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.
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/m². Waist and hip circumferences were taken over light clothing using a SECA 201 tape measure at the umbilicus level and around the

Figure 1 Participation flow chart describing inclusion and exclusion of cases and controls.
widest portion of the buttocks, respectively [23, 28]. A third measure was taken if the first two were >0.7 cm 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 fingerpick 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 (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 accelerometry 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 activity 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 Kisu [23, 28]. A third measure was taken if the first two were >0.7 cm apart [29].

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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).

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

|                  | Controls                     | Cases                        | P value Control vs. Case |
|------------------|------------------------------|------------------------------|--------------------------|
|                  | Women n = 37 | Men n = 33 | Women n = 37 | Men n = 33 |                              |
| 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)                      | 6 (16)                     | 1 (3)                  | 0.879                         |
|                  | Primary | 22 (59)                      | 21 (57)                     | 16 (48)                |                               |
|                  | Secondary | 11 (30)                      | 5 (14)                      | 11 (33)                |                               |
|                  | Tertiary | 1 (3)                        | 5 (14)                      | 5 (15)                 |                               |
| Wealth index     | 1 (Poorest) | 4 (11)                      | 10 (27)                     | 9 (27)                 | 0.220                         |
|                  | 2       | 11 (30)                      | 8 (22)                      | 6 (18)                 |                               |
|                  | 3       | 13 (35)                      | 8 (22)                      | 8 (24)                 |                               |
|                  | 4 (Wealthiest) | 9 (24)                      | 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          |               | Odds ratios (95% CI)              |
|----------------------|----------------|----------------|---------------|-----------------------------------|
|                      | Women          | Men            | Women         | Men                               |
|                      | Mean (SD)      | Mean (SD)      | Mean (SD)     | Mean (SD)                         |
| Height (cm)          | 160.5 (5.7)    | 172.1 (7.0)    | 157.2 (5.9)   | 169.1 (5.8)                       |
|                      | 1.68 (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 Mean (SD) | Cases Mean (SD) | Odds ratios (95% CI) |
|--------------------------------------|--------------------|-----------------|----------------------|
|                                      | Women n = 37       | Men n = 33      | 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. **P < 0.01, ***P < 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.
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 recognized 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 suggest 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-
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

### Table 5

| Accelerometry and self-reported measures of physical activity in cases and controls and their association with T2D |
|---------------------------------------------------------------------------------------------------------------|
| **Controls**                                                                                                   |
| **Women**                                                                                                      |
| **Men**                                                                                                        |
| **n = 35**                                                                                                     |
| **n = 25**                                                                                                     |
| **Mean (SD)**                                                                                                   |
| **Cases**                                                                                                       |
| **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                                                                                                      |
| MVPA                                                                                                           |
| Steps/day                                                                                                      |
| Self-reported physical activity                                                                               |
| Work related physical activity – minutes/week                                                                 |
| MVPA                                                                                                           |
| Travel related physical activity – minutes/week                                                                |
| Walking/cycling                                                                                                 |
| Leisure related physical activity – minutes/week                                                              |
| MVPA                                                                                                           |
| METs                                                                                                           |
| Sedentary time (minutes/week)                                                                                   |
| 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, ***<0.001.

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.
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 hand grip 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 threelfold 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 mea-
ures. 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, diet-
ary 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 physi-
ical 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 peo-
ple 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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References

1. WHO. Noncommunicable diseases. 2018 (Available from:
http://www.who.int/news-room/fact-sheets/detail/noncommu-
icable-diseases). [22 April 2021]

2. IDF. IDF Diabetes atlas 9th edition 2019. 2019 (Available
from: https://diabetesatlas.org/en/). [22 April 2021]

3. Bellou V, Belbasis L, Tzoulaki I, Evangelou E. Risk factors
for type 2 diabetes mellitus: an exposure-wide umbrella
review of meta-analyses. PLoS One 2018: 13; e0194127.

4. Noble D, Mathur R, Dent T, Meads C, Greenhalgh T. Risk
models and scores for type 2 diabetes: systematic review.
BMJ 2011: 343.

5. Dunkley AJ, Bodicoat DH, Greaves CJ et al. Diabetes pre-
vention in the real world: effectiveness of pragmatic lifestyle
interventions for the prevention of type 2 diabetes and of
the impact of adherence to guideline recommendations. A
Systematic Review and Meta-analysis. Diabetes Care 2014:
37: 922–933.

