BMJ Open

Comparison of central obesity prevalence among adults living with and without HIV in Botswana: a cross-sectional study

Thato Moshomo,[1] Moagedi Mawi,[2] Christopher G Williams,[3] Kesaobaka Molebatsi,[3] Tiny Masupe ,[4] Kutlo Manyake,[3] Shahin Lockman,[3,5] Onkabetse Julia Molefe-Baikai ,[1] Atsile Leero,[1] Joseph N Jarvis,[3,6] Tendani Gaolathe,[1,3] Mosepele Mosepele [1,3,7]

To cite: Moshomo T, Mawi M, Williams CG, *et al*. Comparison of central obesity prevalence among adults living with and without HIV in Botswana: a cross-sectional study. *BMJ Open* 2025;**15**:e096170. doi:10.1136/bmjopen-2024-096170

► Prepublication history and additional supplemental material for this paper are available online. To view these files, please visit the journal online (https://doi.org/10.1136/bmjopen-2024-096170).

Received 05 November 2024
Accepted 04 April 2025

For numbered affiliations see end of article.

Correspondence to
Dr Mosepele Mosepele; mosepele.mosepele@gmail.com

ABSTRACT

Objectives The aim was to establish the community prevalence of central obesity in Botswana and assess its association with HIV status.

Design We performed a one-time central obesity assessment nested within a community-based cluster-randomised controlled HIV treatment and prevention trial (Botswana Combination Prevention Project (BCPP)) conducted in Botswana.

Setting The BCPP enrolled consenting adults from a random sample of 20% of households in 30 rural/peri-urban communities.

Participants A subset of participants from 22 communities was selected for a nested central obesity study.

Primary and secondary outcome measures Central obesity was defined as a waist-to-hip ratio (WHR)>0.90 for males and >0.85 for females or as a waist circumference (WC) ≥94 cm for males and ≥80 cm for females. A modified Poisson regression model was used to ascertain the association between central obesity and HIV status. Additionally, the same model was used to estimate the adjusted prevalence ratio (aPR) for central obesity among participants with missing waist and hip measurements by applying inverse probability weighting, and then adjusting for sex and age in the final multivariate models.

Results Of the 3981 adults, 2039 (51%) completed central obesity assessment (67% female, 29% people living with HIV and median age 35.4 years (IQR 26.4–48.3 years). Central obesity prevalence was 43.5% (95% CI 41.4% to 45.7%) and 50.8% (95% CI 48.6% to 52.9%) as defined by WHR and WC, respectively, and was higher among females than males by WHR (46.9% (95% CI 44.2% to 49.5%) vs 36.7% (95% CI 33.1% to 40.4%)) and WC 68.5% ((95% CI 65.9% to 70.9%) vs 15.1% (95% CI 12.4% to 17.8%)) and increased with age. In fully adjusted models, there was no difference in central obesity by HIV status for both WHR and WC, aPR 0.99 (95% CI 0.90 to 1.09), p value 0.88, and 0.93 (95% CI 0.85 to 1.01), p value 0.06, respectively.

Conclusion Over two-thirds of adult females in Botswana had central obesity; however, living with HIV was not consistently associated with central obesity.

STRENGTHS AND LIMITATIONS OF THIS STUDY

⇒ This study used a large, community-based sample from rural and peri-urban settings, enhancing the generalisability of findings to measure central obesity by HIV status in Botswana.
⇒ Central obesity was assessed using two established criteria (waist-to-hip ratio and waist circumference), allowing for a comprehensive evaluation.
⇒ We had a high refusal rate for the central obesity assessment among Botswana Combination Prevention Project survey participants, with 49% of approached individuals declining obesity measurements, which may have introduced selection bias.
⇒ We accounted for missing measurements by applying inverse probability weighting to reduce selection bias.
⇒ The lack of longitudinal data prevented an analysis of the impact of antiretroviral therapy regimen and duration on central obesity among people living with HIV.

Trial registeration number NCT01965470.

