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# Comparison of risk factors between people with type 2 diabetes and matched controls in Nairobi, Kenya

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**Abstract**

OBJECTIVE To identify risk factors associated with type 2 diabetes (T2D) in Nairobi, Kenya.
METHODS A case–control study comparing 70 (53% women) recently diagnosed T2D cases with age-, sex- and socioeconomic status-matched normoglycemic controls (1:1). Objectively measured data were obtained on anthropometrics, handgrip strength and physical activity (by accelerometer). Self-reported data were collected on demographic characteristics and lifestyle factors. Logistic regression models, adjusted for covariates, were used to analyse the data.
RESULTS Each standard deviation (SD) increase in height was associated with lower odds for T2D (adjusted odds ratio (AOR) = 0.34 (95% confidence intervals [CIs] 0.17, 0.66), $P = 0.0031$). Fat-free mass was inversely associated with T2D (AOR = 0.42 (95% CI 0.24, 0.75), $P = 0.0032$, per SD increase). Grip strength was associated with a lower risk of T2D (AOR = 0.20 (95% CI 0.08, 0.45), $P < 0.001$, per SD increase). BMI was not associated with T2D, but higher waist-to-hip ratio was associated higher odds of T2D (AOR = 2.28 (95% CI 1.38, 3.79), $P = 0.0014$, per SD increase). Physical activity was not associated with T2D. Cases reported higher intakes of fruits and vegetables and a lower intake of sugar than controls.
CONCLUSIONS Central obesity, rather than BMI, may have more utility for T2D risk stratification in Kenya, and interventions that increase muscle mass and strength, as well as support weight loss, may be useful for T2D prevention in this and other SSA populations. However, more evidence is needed to determine whether low muscle mass, strength and height are causally related to T2D risk and/or are indicators of adverse early-life environment.

**keywords** case–control study, Kenya, risk factors, Type 2 diabetes mellitus

**Sustainable Development Goal**: Good Health and Well-being

## Introduction

Diabetes is one of the top four non-communicable diseases (NCDs) globally, with the burden increasingly falling on low and middle-income countries [1, 2]. The International Diabetes Federation projects that diabetes prevalence in sub-Saharan Africa (SSA) will increase from 19 million cases in 2019 to 47 million by 2040 [2]. Over 90% of people with diabetes have type 2 diabetes (T2D), which despite having a number of non-modifiable risk factors (including age, family history and ethnicity) [3, 4] has been shown to be preventable through lifestyle modification (dietary changes, weight loss, increased physical activity) [5]. There is also emerging evidence that other factors, including muscular strength [6], early-life environment [7] and sleep [8], can influence T2D risk.

Kibirige *et al* suggest that T2D has a distinct phenotype in SSA characterised by impaired insulin secretion, development at an early age and occurrence at a low body mass index (BMI) [9]. Such a phenotype emphasises the role of metabolic health rather than solely high BMI in T2D development in the region. A metabolic unhealthy normal weight profile has been associated with increased central obesity, reduced cardiorespiratory fitness, increased insulin resistance and reduced insulin secretion capacity which consequently increases T2D risk [10]. Further, although low muscle strength has been shown to be associated with risk of adverse metabolic health

outcomes mainly in high-income countries [11], there is limited evidence of this from SSA. There is therefore a need for detailed evidence to better characterise the T2D phenotype in SSA [9].

In Kenya, the National Strategy for the Prevention and Control of Noncommunicable Diseases 2015–2020 recognises T2D as one of the main NCDs and affirms the country's commitment to its prevention [12]. However, although Kenyan diabetes policies are well aligned to international recommendations, such as the Global Action Plan for the Prevention and Control of NCDs, they are based on limited local evidence [13]. This presents a challenge in development of T2D prevention interventions that are effective within the local context. The few Kenyan studies that have been conducted have reported associations between T2D and obesity [14–17], age [14–16], hypertension [14–16], physical inactivity [16], alcohol intake [16], tobacco use [16], family history [17], handgrip strength [18] and childhood starvation [17]. While most of these studies have explored the common T2D risk factors, other emerging risk factors such as muscle mass and strength, height and childhood starvation have been considered in relatively few investigations. The current study therefore was designed to add to the limited evidence on the phenotype and risk factor profile of T2D in Kenya by comparing people with recently diagnosed T2D to age-, sex- and socioeconomic status-matched normoglycemic controls in Nairobi. Specifically, we aimed to test five hypotheses: (1) T2D is positively associated with adiposity (anthropometric measures, fat mass); (2) T2D is inversely associated with muscle mass and strength; (3) T2D is positively associated with childhood starvation and low adult height; (4) T2D is positively associated with family history of diabetes; and (5) T2D is positively associated with unhealthy lifestyle factors (e.g. low fruit and vegetable intake, short sleep duration, physical inactivity, smoking and alcohol intake).

## Methods

### Study design, site and sampling

A matched case–control study with people who were recently diagnosed with T2D (cases) and normoglycemic controls (controls) was conducted in Nairobi city county, an urban setting in Kenya. Cases were recruited from the Kenyatta National Hospital and Ngaira Health Centre diabetes clinics, and referral from community health volunteers (CHVs) and local diabetes support groups. Matching (1:1) was done on sex, age (interval matching, ± 5 years) and socioeconomic status (education and wealth level). Controls were recruited by asking patients to come with a friend of the same sex and age group, and where this was not possible, recruited by CHVs from similar residential areas to the cases. These approaches aimed to ensure matching of socioeconomic status; for example, it was unlikely a case with no formal education and in the lowest wealth level would come with a control with tertiary level education or with the highest wealth level. Age was computed using self-reported year of birth. To determine the matching effectiveness of these approaches, we tested whether there were differences between cases and controls in the matching variables.

