diabetes risk groups were not significant, but there was a significant negative correlation between plain water intake and T2D risk score; an increased risk score was associated with reduced plain water intake, thus confirming our hypothesis. In addition, regression analyses showed that none of the participant characteristics which determined the T2D risk score explained any variance on plain water intake. Nevertheless, plain water intake was a significant predictor of the overall T2D risk score.

Despite variances in plain water intake between T2D risk score categories not being significantly different, it is worth noting that once the moderate and high risk groups were collapsed to reduce the effect of a small sample size, the differences approached significance (*P*=0·052). This could suggest that a larger sample size may have resulted in plain water intake in participants of different risk score categories being significantly different. The significant correlation found supports this hypothesis, as it showed that participants with lower risk scores had higher intakes of plain water.

To some degree, the results of the current study support the findings of Roussel *et al.* who found that compared to those drinking <0.5 liters per day, a lower risk of hyperglycemia was found in those consuming 0.5-1 liters per day (odds ratio = 0·68, 95% CI=0·52, 0·89) and >1 liter per day (odds ratio = 0·79, 95% CI=0·59, 1·05) (*P* for difference=0·016) [15]. Conversely, Pan *et al*. found that water intake was not associated with T2D risk (relative risk for one glass per day = 0·93, 95% CI=0·82, 1·05 compared to relative risk for ≥six glasses per day = 1·06, 95% CI=0·91, 1·23, $P_{trend}$=0·15) in 82,902 female participants [14] - a much larger sample than Roussel *et al*. (n=3,615) [15] and the current study (n=138). However, as the sample recruited by Pan *et al*. was exclusively female, this could suggest a potential gender effect, partially explaining the conflicting results. Further to this, Pan *et al*. suggested that BMI was a confounding factor, which may have led to reverse causality, despite the longitudinal design of the study (as the primary outcome measure was T2D incidence, rather than BMI) [14].

In the current study, plain water intake had a significant correlation with diabetes risk score, suggesting it may have a role in the development or prevention of T2D, as each cup of water consumed per day reduced the T2D risk score by 0·72 points. Considering the mean participant intake of 567mL plain water per day, this would equal a 1·7 point reduction in T2D risk score, which could have significant health benefits at population level. Kant, Graubard and Atchison found that US adults consumed a mean intake of 1049mL of plain water per day [12], meaning the average risk score reduction would equal 3·2 points, which would have greater impact on both individual and public health.

Of interest was the significantly lower intake of plain water among those reporting hypertension or taking antihypertensive medication compared to normotensive participants. Hypertension can be regulated through hydration status by altering blood volume, hence diuretics are a commonly prescribed medication [14]. The lower plain water intake in this group could be due to the diuretic effects of medication and patients wishing to avoid excessive urination with increased water intake. Alternatively, reduced plain water intake could be attributed to a generally unhealthier lifestyle in this group, which may be the reason for hypertension (for example, in this study, fruit and vegetable consumption was positively associated with increased plain water intake). Nonetheless, this finding provides support for the mechanism proposed by Roussel *et al*., which stated that hydration may affect T2D risk by affecting the secretion of arginine vasopressin [15]. Although this anti-diuretic hormone is traditionally associated with blood pressure regulation, it has also been shown to affect glucose homeostasis and insulin resistance [24-26].

The link between arginine vasopressin, hydration status and glucose intolerance has recently been demonstrated in rats; elevated arginine vasopressin levels led to higher fasting glycemia in lean rats and hyperinsulinemia and glucose intolerance in obese rats [27]. Additionally, low arginine vasopressin was induced by increasing some rats' plain water intake. These rats had lower fasting glycemia than normal or higher arginine vasopressin rats. Although this link needs to be further explored in humans, this study provides support for the findings of the current study and the mechanisms proposed by Roussel *et al.*

Earlier research studies have proposed that replacing high energy beverages with plain water can aid weight loss [11,13]. This suggests that plain water may reduce T2D risk by decreasing adiposity (a known risk factor for the development of T2D), possibly through increasing satiety and subsequently reducing energy intake [28]. Thus, the possibility exists that the weight loss reported in these studies may have been due to a reduction in energy intake rather than the addition of water in the diet. Nonetheless, Burge *et al*. found that dehydration increased plasma concentrations of glucose in participants with type 1 diabetes [29]. This further provides support for the hypothesis that water directly affects glucose homeostasis, though this may only be applicable to those with type I diabetes.