INTRODUCTION

Background/rationale

Obesity is a major global health concern and well-established risk factor for many chronic diseases, including cardiovascular disease, diabetes mellitus, chronic kidney disease and malignancies.[1] While body mass index is typically used as a general obesity index, excess abdominal fat (also known as central obesity) has a greater predictive value for health problems[2–5] such as cardiometabolic diseases, insulin resistance and diabetes mellitus, liver disease, neurocognitive impairment and malignancies.[1 3 6 7] Measures of central obesity, such as waist circumference (WC) and waist-to-hip ratio (WHR), are commonly employed to evaluate central fat distribution.[3 8 9]

The association between central obesity and HIV infection has been evident since the advent of modern antiretroviral therapy (ART) in the late 1990s.[7 10–12] Factors such as the 'return to health' phenomenon may cause some weight gain among those who initiate ART at low CD4 counts in the context of pre-ART weight loss; however, this is not sufficient to explain the weight gain and central obesity associated with some ART, particularly integrase inhibitors[12 13] such as dolutegravir, which has been a recommended first-line agent for the treatment of HIV infection in adults in Botswana since 2016.[14] ART is associated with weight gain through several factors, such as the reduction of the catabolic state and inflammatory markers of HIV, improved appetite and nutrient absorption.[7]

In sub-Saharan Africa, where HIV prevalence is high, there is a growing concern about the increasing burden of obesity among people living with HIV (PLWH). Despite potentially different national ART regimens and socio-environmental factors among PLWH in sub-Saharan Africa, the number of published large community and population-based studies focusing on central obesity and its distribution by HIV status in low-income and middle-income countries is limited, with significant heterogeneity in the study population and diagnostic criteria.[15 16] This study seeks to address this gap by examining central obesity prevalence in Botswana, offering insights into how HIV may influence obesity patterns in the region.

Objectives

1. To determine the community prevalence of central obesity among adults in Botswana, stratified by two sex-based criteria using the WHR and WC.
2. To assess whether central obesity prevalence differs by HIV status.

METHODS

Between April 2017 and March 2018, we conducted a single assessment of central obesity within participants' homes as part of the Botswana Combination Prevention Project (BCPP or Ya Tsie), a community-based pair-matched cluster-randomised trial (NCT01965470).[17 18] This trial, conducted in rural and peri-urban communities in Botswana, aimed to evaluate the efficacy of a set of enhanced interventions in reducing HIV incidence over a 3-year period. The first-line ART regimen provided by the government to all trial communities at the time of obesity assessment was tenofovir disoproxil fumarate (TDF)–emtricitabine (FTC)–dolutegravir (DTG).

The source population for the main trial was approximately 10% of the entire population of Botswana, representing 180 000 people drawn from 30 rural and peri-urban communities across the country.[17 18]

Settings and participants

The BCPP study[17] enrolled a community-based survey cohort of adults, in which this central obesity study was nested. This community-based cohort was established by enrolling all consenting residents aged 16–64 years who lived in a random sample of 20% of all households in the 30 participating villages. Only 22 out of the 30 communities were included in the central obesity assessment as ethical approval for the central obesity evaluation was granted after eight communities had already completed the primary trial assessments. During the third and final household survey for the main trial, all adults taking part in the BCPP household survey in these 22 communities were invited to participate in the central obesity assessment substudy. Potential participants could decline to participate in all or some of the planned central obesity evaluations.

Study procedures

Trained research assistants administered an investigator-developed questionnaire to all participants. Waist and hip circumferences were measured in duplicate by trained research assistants using a non-stretchable tape measure under the participant's clothes, with the participant in the upright position and the tape parallel to the floor.[19] The WC was measured at the midpoint position defined as the location of the umbilicus. Hip circumference was measured at the widest hip circumference (including the most protuberant buttock and lateral upper thigh).[19]

Patient and public involvement

This research was carried out with patients and community leaders. Community leaders and Botswana Harvard Partnership community advisory board (BHP CAB) members provided input during planning of the main BCPP trial, particularly in recruitment strategies and shaping study procedures to align with community norms. They also promoted the study within participating villages and ultimately assisted in sharing findings with the community through local gatherings. Post-trial dissemination was done on radio and BHP CAB meetings.