All data were collected at Ngaira Health Centre, a public health facility in Nairobi, between October 2019 and March 2020. The participant flow is shown in Figure 1. We included 140 participants (70 cases (37 women, 33 men), 70 controls (37 women, 33 men)) in the analysis. This sample size had 80% power (1-β) to detect associations between potential exposures (with a prevalence of ≥30% in the general population) and T2D with an odds ratio of ≥2.7 at a 5% significance level (α) [19].

### Case and control definition, inclusion and exclusion criteria

A case was defined as a person who self-reported diabetes diagnosis by a health worker in the last two years. In Kenya, diabetes is diagnosed using a single a fasting blood glucose of ≥7 mmol/l or a random blood glucose of ≥11 mmol/l in symptomatic people, and at least two abnormal blood glucose results on separate occasions in asymptomatic people [20]. A control was defined as a person with normoglycemic status, which was confirmed using a blood glucose test. Participants in both groups were included if they had lived in Nairobi for ≥5 years (to capture permanent residents rather than visitors) and did not have any self-reported or known health conditions that would have an effect on cardiometabolic health, such as HIV, cancer, stroke and heart diseases. We included participants aged 35–64 years, as diabetes prevalence is highest in this age group in Kenya [15] and any incident cases are unlikely to be type 1 diabetes [21]. Participants with T2D were excluded if they self-reported any microvascular chronic complications since diagnosis such as stroke, heart disease and diabetic foot ulcers [22]. In an instance where a case referred a control who was not a match (e.g. >5 years older), they were requested to refer another control who would be a match hence the recruitment of more controls ($n$ = 97) than cases ($n$ = 82). Figure 1 shows the inclusion and exclusion of cases and controls.

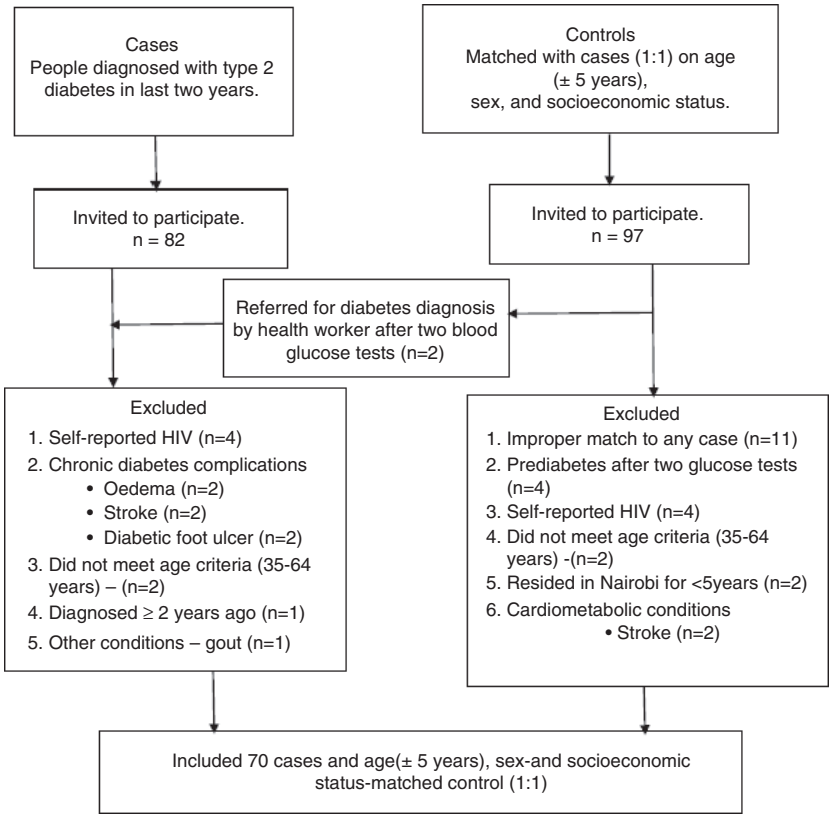


**Figure 1** Participation flow chart describing inclusion and exclusion of cases and controls.

### Data collection procedures and measurements

A questionnaire was administered to participants by AMM or by one of four research assistants (trained and supervised by AMM) who helped with data collection. The questions administered were adapted from validated tools used in other Kenyan studies [17, 23, 24] and translated and administered in Swahili. They assessed the sociodemographic characteristics (e.g. age, sex, education level, tribe, employment status), childhood starvation, medical history (diagnosed chronic diseases and family history of diabetes), current dietary intake, tobacco and alcohol use, self-reported physical activity, and sleep duration. Wealth index was computed using a principal components analysis commonly used in demographic and health surveys [25]. Alcohol and tobacco use was categorised as current user, former user or never used. Data on frequency of adding sugar to beverages and intake of highly processed food high in sugar were converted from a Likert scale measure to a dummy variable, rarely (0) representing *sometimes, rarely,* and *never* responses, while often (1) represented *always* and *often* responses.