In the current study, only combined fruit and vegetable intake had significant explanatory power for predicting plain water intake, further supporting the theory that plain water intake could be an indicator of a healthier lifestyle. Additionally, those who consuming more plain water also consumed more fruit and vegetables, less SSBs and were more physically active. These findings suggest that water could be a mediating factor against T2D. However, some

have proposed more direct mechanisms, such as the role of water in cell metabolism [30], which if disrupted may lead to insulin resistance [31], along with its roles in nutrient transportation and hydrolysis [10]. Although further research needs to be conducted, it is fair to conclude that dehydration could negatively impact these functions, thus increasing T2D risk; a mechanism which supports the findings of the first regression analysis in this study.

Due to the limited research in this field, it is also possible that a suitable mechanism has not yet been established and other theories may offer viable explanations. For example, high glycaemic load diets have been associated with increased risk of T2D [6]; as water has no calories, its consumption with meals may reduce the glycaemic load of food, thus dampening the postprandial glycaemic response, leading to reduced risk of T2D. Another theory is that the increase in blood volume caused by better hydration [32] reduces the glucose concentration in the blood, thus reducing the subsequent insulin response, which would decrease the risk of T2D. This theory is supported to some extent by Burge *et al*. [29], however further research needs to be conducted to establish the validity of these mechanisms independently and cumulatively.

A key limitation of this study was its cross-sectional design, which did not allow for the temporal direction of trends to be established, thus leaving the potential for reverse causality. However, a common symptom of glucose disorders is thirst, therefore the results found may rule out to some extent reverse causality as those with a higher risk score consumed less (rather than more) water (with no differences in total beverage intake). Furthermore, only correlations rather than causation can be inferred. The online survey was based on participant

recall, which might have led to errors due to poor memory, bias, and social desirability of responses in recalling PA and eating and drinking behaviours. Social desirability of responses may also be an issue when considering factors such as body weight and unhealthy behaviours, such as the amount of time spent in sedentary activities. However, the anonymity provided by the online survey may have minimized this effect. To improve reliability, validated measures with as few modifications as possible were used, as well as offering email support to clarify any participant queries.

The study was conducted without funding support and so was limited in the measurement instruments that could be employed, thus was consequently reliant on self-reported data. The short version of the IPAQ was used, which may not deliver the level of detail and accuracy for PA assessment as the full version, however, it does reduce participant burden as it is not as time consuming to complete. For food intake assessment, only fruit and vegetables were enquired about. This meant there may have been other interactions that were not accounted for. Further to this, PA and diet were only assessed for the previous seven days, which for some participants may not have been a representative week. There is currently no standard measure used for collecting data regarding plain water intake; in this study it was added into the beverages FFQ in the same format as the other beverages, so its validity can only be inferred.

Lastly, the sample of 138 participants may not be representative of the wider population, reducing the generalizability of the results. Lack of representativeness was minimized to some degree by recruiting participants from different areas. Due to the method of data

collection, we cannot ascertain the response rate, meaning more health conscious people may have responded more readily. This can to some extent be demonstrated by most participants (n=77) having a low risk score, with only 23 participants having moderate to high risk scores. Additionally, the sample largely consisted of those aged <50 years (n=102), which may not be representative of a high risk population.

Nevertheless, this study makes an important contribution to the limited literature on plain water intake trends and their role in T2D risk, which to our knowledge has not been previously investigated in the UK. The findings are valuable because increasing water intake could be an easy to understand, inexpensive and accessible dietary modification to help reduce the risk of T2D. This study also focused on T2D risk, rather than prevalence, as prevention is more cost-effective and efficacious than treating existing disease [18]. The broad research questions allowed the exploration of many interactions and a more specific hypothesis to be generated. For example, this study showed a link between hypertension, T2D risk score and plain water intake, which future research could explore to examine whether these were causative. This design also allowed for potential mediating variables to be explored, which can help provide theory for future research.