6. Kunutsor SK, Isiozor NM, Khan H, Laukkonen JA. Hand-
grip strength—a risk indicator for type 2 diabetes: systematic
review and meta-analysis of observational cohort studies.
Diabetes/Metabolism Res Rev 2020: 37: e3365.

7. Berends L, Ozanne S. Early determinants of type-2 diabetes.
Best Pract Res Clin Endocrinol Metab 2012: 26: 569–580.

8. Grandner MA, Seixas A, Shetty S, Shenoy S. Sleep duration
and diabetes risk: population trends and potential mecha-
nisms. Curr Diab Rep 2016: 16: 106.

9. Kibirige D, Lumu W, Jones AG et al. Understanding the
manifestation of diabetes in sub Saharan Africa to inform
therapeutic approaches and preventive strategies: a narrative
review. Clinical Diabetes and Endocrinology 2019: 5: 2.

10. Stefan N, Schick F, Häring H-U. Causes, characteristics, and
consequences of metabolically unhealthy normal weight in
humans. Cell Metab 2017: 26: 292–300.

11. McGrath R et al. What are the association patterns between
handgrip strength and adverse health conditions? A topical
review. SAGE Open Med 2020: 8: 205312120910358.

12. MOH. Kenya National Strategy for the Prevention and
Control of Non-Communicable Diseases 2015-2020, D.o.N.-c.
Diseases, Editor. 2015, Ministry of Health: Nairobi, Kenya.

13. Shiroya V, Neuhann F, Müller O, Deckert A. Challenges in
policy reforms for non-communicable diseases: the case of
diabetes in Kenya. Global Health Action 2019: 12:
1611243.

14. Ayah R, Joshi MD, Wanjiru R et al. A population-based
survey of prevalence of diabetes and correlates in an urban
slum community in Nairobi, Kenya. BMC Public Health
2013: 13: 371.

15. Mohamed SF, Mwangi M, Mutua MK et al. Prevalence and
factors associated with pre-diabetes and diabetes mellitus in
Kenya: results from a national survey. BMC Public Health
2018: 18: 1215.

16. Sarah K, Abdillahi HS, Reuben M, Lydia A. Prevalence and
risk factors associated with diabetes in Meru County,
Kenya: a cross-sectional study. Int J Diab Dev Countries
29. Willis LH, Slentz CA, Houmard JA

27. Chalmers JO, MacMahon S, Mancia G

21. Diaz-Valencia PA, Bougn

24. Ramsay M, Crowther N, Tambo E

30. Roberts HC, Denison HJ, Martin HJ

32. Troiano RP, Berrigan D, Dodd KW, M

19. Sullivan KM, Dean A, Soe MM. OpenEpi: a web-based epidemiologic and statistical calculator for public health. Public Health Reports, 2009: 124: 471–474.

20. NDCP. National Clinical Guidelines for Management of Diabetes Mellitus. 2010.

21. Diaz-Valencia PA, Bougnères P, Valleron A-J. Global epidemiology of type 1 diabetes in young adults and adults: a systematic review. BMC Public Health 2015: 15: 255.

22. Vinik A, Flemmer M. Diabetes and macrovascular disease. J Diabetes Complications 2002: 16: 235–245.

23. MOH. Kenya STEPwise survey for non-communicable diseases risk factors 2015 report. 2015.

24. Ramsay M, Crowther N, Tambo E et al. H3Africa AWI-Gen Collaborative Centre: a resource to study the interplay between genomic and environmental risk factors for cardiometabolic diseases in four sub-Saharan African countries. Global Health Epidemiol Genom 2016: 1. https://doi.org/10.1017/gheg.2016.17. [25 April 2021]

25. Vyas S, Kumaranayake L. Constructing socio-economic status indices: how to use principal components analysis. Health Policy and Planning 2006: 21: 459–468.

26. Muntner P, Shimbo D, Carey RM et al. Measurement of blood pressure in humans: a scientific statement from the American Heart Association. Hypertension 2019: 73: e35–e66.

27. Chalmers JO, MacMahon S, Mancia G et al. 1999 World Health Organization-International Society of Hypertension Guidelines for the management of hypertension. Guidelines sub-committee of the World Health Organization. Clin Exp Hypertens, 1999: 21: 1009–1060.