Blood pressure measurements were taken at one minute intervals on the left upper arm using the Omron 705IT monitor (HEM-759-E, Omron Corporation, Kyoto, Japan), with arms relaxed on an armrest chair [26]. Two readings were taken if the systolic and diastolic measures were ≤120 and ≤80, respectively, and a third reading taken if either measure was above the cut-off. The average value of the last two readings was used in the analysis [26]. Hypertension was defined as a systolic blood pressure (SBP) ≥ 140 mmHg, or a diastolic blood pressure (DBP) ≥ 90 mmHg [27], or a previous diagnosis by a health worker.

Height was measured to the nearest 0.1cm using a Leicester Height Measure stadiometer. Body composition and weight were measured using a Tanita TBF 300A body composition analyser after participants had taken off heavy clothing. The analyser used bio-impedance to estimate percentage body fat, fat mass and fat-free mass, and calculated BMI in kg/m$^2$. Waist and hip circumferences were taken over light clothing using a SECA 201 tape measure at the umbilicus level and around the

 

widest portion of the buttocks, respectively [23, 28]. A third measure was taken if the first two were >0.7cm apart [29].

Handgrip strength was assessed using a Jamar 5030J1 hand hydraulic dynamometer using the Southampton protocol [30]. Time of last food/drink intake was recorded, and blood glucose level measured in mmol/l using a finger-pick and an Accu-Chek™ Active glucometer. Controls who had a glucose level of ≥7 mmol/l with less than eight hours of fasting were asked to come back for another test while fasted in the morning. Prediabetes was defined as an impaired fasting blood glucose of 6.1–6.9 mmol/l [15, 31] —controls with prediabetes were excluded (Figure 1). Those with a fasting blood glucose of 7 mmol/l and above were referred to their nearest health facility for possible diabetes diagnosis by a health worker. On confirmation of diagnosis, they were included as cases (Figure 1).

Actigraph GTX3 accelerometers (Actigraph, Pensacola, FL, USA) were issued for participants to wear for seven consecutive days, except when showering or sleeping. Time spent in different intensities of physical activity were determined using the Troiano *et al* (2008) algorithm [32]. A valid accelerometery week was defined as at least 10 hours of wear time for three days [33–35]. Three days of wear time has been shown to provide physical activity values within 10% of a complete seven-day measure [36]. To provide information on the contexts in which physical activities occurred, the Global Physical Activity Questionnaire (GPAQ) was used [24]. Metabolic equivalent (MET) minutes of physical activity were calculated from GPAQ data based on values from the WHO STEPS Surveillance Manual [37].

All glucose and handgrip strength measurements were taken by AMM, while blood pressure, body composition and anthropometrics were taken by AMM assisted by one research assistant or research assistants under the supervision of AMM. The questionnaire and all measurements were pilot tested by the research team before data collection.

### Ethical considerations

Ethical approval was provided by the Great Lakes University of Kisumu Research Ethics Committee (ID GREC 023/19), the Kenyatta National Hospital – University of Nairobi Ethics and Research Committee (P383/05/2019) and the University of Glasgow Medicine, Veterinary & Life Sciences College Ethics Committee (project 200180144). Further, the conduct of the research was approved by the National Commission for Science, Technology, and Innovation (NACOSTI/P/19/42210/29956), the Nairobi City County Health Office and the Ministry of Education. Written informed consent was sought from all participants before data collection.

### Data analysis

All data analyses were performed using R statistical package version 3.5.2 [38] and ActiLife software version 6.13.4 (www.actigraphcorp.com/actilife/) for accelerometery data. Descriptive characteristics of all variables, disaggregated by sex, were presented in means (and standard deviations) for continuous variables and frequencies (and percentages) for categorical variables. The analysis of variance (ANOVA) test and Fisher's exact test were used to assess significant differences between cases and controls on sociodemographic characteristics. Associations between exposures and T2D were assessed using multivariable logistic regression models and odds ratios (with 95% confidence intervals). Unconditional regression models (using *glm* function) were used, as our data were loose-matched on few sociodemographic variables [39]. Three logistic models, two adjusted for confounders, were used on all potential risk factors. Model 1 was unadjusted, while model 2 was adjusted for age, sex, education and wealth index (since matching does not eliminate confounding [40]). Model 3 was adjusted for variables in model 2 and further adjusted for waist-to-hip ratio (WHR), childhood starvation, smoking status, alcohol intake status, family history of diabetes, sleep duration, fruit and vegetable intake, hypertension and MET for self-reported physical activity. Adjustment was not done (e.g. for sleep duration), when the variable was the main independent variable (e.g. sleep duration). WHR was used to adjust for obesity, as it was strongly associated with T2D in our study compared with other obesity measures. Continuous and categorical covariates were used in the models as continuous variables. All continuous predictor variables were standardised in the models to determine effect of one standard deviation increase on the odds of T2D. Statistical significance was set at $P < 0.05$. $P$ values were not adjusted for multiple comparisons as effect sizes (standard deviations) and 95% confidence intervals of odds ratios were reported, and these provide important information on magnitude of associations without sole reliance on $P$ values [41].

## Results

### Sociodemographic characteristics and glucose levels

Table 1 summarises sociodemographic characteristics, time since diagnosis (for cases) and glucose levels of participants disaggregated by sex and study groups. Matched

characteristics (age, sex and socioeconomic status) were similar between cases and controls. Cases had higher glucose levels, and time since diagnosis was 15 months in women and 13 months in men.