Furthermore, the results have shown support for two key theories which have been previously proposed by research [14, 15]. Firstly, the role of hydration status on arginine vasopressin which directly affects glucose homeostasis and secondly, the role of plain water intake as a marker of a healthier lifestyle. Other theories have been suggested, which may also help to explain the results: that water reduces the glycaemic load of food, and that hydration

increases blood volume, thus reducing blood glucose concentration. These suggest that the relationship found is less likely to be an artefact of the research, as there are multiple potential explanatory theories available to explain the findings. As this was exploratory work, these theories need to be researched further to identify any causal mechanisms.

Overall, this research showed that plain water intake was significantly and negatively associated with T2D risk, with every 240mL cup of water consumed per day reducing risk by 0·72 points. Fruit and vegetable intake also predicted plain water intake. It is not clear whether the relationship between water intake and the T2D risk score was a direct effect of plain water intake or whether plain water intake, as a potential marker of a generally healthier lifestyle (such as increased fruit and vegetable intake), might play a role in T2D risk. Nonetheless, these findings provide some interesting relationships with multiple proposed explanatory theories worthy of future investigation.

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Conflicts of interest: None.

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**Table 1: Participant characteristics according to their daily total beverage intake (mL/d) and daily plain water intake (mL/d)**

| | n | Total beverages (mL/d) | Plain water (mL/d) |
|---|---|---|---|
| **Total** | 138 | 2078 ± 72 | 567 ± 41 |
| **Males** | 60 | 2176 ± 103 | 544 ± 65 |
| **Females** | 78 | 2003 ± 100 | 585 ± 52 |
| **Age <50 years** | 102 | 2022 ± 86 | 645 ± 49 |
| **Age 50-59 years** | 27 | 2389 ± 151 | 413 ± 66 |
| **Age 60-69 years** | 6 | 1917 ± 246 | 123 ± 34 |
| **Age >69 years** | 3 | 1510 ± 202 | 200 ± 200**† |
| **Underweight[b]** | 2 | 3006 ± 474 | 557 ± 523 |
| **Normal weight[b]** | 79 | 1999 ± 94 | 613 ± 55 |
| **Overweight[b]** | 39 | 2199 ± 122 | 512 ± 71 |
| **Obese[b]** | 18 | 2064 ± 250 | 489 ± 112 |
| **White European** | 127 | 2065 ± 73 | 560 ± 43 |
| **Other ethnicity** | 11 | 2236 ± 335 | 653 ± 122 |
| **Waist <90cm** | 86 | 2088 ± 89 | 641 ± 52 |
| **Waist 90-99.9cm** | 31 | 2085 ± 130 | 461 ± 86 |
| **Waist 100-109.9cm** | 16 | 1917 ± 293 | 336 ± 76 |
| **Waist >110cm** | 5 | 2388 ± 394 | 693 ± 252*‡ |
| **Diabetic relative** | 34 | 2023 ± 148 | 487 ± 68 |
| **No diabetic relative** | 104 | 2096 ± 83 | 593 ± 49 |
| **Taking blood pressure medicine** | 13 | 1789 ± 157 | 223 ± 64 |
| **Not taking blood pressure medicine** | 125 | 2108 ± 78 | 603 ± 43** |

Values are means ± SEM

Differences in total beverages intake were assessed using an independent samples T-test or an ANOVA

Differences in total plain water intake were assessed using the Mann Whitney U test or the Kruskal Wallis test.