Central obesity definitions

Central obesity was defined according to the WHO[19] and International Diabetes Federation (IDF)[20] as a WHR >0.90 for males and >0.85 for females.

In addition, central obesity was also defined using WCs of ≥94 cm in males and ≥80 cm in females among sub-Saharan Africans as per a joint statement by the IDF Task Force on Epidemiology and Prevention, National Heart, Lung, and Blood Institute, American Heart Association Science Advisory and Coordinating Committee, World Heart Federation, International Atherosclerosis Society and International Association for the Study of Obesity.[21]

Statistical considerations

Normally distributed continuous characteristics were summarised by their mean and SD, while a median and its IQR are presented for skewed continuous data. A Student's t-test was used to assess the association between HIV status and continuous variables if normally distributed; otherwise, a Wilcoxon sum test was used for skewed data. To compare the covariates by HIV status, we used a χ^2 test or Fisher's exact test.

Prevalence and prevalence ratios (PRs) of the binary outcome of central obesity were derived from modified Poisson generalised estimating equations, along with corresponding Huber robust standard estimates and 95% Wald CIs. Of those taking part, 1942 (48.8%) had missing WC readings. To account for potential selection bias due to missing outcome data, we applied complete-case inverse probability weighting (IPW) to re-estimate the overall prevalence of central obesity and the corresponding PRs. Our approach adjusts for missing WC and WHR readings by accounting for potential correlates between missing WC and WHR reading and other demographic, clinical and social characteristics (see online supplemental tables). We also performed a sensitivity analysis using WC ≥102 cm for males and ≥88 cm for females, which represents a substantially increased risk of metabolic complications according to WHO recommendations.[19] Results with p values<0.05 were considered significant in all analyses. We used R statistical software, R V.4.3.2 (31 October 2023).[17]

RESULTS

Participant demographics

A total of 3981 participants were approached for central obesity assessment, and 2039 (51.2%) of them completed all the necessary assessment such that we could calculate WHR and WC measurements. The remaining 1942 (48.8%) declined to undergo physical measurements for WC and WHR for various reasons (figure 1 and online supplemental table 1).

Online supplemental table 1 provides a detailed comparison of those who agreed to complete the central obesity assessments versus those who did not. Those who agreed to complete the assessments were mostly female (66.8% vs 33.2%, p<0.001) and living without HIV (71.3% vs 28.7%, p=0.04), while those who refused tended to have reported higher levels of counselling for alcohol intake (30.4% vs 22.5%, p<0.001) and were more likely to report being screened for cholesterol (17.5% vs 9.7%, p<0.001) and diabetes (23.6% vs 18.6%, p<0.001), respectively.

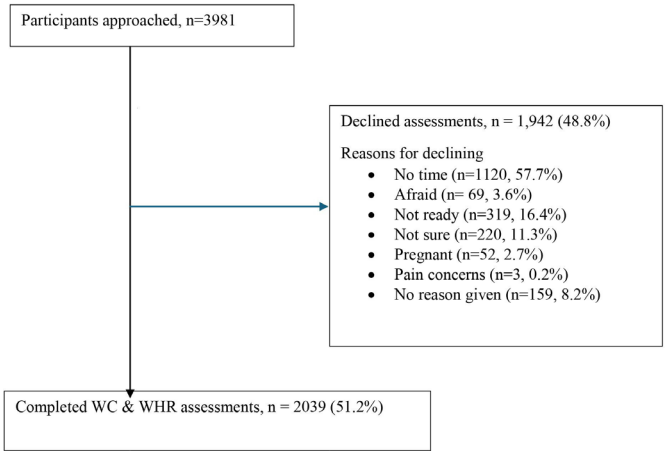

Figure 1 Participant flow diagram. WC, waist circumference; WHR, waist-to-hip ratio.