### Body composition, obesity, handgrip and sleep duration

As presented in Table 2, each standard deviation increase in height, fat-free mass and grip strength was associated with lower odds of T2D. There were no significant associations between BMI, fat mass, waist circumference and T2D. However, each standard deviation increase in WHR was associated with an over two times higher odds of T2D. There was no significant association between sleep duration and T2D.

### Dietary practices

Table 3 shows that a higher proportion of cases had been advised by a health worker to change their diet. Consequently, each standard deviation increase in meals bought from venders per week was associated with a lower odds of T2D, while a standard deviation increase in daily fruits and vegetables intake was associated with a higher odds of T2D. Also, adding sugar to beverages and intake of highly processed food *often* in comparison with *rarely* was associated with a lower odd of T2D.

### Childhood starvation, hypertension, family history, smoking and alcohol intake status associations with T2D

As shown in Table 4, exposure to childhood starvation was not significantly associated with T2D. Being hypertensive was associated with over two times higher odds of T2D, but this association attenuated to non-significant level after adjusting for all covariates. Having a first degree relative with diabetes was associated with over three times higher odds of T2D with ~50% reduction in odds after adjustment for all covariates. Smoking and alcohol intake status was not significantly associated with T2D.

### Physical activity

As shown in Table 5, there were no significant associations between accelerometer-measured physical activity and sedentary time, and T2D. Physical activity levels were high in both cases and controls, and men were more physically active than women. Each standard deviation increase in leisure time self-reported moderate and vigorous physical activity (MVPA) was associated with two times higher odds in model 2, but this association attenuated to nonsignificant level in the fully adjusted model (model 3).

**Table 1** Sociodemographic characteristics, glucose levels and time since diagnosis (for cases) disaggregated sex and study groups

| | Controls | | Cases | | |
|---|---|---|---|---|---|
| | Women $n = 37$ Mean (SD) | Men $n = 33$ Mean (SD) | Women $n = 37$ Mean (SD) | Men $n = 33$ Mean (SD) | *P* value Control vs. Case |
| Age in years | 47.7 (6.7) | 48.4 (9.2) | 50.8 (8.3) | 49.7 (8.6) | 0.112 |
| Residence in Nairobi in years | 23.7 (11.9) | 27.3 (15.1) | 26.1 (14.2) | 27.7 (11.1) | 0.514 |
| Education level | | | | | |
| No formal education | 3 (8) | 3 (9) | 6 (16) | 1 (3) | 0.879 |
| Primary | 22 (59) | 14 (42) | 21(57) | 16 (48) | |
| Secondary | 11 (30) | 9 (27) | 5 (14) | 11 (33) | |
| Tertiary | 1 (3) | 7 (21) | 5 (14) | 5 (15) | |
| Wealth index | | | | | |
| 1 (Poorest) | 4 (11) | 8 (24) | 10 (27) | 9 (27) | 0.220 |
| 2 | 11 (30) | 11 (33) | 8 (22) | 6 (18) | |
| 3 | 13 (35) | 7 (21) | 8 (22) | 8 (24) | |
| 4 (Wealthiest) | 9 (24) | 7 (21) | 11 (30) | 10 (30) | |
| Glucose level (mmol/l) | 4.9 (0.6) | 4.6 (0.7) | 10.3 (6.0) | 9.1 (6.1) | <0.001 |
| Months since diagnosis | N/A | N/A | 15.4 (8.8) | 13.8 (8.5) | N/A |

SD, standard deviation; N/A, not applicable.
*P* values based on ANOVA test for continuous variables and Fishers exact test for categorical variables.