[b] BMI categories: underweight <18.5kg/m$^2$; normal weight=18.5-24.9kg/m$^2$; overweight=25-29.9kg/m$^2$; obese >30kg/m$^2$

* $P$<0.05

** $P$<0.01

† Post hoc comparison found significant differences in plain water intake between age <50 compared to age 60-69 ($P$=0.030 after adjusting with Dunn-Bonferroni correction)

‡ Post hoc comparison found significant differences in plain water intake between <90cm compared to 90-99.9cm ($P$=0.035) and between <90cm compared to 100-109.9cm ($P$=0.023), however, these significant differences diminished after Dunn-Bonferroni correction ($P$=0.211 and $P$=0.140, respectively)

**Table 2: Differences and associations between total daily beverage intake (mL/d) and daily plain water intake (mL/d) between diabetes risk categories**

| | n | Total beverages (mL/d) | Total plain water (mL/d) |
|---|---|---|---|
| **Low risk** | 77 | 2061 ± 96 | 652 ± 57 |
| **Increased risk** | 38 | 2160 ± 137 | 500 ± 72 |
| **Moderate risk** | 18 | 1947 ± 190[†] | 427 ± 97[‡] |
| **High risk** | 5 | 2200 ± 574[†] | 278 ± 136[‡] |
| **Diabetes risk score correlation** | 138 | Pearsons r = 0.024 | Kendall's τ = -0.180[*] |

Values are means ± SED
Differences in total beverage intake were assessed using an ANOVA.
Differences in total plain water intake were assessed using the Kruskal Wallis test.
The diabetes risk score correlation with total beverages was assessed using Pearson's correlation coefficient, and the correlation with total plain water intake was assessed using Kendall's tau.
[†] Mean beverage intake (mL/d) for moderate and high risk groups collapsed (n=23) = 2002 ± 188.221, *P*=0.754.
[‡] Mean plain water intake (mL/d) for moderate and high risk groups collapsed (n=23) = 394 ± 81.498, *P*=0.052.

**Table 3: Associations between intake of beverages (mL/d) and diabetes risk score**

| | Model 1[a] | | | Model 2[b] | | | Model 3[c] | | | Model 4[d] | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | ***B*** | **95% CI** | **β** | ***P*** | ***B*** | **95% CI** | **β** | ***P*** | ***B*** | **95% CI** | **β** | ***P*** | ***B*** | **95% CI** | **β** | ***P*** |
| **Constant** | 6.6126** | 4.064, 8.188 | | <0.001 | 6.777** | 4.496, 9.058 | | <0.001 | 5.827** | 3.630, 8.023 | | <0.001 | 8.137** | 5.393, 11.241 | | <0.001 |
| **Caffeinated beverage intake** | 0.003* | 0.000, 0.005 | 0.177 | 0.038 | 0.002* | 0.000, 0.005 | 0.170 | 0.046 | 0.003 | 0.000, 0.005 | 0.181 | 0.024 | 0.002 | -0.001, 0.004 | 0.114 | 0.168 |
| **Fruit juice intake** | | | | | -0.005 | -0.013, 0.003 | -0.110 | 0.193 | -0.004 | -0.012, 0.003 | -0.094 | 0.240 | -0.004 | -0.011, 0.004 | -0.076 | 0.335 |
| **Full-fat milk intake** | | | | | | | | | 0.031** | 0.017, 0.046 | 0.336 | <0.001 | 0.031** | 0.016, 0.045 | 0.330 | <0.001 |
| **Plain water intake** | | | | | | | | | | | | | -0.003* | -0.006, -0.001 | -0.207 | 0.014 |
| **Adjusted $R^2$** | 0.024* (*P*=0.024) | | | | 0.029 (*P*=0.050) | | | | 0.137* (*P*<0.001) | | | | 0.169* (*P*<0.001) | | | |

Data presented are based on linear regression analyses with diabetes risk score as the dependent variable and different beverages as the predictor variables.