Table 1 shows the demographic and clinical characteristics of participants by HIV status. Of the 2039 participants who completed all required obesity assessments, 586 (28.7%) were living with HIV. PLWH were significantly older, with a median age of 42.0 years (IQR 35.0–49.9) vs 31.7 years (IQR 24.7–46.3) for those without HIV, p value<0.001. The majority of participants were female (66.8%).

Of the 586 PLWH, 553 (94.4%) were on ART (TDF-FTC-DTG), 28 (4.8%) were ART-naïve and 5 (0.9%) had stopped taking treatment. The CD4+ cell count was available for 163 participants, with a median CD4+ count of 560 cells/µL (IQR 411–745).

Central obesity prevalence: overall, HIV status and sex group

The overall prevalence of central obesity was 43.5% as defined by the WHR and 50.8% as defined by WC (table 2). The central obesity prevalence was higher among females than males, 46.9% vs 36.7% (WHR) and 68.5% vs 15.1% (WC), and was associated with increasing age, evidenced by an increase of approximately 10% with each decade of life between 18 and 68 years of age (see figure 2).

Factors associated with central obesity

In the unadjusted analysis, PLWH had a higher prevalence of central obesity defined by the WHR (47.8% (95% CI% 43.7 to 51.8%)) compared with those without HIV (41.8% (95% CI 39.2% to 44.3%)) (table 2), p value 0.02. After fully adjusting for age, sex and missing waist and hip measurements, this trend was attenuated, and there was no significant difference in the prevalence of central obesity defined by the WHR between PLWH and those without HIV (adjusted prevalence ratio (aPR) 0.99 (95% CI 0.90 to 1.09, p value 0.88)) (table 3).

Similarly, in the unadjusted analysis of the prevalence of central obesity defined by WC, PLWH had a higher prevalence of central obesity (55.3% (95% CI 51.3% to 59.3%)) compared with people living without HIV (48.9% (95% CI 46.4% to 51.5%)), p value 0.01 (table 2). However, fully adjusted analysis (table 3) revealed there was no significant difference in the prevalence of central obesity defined by the WC between PLWH and those without HIV (aPR 0.93 (95% CI 0.85 to 1.01), p value 0.06).

Given that the proportion of females is different between PLWH and people without HIV (table 1), and the waist values are different between males and females, we performed a sex-stratified analysis of central obesity by HIV status. This revealed that the aPR of central obesity defined by WC was lower for PLWH than those living without HIV in both males and females (table 4).

Table 1 Demographic and clinical characteristics of the participants

Characteristic		Overall	People living without HIV	PLWH	
	N	2039	1453	586	P value*
Age	Median (IQR)	35.4 (26.4– 48.3)	31.7 (24.7–46.3)	42.0 (35.0–49.9)	<0.001
Age group (years)	16–24	399 (19.6%)	376 (25.9%)	23 (3.9%)	
	25–34	590 (28.9%)	472 (32.5%)	118 (20.1%)	
	35–44	430 (21.1%)	220 (15.1%)	210 (35.8%)	
	45–54	307 (15.1%)	158 (10.9%)	149 (25.4%)	
	55–68	313 (15.4%)	227 (15.6%)	86 (14.7%)	
Sex	Female	1363 (66.8%)	917 (63.1%)	446 (76.1%)	<0.001
Education level	Non-formal	222 (10.9%)	143 (9.8%)	79 (13.5%)	<0.001
	Primary	421 (20.6%)	242 (16.7%)	179 (30.5%)	
	Junior secondary	744 (36.5%)	502 (34.5%)	242 (41.3%)	
	Senior secondary	340 (16.7%)	291 (20%)	49 (8.4%)	
	Higher than secondary	304 (14.9%)	271 (18.7%)	33 (5.6%)	
	Missing	8 (0%)	4 (0%)	4 (0%)	
Smoking: ever	Yes	254 (12.5%)	171 (11.8%)	83 (14.2%)	0.159
SBP (mm Hg)	Median (IQR)	119 (109–130)	119 (110–130)	118 (109–128)	0.043
DBP (mm Hg)	Median (IQR)	80 (73, 88)	80 (72–88)	80 (73–88)	0.826
Hypertension meds: ever	Yes	191 (9.4%)	134 (9.2%)	57 (9.7%)	0.787
WHR: females	Median (IQR)	0.84 (0.79–0.91)	0.84 (0.78–0.91)	0.85 (0.80–0.92)	
WHR: males	Median (IQR)	0.87 (0.81–0.93)	0.86 (0.81–1.34)	0.88 (0.83–0.94)	
WC: females (cm)	Median (IQR)	87.0 (76.5–97)	87.0 (76–129)	86.4 (77.1–95)	
WC: males (cm)	Median (IQR)	78.3 (72.5–88)	78.6 (72–88.3)	78.3 (73.8–86.1)	
Current CD4+ count (cells/µL)	Median (IQR)	–	–	560 (411–745)	
Current ART status	N	–	–	586	
	ART-naïve	–	–	28 (5%)	
	Defaulted ART	–	–	5 (1%)	
	On ART	–	–	553 (94%)	