28. WHO. Waist circumference and waist-hip ratio: report of a WHO expert consultation, Geneva, 8–11 December 2008. 2011.

29. Willis LH, Slentz CA, Houmard JA et al. Minimal versus umbilical waist circumference measures as indicators of cardiovascular disease risk. Obesity 2007: 15: 753–759.

30. Roberts HC, Denison HJ, Martin HJ et al. A review of the measurement of grip strength in clinical and epidemiological studies: towards a standardised approach. Age Ageing 2011: 40: 423–429.

31. WHO. Definition and diagnosis of diabetes mellitus and intermediate hyperglycaemia: report of a WHO/IDF consultation. 2006.

32. Troiano RP, Berrigan D, Dodd KW, Mässé LC, Tiliert T, Mcdowell M. Physical activity in the United States measured by accelerometer. Med Sci Sports Exerc 2008: 40: 181–188.

33. Katapally TR, Muhajarine N. Towards uniform accelerometer analysis: a standardization methodology to minimize measurement bias due to systematic accelerometer wear-time variation. J Sports Sci Med 2014: 13: 379.

34. Keino S, van den Borne B, Plasqui G. Body composition, water turnover and physical activity among women in Narok County, Kenya. BMC Public Health 2014: 14: 1212.

35. Wennman H et al. Gender, age and socioeconomic variation in 24-hour physical activity by wrist-worn accelerometers: the FinHealth 2017 Survey. Sci Rep 2019: 9: 1–9.

36. Doherty A, Jackson D, Hammerla N et al. Large scale population assessment of physical activity using wrist worn accelerometers: the UK biobank study. PLoS One 2017: 12: e0169649.

37. WHO. The WHO STEPwise approach to noncommunicable disease risk factor surveillance. 2017. 2017, 3-5-5.

38. RCT. R: A language and environment for statistical computing. 2013, Vienna, Austria.

39. Kuo C-L, Duan Y, Grady J. Unconditional or conditional logistic regression model for age-matched case–control data? Frontiers in Public Health 2018: 6.

40. Pearce N. Analysis of matched case-control studies. BMJ 2016: 352: i699.

41. Althouse AD. Adjust for multiple comparisons? It’s not that simple. Ann Thorac Surg 2016: 101: 1644–1645.

42. Jo A, Mainous AG III. Informational value of percent body fat with body mass index for the risk of abnormal blood glucose: a nationally representative cross-sectional study. BMJ open 2018: 8: e019200.

43. Balde NM, Diallo I, Balde MD et al. Diabetes and impaired fasting glucose in rural and urban populations in Futa Jallon (Guinea): prevalence and associated risk factors. Diabetes Metab 2007: 33: 114–120.

44. Frank LK, Heraclides A, Danquah I, Bedu-Addo G, Mockenhaupt FP, Schulze MB. Measures of general and central obesity and risk of type 2 diabetes in a Ghanaian population. Tropical Med Int Health 2013: 18: 141–151.

45. Haregu TN, Ori S, Egondi T, Kyobutungi C. Measurement of overweight and obesity an urban slum setting in sub-Saharan Africa: a comparison of four anthropometric indices. BMC Obesity 2016: 3: 46.

46. Mbanya VN, Kengne AP, Mbanya JC, Akhtar H. Body mass index, waist circumference, hip circumference, waist–hip ratio and waist–height-ratio: Which is the better discriminator of prevalent screen-detected diabetes in a Cameroonian population? Diabetes Res Clin Pract 2015: 108: 23–30.

47. Tesfaye T, Shikur B, Shimels T, Firdu N. Prevalence and factors associated with diabetes mellitus and impaired fasting glucose level among members of federal police commission residing in Addis Ababa, Ethiopia. BMC Endocrine Dis 2016: 16: 68.