*B* = unstandardised beta coefficient; β = standardised beta coefficient

[a] Model 1: Adjusted for caffeinated beverage intake

[b] Model 2: Adjusted for (as Model 1+) fruit juice intake

[c] Model 3: Adjusted for (as Model 2+) full-fat milk intake

[d] Model 4: Adjusted for (as Model 3+) plain water intake

*$P$<0.05

**$P$<0.001

**Table 4: Associations between diabetes risk score components, intake of beverages and fruits and vegetables, and daily plain water intake (mL/d)**

| | Model 1[a] | | | | Model 2[b] | | | |
|---|---|---|---|---|---|---|---|---|
| | ***B*** | **95% CI** | **β** | ***P*** | ***B*** | **95% CI** | **β** | ***P*** |
| **Constant** | 135.301 | -1072.490, 1342.093 | | 0.825 | -46.531 | -1193.385, 1100.324 | | 0.936 |
| **Gender[c]** | 10.803 | -182.514, 160.908 | -0.011 | 0.901 | -40.841 | -202.717, 121.036 | -0.043 | 0.618 |
| **Age ≥70 years[d]** | -171.029 | -870.864, 528.806 | -0.053 | 0.629 | -112.007 | -779.652, 555.639 | -0.034 | 0.740 |
| **Age 60-69 years[d]** | -429.258 | -953.519, 95.002 | -0.184 | 0.108 | -366.656 | -860.098, 126.786 | -0.158 | 0.144 |
| **Age 50-59 years[d]** | -168.577 | -388.757, 51.602 | -0.141 | 0.132 | -116.650 | 340.762, 107.461 | -0.098 | 0.305 |
| **Ethnicity[e]** | 117.816 | -193.732, 429.364 | 0.067 | 0.456 | 135.319 | -166.341, 436.979 | 0.077 | 0.376 |
| **Diabetic relative[f]** | 94.251 | -106.272, 294.775 | 0.086 | 0.354 | 125.530 | -12.063, 313.122 | 0.114 | 0.188 |
| **Waist 90-99cm[g]** | -98.897 | -342.549, 144.755 | -0.087 | 0.423 | -118.519 | -347.543, 110.506 | -0.104 | 0.308 |
| **Waist 100-109cm[g]** | -235.613 | -558.148, 86.921 | -0.159 | 0.151 | -223.867 | -539.405, 91.671 | -0.151 | 0.163 |
| **Waist ≥110cm[g]** | 47.599 | -431.009, 526.207 | 0.019 | 0.844 | 17.678 | -451.078, 486.433 | -0.007 | 0.941 |
| **BMI[h]** | 4.178 | -20.223, 28.579 | 0.039 | 0.735 | 12.923 | -10.766, 36.612 | 0.121 | 0.282 |
| **Hypertensive[i]** | 81.200 | -326.968, 489.369 | 0.050 | 0.694 | 100.264 | -282.878, 483.407 | 0.062 | 0.605 |
| **Caffeinated beverage intake** | | | | | -0.013 | -0.314, 0.288 | -0.015 | 0.932 |
| **Fruit juice intake** | | | | | 0.176 | -0.431, 0.782 | 0.063 | 0.567 |
| **Semi-skimmed milk intake** | | | | | -0.273 | -0.711, 0.165 | -0.137 | 0.220 |
| **Non caloric drink intake** | | | | | -0.210 | -0.479, 0.059 | -0.250 | 0.125 |
| **Caloric drink intake** | | | | | -0.061 | -0.392, 0.270 | -0.050 | 0.716 |
| **Combined fruit and vegetable intake** | | | | | 21.928* | 0.672, 43.183 | 0.173 | 0.043 |
| **Adjusted $R^2$** | 0.047 ( *P*=0.102) | | | | 0.185* (*P*=0.001) | | | |

Data presented are based on linear regression analyses with daily water intake as the dependent variable and diabetes risk score components, different beverages and fruit and vegetable intake as the predictor variables.

*B* = unstandardised beta coefficient; β = standardised beta coefficient

[a] Model 1: Adjusted for gender, age, ethnicity, first degree relative with diabetes, waist circumference, BMI and history of hypertension/use of antihypertensive medication

[b] Model 2: Adjusted for (as Model 1+) intake of caffeinated beverages, fruit juice, semi-skimmed milk intake, non-caloric drinks intake, caloric drinks intake and combined total fruit and vegetable intake

[c] Males (baseline) compared to females
[d] All age groups are compared to age <50 years as baseline
[e] White European (baseline) compared to other ethnic groups
[f] First degree family relative with diabetes (baseline) compared to no first degree family member with diabetes
[g] All waist circumference categories are compared to waist circumference <90cm as baseline
[h] BMI = $kg/m^2$
[i] History of hypertension/use of hypertensive medication compared to normotensive
[*] $P<0.05$