ART, antiretroviral therapy; DBP, diastolic blood pressure; SBP, systolic blood pressure; WC, waist circumference; WHR, waist-to-hip ratio.

Table 2 Prevalence of central obesity overall and by sex-stratified and HIV-stratified groups

		Central obesity defined by WHR			Central obesity defined by WC		
	N	Prevalence, N (%)	95% CI	P value	Prevalence, N (%)	95% CI	P value
Overall	2039	887 (43.5)	(41.4, 45.7)		1035 (50.8)	(48.6, 52.9)	
PLWH	586	280 (47.8)	(43.7, 51.8)	0.02	324 (55.3)	(51.3, 59.3)	0.01
Without HIV	1453	607 (41.8)	(39.2, 44.3)		711 (48.9)	(46.4, 51.5)	
Female	1363	639 (46.9)	(44.2, 49.5)		933 (68.5)	(65.9, 70.9)	
PLWH	446	225 (50.4)	(45.8, 55.1)	0.08	305 (68.4)	(63.9, 72.5)	> 0.99
Without HIV	917	414 (45.1)	(42, 48.3)		628 (68.5)	(66, 70.9)	
Male	676	248 (36.7)	(33.1, 40.4)		102 (15.1)	(12.4, 17.8)	
PLWH	140	55 (39.3)	(31.6, 47.6)	0.54	19 (13.6)	(8.9, 20.2)	0.67
Without HIV	536	193 (36)	(32.1, 40.2)		83 (15.5)	(12.7, 18.8)	

PLWH, people living with HIV; WC, waist circumference; WHR, waist-to-hip ratio.