**Table 2** Body composition, obesity, handgrip and sleep duration characteristics and associations with type 2 diabetes

| | Controls | | Cases | | | | |
|---|---|---|---|---|---|---|---|
| | Women $n = 37$ Mean (SD) | Men $n = 33$ Mean (SD) | Women $n = 37$ Mean (SD) | Men $n = 33$ Mean (SD) | Odds ratios (95% CI) | | |
| | | | | | Model 1 | Model 2 | Model 3 |
| Height (cm) | 160.5 (5.7) | 172.1 (7.0) | 157.2 (5.9) | 169.1 (5.8) | **0.68 (0.48, 0.96)*** | **0.47 (0.28, 0.80)**** | **0.34 (0.17, 0.66)**** |
| Weight (kg) | 81.0 (19.3) | 68.8 (17.9) | 73.0 (17.0) | 69.8 (19.1) | 0.82 (0.58, 1.15) | 0.79 (0.55, 1.14) | 0.78 (0.53, 1.14) |
| Body fat (%) | 39.9 (8.7) | 19.1 (9.0) | 38.5 (6.9) | 21.7 (10.5) | 1.05 (0.75, 1.46) | 1.00 (0.60, 1.69) | 0.98 (0.55, 1.75) |
| Body fat-mass (kg) | 33.7 (13.2) | 14.5 (11.4) | 29.1 (12.1) | 16.9 (12.5) | 0.92 (0.66, 1.29) | 0.86 (0.57, 1.31) | 0.84 (0.54, 1.31) |
| Body fat-free mass (kg) | 47.2 (7.0) | 54.3 (7.5) | 43.9 (5.4) | 53.0 (7.9) | 0.73 (0.52, 1.04) | **0.65 (0.42, 0.99)*** | **0.42 (0.24, 0.75)**** |
| BMI ($kg.m^{-2}$) | 31.3 (6.4) | 23.1 (5.4) | 29.6 (6.5) | 24.2 (5.5) | 0.94 (0.67, 1.31) | 0.90 (0.60, 1.34) | 0.89 (0.58, 1.37) |
| WC (cm) | 95.0 (15.4) | 82.8 (15.7) | 93.4 (12.4) | 89.3 (15.6) | 1.16 (0.83, 1.61) | 1.13 (0.78, 1.63) | 1.06 (0.71, 1.60) |
| HC (cm) | 111.0 (14.9) | 95.1 (11.8) | 106.1 (12.5) | 95.9 (13.5) | 0.86 (0.62, 1.20) | 0.79 (0.53, 1.18) | 0.76 (0.49, 1.18) |
| WHtR | 0.59 (0.09) | 0.48 (0.09) | 0.60 (0.08) | 0.53 (0.08) | 1.28 (0.91, 1.79) | 1.31 (0.88, 1.96) | 1.25 (0.80, 1.96) |
| WHR | 0.86 (0.07) | 0.87 (0.07) | 0.88 (0.05) | 0.93 (0.06) | **2.02 (1.38, 2.97)***** | **2.09 (1.38, 3.17)***** | **2.28 (1.38, 3.79)**** |
| Handgrip strength (kg) | 28.4 (4.7) | 40.1 (5.6) | 23.9 (4.4) | 37.4 (6.3) | **0.63 (0.44, 0.89)**** | **0.33 (0.18, 0.61)***** | **0.20 (0.08, 0.45)***** |
| Average sleep (hours/day) | 7.9 (1.0) | 7.5 (2.4) | 7.3 (1.4) | 7.6 (1.2) | 0.85 (0.60, 1.19) | 0.86 (0.61, 1.21) | 0.80 (0.52, 1.22) |

SD, standard deviation; BMI, Body mass index; WHR, waist-to-hip ratio; WHtR, waist-to-height ratio.
$P < 0.05$ for odds ratios in bold. *<0.05, **<0.01, ***<0.001.
Odds ratios are for being a Case compared with Control per SD increase in variable. Model 1 – unadjusted odds ratios (95% confidence intervals (CI)); Model 2 – odds ratios (95% CI) adjusted for age, sex, wealth index and education; Model 3 – Model 2 + adjusted for smoking status, alcohol drinking status, childhood starvation, fruit and vegetable intake, hypertension, physical activity (metabolic equivalents), sleep duration (except in average sleep models) and family history of diabetes. Odds ratios for height, fat-free mass, grip strength and average sleep in Model 3 have been adjusted further for WHR.

**Table 3** Self-reported dietary practices and their associations with T2D

| | Controls | | Cases | | Odds ratios (95% CI) | | |
|---|---|---|---|---|---|---|---|
| | Women $n = 37$ | Men $n = 33$ | Women $n = 37$ | Men $n = 33$ | | | |
| | Mean (SD) | Mean (SD) | Mean (SD) | Mean (SD) | Model 1 | Model 2 | Model 3 |
| Meals bought from vendor/week | 1.8 (3.3) | 7.1 (5.9) | 1.2 (2.2) | 4.7 (4.6) | 0.72 (0.50, 1.03) | 0.67 (0.45, 1.02) | **0.56 (0.34, 0.91)*** |
| Bread slices/day | 1.3 (1.6) | 1.4 (1.3) | 1.1 (1.2) | 2.1 (2.4) | 1.15 (0.82, 1.61) | 1.16 (0.82, 1.66) | 1.08 (0.73, 1.60) |
| Sugary drinks/week | 1.0 (2.3) | 2.0 (2.4) | 0.7 (2.0) | 1.0 (2.3) | 0.68 (0.46, 1.01) | 0.68 (0.45, 1.03) | 0.81 (0.53, 1.24) |
| Fruit & vegetable servings/day | 2.2 (0.9) | 2.4 (1.4) | 3.6 (2.2) | 3.5 (1.6) | **2.57 (1.60, 4.14)***** | **2.53 (1.57, 4.08)***** | **2.58 (1.54, 4.33)***** |
| Frequency of adding sugar to beverages– *n* (%) | | | | | | | |
| Rarely | 13 (35) | 7 (21) | 33 (89) | 25 (76) | Ref. | Ref. | Ref. |
| Often | 24 (65) | 26 (79) | 4 (11) | 8 (24) | **0.09 (0.04, 0.19)***** | **0.07 (0.03, 0.18)***** | **0.06 (0.02, 0.17)***** |
| Frequency of intake of processed foods high in sugar– *n* (%) | | | | | | | |
| Rarely | 32 (86) | 26 (79) | 36 (97) | 32 (97) | Ref. | Ref. | Ref. |
| Often | 5 (14) | 7 (21) | 1 (3) | 1 (3) | **0.14 (0.03, 0.66)*** | **0.15 (0.03, 0.69)*** | **0.12 (0.02, 0.75)*** |
| Advised by health worker to change diet – n (%) | | | | | | | |
| No | 18 (49) | 26 (79) | 2 (5) | 5 (15) | Ref. | Ref. | Ref. |
| Yes | 19 (51) | 7 (21) | 35 (95) | 28 (85) | **15.2 (6.08, 38.19)***** | **19.1 (7.00, 52.2)***** | **21.1 (7.00, 63.6)***** |

Ref. – Reference. $P < 0.05$ for odds ratios in bold. *<0.05, **<0.01, ***<0.001.