48. Woldegebriel AG, Fenta KA, Aregay AB et al. Effectiveness of anthropometric measurements for identifying diabetes and prediabetes among civil servants in a regional city of northern Ethiopia: a cross-sectional study. J Nutr Metab 2020: 2020: 8425912.
50. Chilunga FP, Henneman P, Meeks KAC et al. Prevalence and determinants of type 2 diabetes among lean African migrants and non-migrants: the RODAM study. J Global Health 2019: 9: 20426.
51. Shrestha S, Rasmussen SH, Pottegård A et al. Critical growth phases for adult short-stunting in early childhood with cardiometabolic risk factors in adulthood. Am J Epidemiol 1990: 131: 837–49.
52. Yuan S, Larsson SC. An atlas on risk factors for type 2 diabetes: a wide-angled Mendelian randomisation study. Diabetes Care 2020: 43: 2403–2410.
53. Teufel F, Geldsetzer P, Manne-Goehler J et al. Analysis of attained height and diabetes among 554,122 adults across 25 low- and middle-income countries. Diabetes Care 2020: 43: 2403–2410.
54. Wood AR, Esco T, Yang J et al. Defining the role of common variation in the genomic and biological architecture of adult human height. Nat Genet 2014: 46: 1173–1186.
55. Luo ZC, Karlberg J. Critical growth phases for adult shortness. Am J Epidemiol 2000: 152: 125–131.
56. Coughlin SS. Recall bias in epidemiologic studies. J Clin Epidemiol 1990: 43: 87–91.
57. De Lucia Rolfe E, de França GVA, Vianna CA et al. Associations of stunting in early childhood with cardiometabolic risk factors in adulthood. PLoS One 2018: 13: e0192196.
58. Jensen B, Braun W, Geisler C et al. Limitations of fat-free mass for the assessment of muscle mass in obesity. Obesity facts 2019: 12: 307–315.
59. Strugnell C, Dunstan DW, Magliano DJ, Zimmet PZ, Shaw JE, Daly RM. Influence of age and gender on fat mass, fat-free mass and skeletal muscle mass among Australian adults: the Australian diabetes, obesity and lifestyle study (AusDiab). J Nutr Health Aging 2014: 18: 540–546.
60. Moon SS. Low skeletal muscle mass is associated with insulin resistance, diabetes, and metabolic syndrome in the Korean population: the Korea National Health and Nutrition Examination Survey (KNHANES) 2009–2010. Endocr J 2014: 61: 61–70.
61. Bohannon RW, Magasi SR, Bubela DJ, Wang Y-C, Gershon RC. Grip and knee extension muscle strength reflect a common construct among adults. Muscle Nerve 2012: 46: 555–558.
62. Tarp J, Stole AP, Blond K, Grøntved A. Cardiorespiratory fitness, muscular strength and risk of type 2 diabetes: a systematic review and meta-analysis. Diabetologia 2019: 62: 1129–1142.
63. Buckner SL, Dankel SJ, Bell ZW, Abe T, Loenneke JP. The association of handgrip strength and mortality: what does it tell us and what can we do with it? Rejuvenation Res 2019: 22: 230–234.
64. Asamoah EA, Obirikorang C, Acheampong E et al. Heritability and genetics of type 2 diabetes mellitus in Sub-Saharan Africa: a systematic review and meta-analysis. J Diab Res 2020: 2020: 3718671.
65. Gudjóns J, Sarfo B. Risk factors for type 2 diabetes mellitus among out-patients in Ho, the Volta regional capital of Ghana: a case–control study. BMC Res Notes 2017: 10: 1–10.
66. Hallal PC, Andersen LB, Bull FC, Guthold R, Haskell W, Ekelund U. Global physical activity levels: surveillance progress, pitfalls, and prospects. Lancet 2012: 380: 247–257.
67. Haskell WL. Physical activity by self-report: a brief history and future issues. J Phys Act Health 2012: 9: S5–S10.
68. Emdin CA, Anderson SG, Woodward M, Rahimi K. Usual blood pressure and risk of new-onset diabetes: evidence from 4.1 million adults and a meta-analysis of prospective studies. J Am Coll Cardiol 2015: 66: 1552–1562.
69. Cheung BMY, Li C. Diabetes and hypertension: is there a common metabolic pathway? Current Atherosc Rep 2012: 14: 160–166.
70. Yeung CHC, Au Yeung SL, Fong SSM, Schooling CM. Lean mass, grip strength and risk of type 2 diabetes: a bidirectional Mendelian randomisation study. Diabetologia 2019: 62: 789–799.
71. Park SW, Goodpaster BH, Strotmeyer ES et al. Accelerated loss of skeletal muscle strength in older adults with type 2 diabetes. Health Aging Body Comp Study 2007: 30: 1507–1512.
72. Park SW, Goodpaster BH, Lee JS et al. Excessive loss of skeletal muscle mass in older adults with type 2 diabetes. Diabetes Care 2009: 32: 1993–1997.