## Figure legends:

**Figure 1: The relationship between mean plain water intake (mL/d) and T2D risk score categories**

## Footnote to Figure 1:

Diabetes risk score categories: Low risk (0-6 points) n=77, increased risk (7-15 points) n=38, moderate + high risk (>15 points) n=23
Error bars: 95% CI

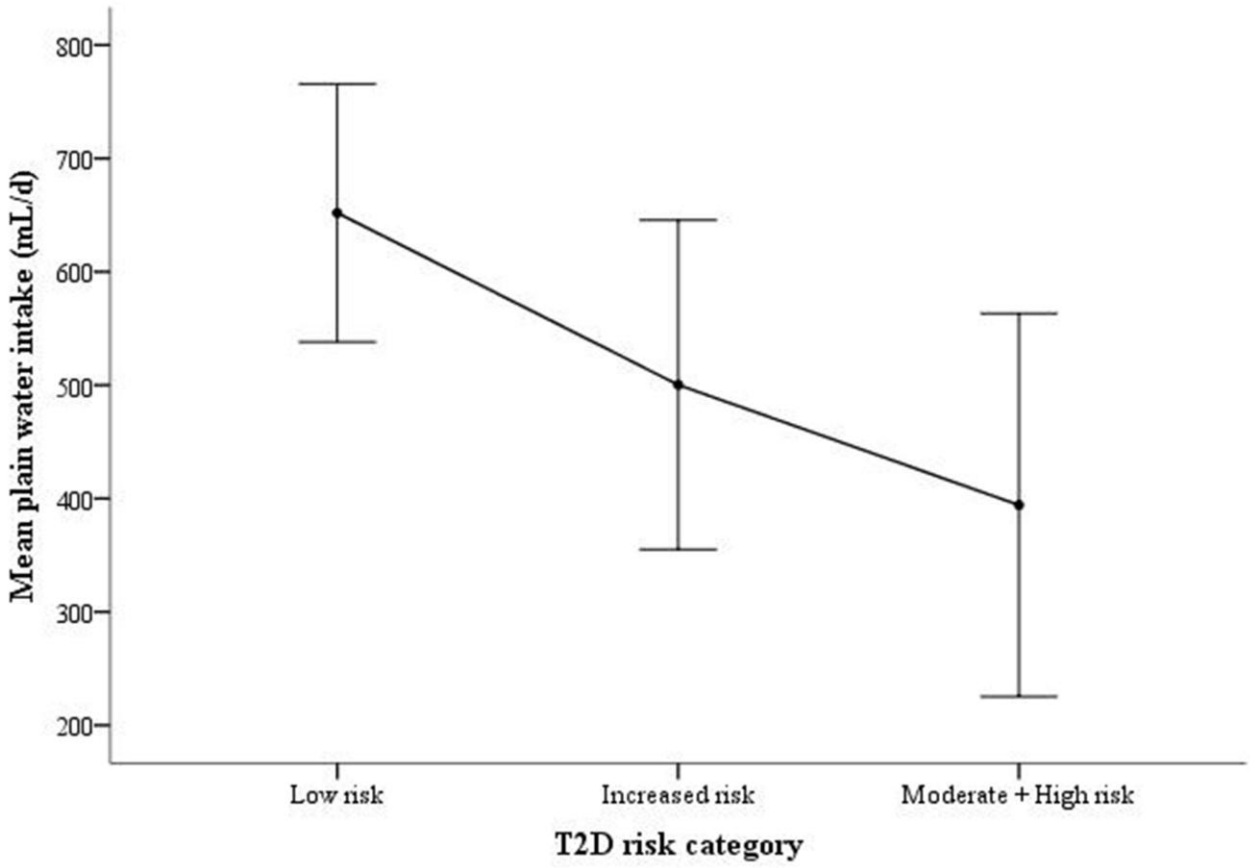


Figure 1