Odds ratios are for being a Case compared with Control per SD increase in variable. Model 1 – unadjusted odds ratios (95% confidence intervals (CI)); Model 2 – odds ratios adjusted for age, sex, wealth index and education (95% CI); Model 3 – Model 2 + adjusted for waist-to-hip ratio, smoking status, alcohol drinking status, childhood starvation, hypertension, physical activity (metabolic equivalents), sleep duration and family history of diabetes.

**Table 4** Childhood starvation, hypertension, family history, smoking and alcohol intake status associations with T2D

| | Controls | | Cases | | | | |
|---|---|---|---|---|---|---|---|
| | Women $n = 37$ $n$ (%) | Men $n = 33$ $n$ (%) | Women $n = 37$ $n$ (%) | Men $n = 33$ $n$ (%) | Odds ratios (95% CI) | | |
| | | | | | Model 1 | Model 2 | Model 3 |
| Experienced childhood starvation | | | | | | | |
| No | 18 (49) | 15 (45) | 19 (51) | 14 (42) | **Ref.** | **Ref.** | **Ref.** |
| Yes | 19 (51) | 18 (55) | 18 (49) | 19 (58) | 1.00 (0.51, 1.94) | **0.97 (0.48, 1.99)** | 0.96 (0.41, 2.24) |
| Hypertensive | | | | | | | |
| No | 22 (59) | 23 (70) | 12 (32) | 17 (51) | **Ref.** | **Ref.** | **Ref.** |
| Yes | 15 (41) | 10 (30) | 25 (68) | 16 (49) | **2.54 (1.29, 5.03)**** | **2.47 (1.13, 5.41)*** | 2.34 (0.94, 5.82) |
| Family history of diabetes | | | | | | | |
| No known history | 26 (70) | 20 (61) | 19 (51) | 12 (37) | Ref. | Ref. | Ref. |
| First-degree relative | 6 (16) | 6 (18) | 17 (46) | 14 (42) | **3.83 (1.71, 8.59)**** | **3.61 (1.60, 8.17)**** | **3.32 (1.27, 8.68)*** |
| Second-degree relative | 5 (14) | 7 (21) | 1 (3) | 7 (21) | 0.99 (0.36, 2.70) | 0.98 (0.34, 2.83) | 0.88 (0.26, 3.03) |
| Smoking status | | | | | | | |
| Never smoked | 35 (95) | 15 (46) | 36 (97) | 13 (39) | Ref. | Ref. | Ref. |
| Former smoker | 2 (5) | 9 (27) | 1 (3) | 13 (39) | 1.30 (0.54, 3.14) | 1.21 (0.43, 3.42) | 1.29 (0.36, 4.65) |
| Current smoke | 0 (0) | 9 (27) | 0 (0) | 7 (22) | 0.79 (0.27, 2.30) | 0.83 (0.23, 3.01) | 1.05 (0.18, 5.98) |
| Alcohol intake status | | | | | | | |
| Never drank | 23 (62) | 8 (24) | 26 (70) | 4 (12) | Ref. | Ref. | Ref. |
| Former drinker | 10 (27) | 10 (30) | 10 (27) | 15 (46) | 1.29 (0.60, 2.80) | 1.16 (0.49, 2.74) | 0.99 (0.35, 2.80) |
| Current drinker | 4 (11) | 15 (46) | 1 (3) | 14 (42) | 0.82 (0.35, 1.89) | 0.77 (0.28, 2.12) | 0.81 (0.19, 3.43) |

$P < 0.05$ for odds ratios in bold. *<0.05, **<0.01, ***<0.001.
Model 1 – unadjusted odds ratios (95% confidence intervals (CI)); Model 2 – odds ratios adjusted for age, sex, wealth index and education (95% CI); Model 3 – Model 2 + adjusted for waist-to-hip ratio, fruit and vegetable intake, physical activity (metabolic equivalents), sleep duration, starvation (except in starvation models), hypertension (except in hypertension models), family history (except in family history models), smoking status (except in smoking status models) and alcohol intake status (except in alcohol intake models).

## Discussion

This study aimed to understand the phenotype and risk factor profile of T2D in Kenya using recently diagnosed diabetes cases and matched controls. Despite BMI and body fat being globally recognised as important risk factors for T2D [42], our study did not detect any differences between cases and controls; and the mean BMI in male cases was a normal weight. However, each standard deviation increase in WHR was associated with more than doubling of the odds of T2D. Thus, it appears that although cases did not have higher BMIs, their body fat was distributed more centrally. This agrees with several other studies in SSA which have reported that central obesity may predict T2D better than BMI [43–49]. Further, our findings, at least in men, confirm a lean diabetes phenotype, which has been reported in other SSA settings [9, 50]. Such a phenotype maybe due to a metabolic unhealthy normal weight, which has previously been associated with a higher visceral obesity that increases the risk of T2D [10]. Therefore, our findings suggests that general messages aimed at weight loss to reduce T2D risk may be less appropriate in Kenya compared with typical populations with or at high risk for T2D in high-income countries. It may also be appropriate to switch focus from BMI to measures of central obesity to identify those at increased T2D risk, and it may be important to intervene to reduce weight in those with high central obesity even if they are normal weight.

Each standard deviation increase in height was associated with three times lower odds of T2D. This finding is consistent with a recent meta-analysis of 16 studies, which included one study from SSA (Nigeria), that reported an inverse association between height and T2D risk in both men and women [51]. However, a recent Mendelian randomisation study using European ancestry data found that the association between adulthood height and T2D did not persist after adjustment for BMI [52]. Further, a recent analysis that included data from 10 SSA countries, including Kenya, did not find an association between height and T2D except among Namibian women and Tanzanian men [53]. The mechanisms that link height and T2D risk remain unclear. First, adult height is to an extent determined by genetic factors, with genome-

**Table 5** Accelerometery and self-reported measures of physical activity in cases and controls and their association with T2D

| | Controls | | Cases | | | | |
|---|---|---|---|---|---|---|---|
| | Women $n = 35$ Mean (SD) | Men $n = 25$ Mean (SD) | Women $n = 34$ Mean (SD) | Men $n = 21$ Mean (SD) | Odds ratios (95% CI) Model 1 | Model 2 | Model 3 |
| Accelerometery measured physical activity level – minutes/day | | | | | | | |
| Sedentary | 595 (68) | 611 (94) | 596 (92) | 584 (117) | 0.92 (0.63, 1.33) | 0.89 (0.60, 1.32) | 0.91 (0.57, 1.46) |
| MVPA | 43 (24) | 74 (35) | 44 (21) | 77 (49) | 0.95 (0.66, 1.38) | 1.00 (0.64, 1.57) | 0.99 (0.58, 1.68) |
| Steps/day | 6986 (2947) | 10394 (3594) | 7252 (3121) | 10364 (3648) | 0.94 (0.65, 1.36) | 0.95 (0.61, 1.47) | 0.88 (0.51, 1.52) |
| Self-reported physical activity | | | | | | | |
| | Women $n = 37$ | Men $n = 33$ | Women $n = 37$ | Men $n = 33$ | | | |
| Work related physical activity – minutes/week | | | | | | | |
| MVPA | 417 (813) | 386 (722) | 166 (380) | 709 (1156) | 1.02 (0.73, 1.42) | 1.08 (0.76, 1.52) | 1.05 (0.71, 1.55) |
| Travel related physical activity – minutes/week | | | | | | | |
| Walking/cycling | 602 (528) | 838 (929) | 639 (738) | 432 (404) | 0.77 (0.54, 1.10) | 0.73 (0.50, 1.06) | 0.82 (0.53, 1.25) |
| Leisure related physical activity – minutes/week | | | | | | | |
| MVPA | 13 (41) | 38 (83) | 51 (188) | 80 (128) | 1.66 (0.93, 2.98) | **2.06 (1.04, 4.09)*** | 2.20 (0.96, 5.05) |
| METs | 4300 (3809) | 6396 (6822) | 3480 (3288) | 6262 (8895) | 0.92 (0.66, 1.29) | 0.95 (0.67, 1.35) | 0.97 (0.65, 1.44) |
| Sedentary time (minutes/week) | 3987 (1627) | 4068 (1765) | 3639 (1379) | 4281 (1640) | 0.95 (0.68, 1.32) | 0.92 (0.64, 1.32) | 0.92 (0.60, 1.40) |

SD, standard deviation; MVPA, Moderate and vigorous intensity physical activity; MET, metabolic equivalents; Ref., Reference; $P < 0.05$ for odds ratios in bold. *<0.05, **<0.01, ***<0.001.
Odds ratios for being a Case compared with Control per SD increase in variable. Model 1 – unadjusted odds ratios (95% confidence intervals (CI)); Model 2 – odds ratios adjusted for age, sex, wealth index and education (95% CI); Model 3 – Model 2 + adjusted for waist-to-hip ratio, smoking status, alcohol drinking status, childhood starvation, fruit and vegetable intake, hypertension, sleep duration and family history of diabetes.

wide data suggesting that common variants explain 60% of heritability [54]. Secondly, adult height is a product of growth across the prenatal, infancy, childhood and puberty periods, with height gain during one period potentially determining growth in the next [55]. However, we did not find differences in self-reported exposure to childhood starvation between cases and controls, which is contrary to a cross-sectional comparative study in rural Kenya that found self-reported childhood starvation to be associated with a doubling of T2D risk [17]. It is likely that this ascertainment in the current study, like other studies that seek to determine exposure retrospectively, was prone to recall bias [56]. Furthermore, our tool – which asked a single question about whether participants experienced childhood starvation – may not have been robust enough to provide a good measure of nutritional status across childhood. However, we found cases to be shorter, with less fat-free mass and higher WHR than controls, and it maybe that an adverse early-life environment contributes to lower height and fat-free mass, and increased propensity to store fat centrally. Prospective evidence has shown that early stunting was associated with not only shorter adult height but also reduced accumulation of subcutaneous fat and lean mass, and increased visceral fat [57]. There is, therefore, a need for more evidence both globally and in SSA to understand the role of height as a T2D risk factor, and the height-T2D association particularly in environments where the prevalence of adverse early life nutrition is high.

Fat-free mass (about half which is skeletal muscle mass [58, 59]) was associated with a 58% lower odds of T2D per standard deviation increase. A Korean prospective study reported that a low (compared to a high) skeletal muscle mass was associated with more than twice higher odds of diabetes [60]. Furthermore, handgrip strength, a proxy measure of overall muscular strength [61], was

 

associated with 80% lower odds of T2D per standard deviation increase. Evidence from a recent meta-analysis shows a 13% reduction in T2D risk per standard deviation increase in hand grip strength [62]. However, there is need for more evidence to determine whether low muscular strength plays a causal role in T2D development or is simply a marker of poor early life nutrition. The human baseline hypothesis proposed that height, placental and maternal factors in early life, and exercise and nutrition during childhood development are critical factors in determining handgrip strength in adulthood [63]. Overall, our findings therefore suggest that muscle mass maintenance/accumulation and muscle strengthening interventions maybe useful for T2D prevention in Kenya.

A positive family history of diabetes, specifically having a first degree relative with diabetes, was associated with more than three times higher odds of T2D, which was reduced by ~50% after adjustment for all covariates. This finding is consistent with a recent meta-analysis of SSA studies, which showed that a positive family history was associated with a threefold increased risk of T2D, with heritability being higher in first-degree, compared with second-degree, relatives [64]. As an independent risk factor, family history has been included in several T2D risk scores [4]. Our data therefore support the use of family history, particularly that of a first-degree relative, in risk stratification for T2D in Kenya.

Cases had healthier dietary practices, for example, each standard deviation increase in fruit and vegetable intake was associated with a more than twice higher odds of T2D. Similar associations between increased fruit and vegetable intake and T2D odds have been reported in a Ghanaian case–control study [65]. More than 80% of cases in the current study had been advised by a health worker to change their diet, meaning that post-diagnosis health education may have been effective, which implies that there is an opportunity to intervene on diet in high-risk groups through health education.

Objectively measured physical activity was higher in men than women, but there was no significant association with T2D. Physical activity levels in our study were substantially higher than those reported in high-income countries [66]. Women in our study accumulated about 43 min of daily MVPA compared with 32 min in high-income countries, whereas men accumulated about 75 min compared with 36 min in high-income countries. The use of objective measurement along with self-report provides an accurate measure of physical activity and the types and context of physical activity, all of which are important for informing interventions [67]. Our self-reported data suggest that the main contributor to physical activity was active travel, except for male cases where work was the main contributor. Leisure-related physical activity was the least important contributor across all subgroups. As physical activity levels were high (particularly in men) and did not differ between cases and controls, asking men to increase physical activity levels to reduce T2D risk in this population may not be the most appropriate intervention. In women, although physical activity levels did not differ between cases and controls, steps per day were lower compared with men and therefore may present an opportunity to intervene. As Kenya continues to develop economically, travel and work-related physical activity may decrease, potentially creating a need to increase leisure-time physical activity, which was the lowest form of physical activity in the current study.

Being hypertensive was associated with over two times higher odds of T2D, but this association attenuated to nonsignificant level on adjustment for all covariates. Hypertension, specifically elevated systolic blood pressure, has been associated with T2D in meta-analysis and Mendelian randomisation studies [3, 52], although it is unclear whether this association is causal [68]. Hypertension and T2D share risk factors such as genetics, adiposity, dietary factors and physical inactivity and hypertension indirectly increases insulin resistance by elevating inflammation and oxidative stress levels [69]. This shared pathway may result to an increased burden of both NCDs but also presents an opportunity to prevent the two diseases simultaneously through lifestyle modification interventions.

The current study adds to the scant evidence available on T2D aetiology in Kenya and SSA. Its strengths are the use of sociodemographically similar study groups to compare risk factor exposure, and objective measurement of physical activity. However, our findings should be interpreted within the limitations of the study design. First, we used a pragmatic convenience sample which may not be fully generalisable. Secondly, single measurement of most exposures (obesity, dietary intake, sleep, physical activity) limited temporality of associations (i.e. whether exposure preceded the outcome). However, it is unlikely that central obesity increased after T2D given that cases had been advised to modify their diets and had healthier dietary practices. Thirdly, there is a possibility of reverse causality in the diabetes-grip strength and diabetes-fat-free mass association as reported in a Mendelian randomisation study [70]. However, in the current study it is unlikely that T2D led to low grip strength, as our cases were all diagnosed within two years and a prospective study reported that grip strength reduction was similar in people with and without diabetes over a three year follow-up period [71]. Also, a prospective study has

reported that loss of fat-free mass is higher in people with undiagnosed, but not diagnosed diabetes when compared to those without diabetes [72]. This suggests that diabetes-related fat-free mass loss in our cases (who were all diagnosed) may have been minimal. Fourthly, use of self-reported data may have introduced recall biases, including social desirability bias [56], in some of the measures. For example, the cases may have reported dietary practices that they knew they should be adhering to, rather than what they were actually doing. Further, dietary intake was assessed during the data collection period, which might not reflect the usual trend of intake.

In conclusion, this study expands the understanding of T2D phenotype and risk factor profile in Kenya. Similar to high-income countries, family history was higher in people with T2D, but in contrast to those in high-income countries, they did not have higher BMI or lower physical activity levels compared with normoglycemic controls. Rather, cases were more centrally obese, shorter and had lower fat-free mass and muscle strength. These findings highlight the utility of family history for risk stratification in Kenya. Our data also suggest that central obesity, rather than BMI, could be used for risk stratification and that intervening to reduce central obesity, even when people have a normal weight, and to increase muscle mass and muscle strength may be useful T2D prevention strategies. Finally, there is need for more research to determine whether height, muscle mass and strength play a causal role in T2D and/or are indicators of an adverse early-life environment.

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