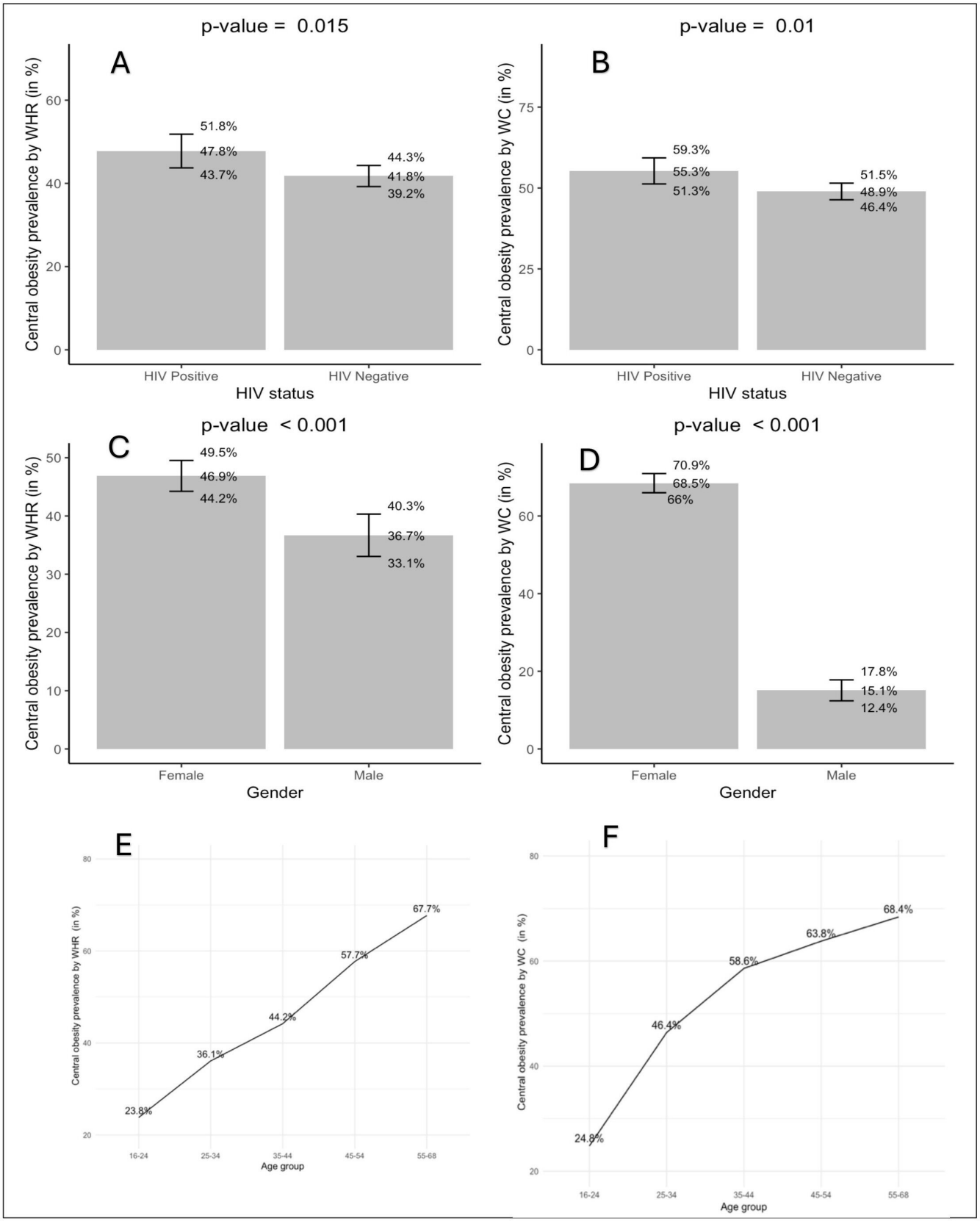

Figure 2 Sex, HIV status and age group-specific prevalences of central obesity defined by waist circumference (WC) and the waist-to-hip ratio (WHR) criteria: central obesity defined as a WC>80 cm or a WHR>0.85 for females and a WC>94 cm or a WHR>0.90 for males. (A) and (B) show central obesity prevalence by WHR and WC, respectively, stratified by HIV status. (C) and (D) show central obesity prevalence by WHR and WC, respectively, stratified by gender. (E) and (F) display the age-specific prevalence of central obesity by WC and WHR, respectively. Prevalence point estimates are presented together with 95% CIs.

Table 3 Unadjusted and adjusted prevalence ratios of central obesity by HIV status

	Waist-to-hip ratio	P value	Waist circumference	P value
Unadjusted	1.14 (0.99, 1.32)	0.01	1.13 (0.99,1.29)	0.01
Adjusted*	1.01 (0.86, 1.16)	0.96	0.94 (0.82, 1.08)	0.14
Fully adjusted†	0.99 (0.90, 1.09)	0.88	0.93 (0.85, 1.01)	0.06

*Model adjusted for age and sex.
†Model adjusted for missing data on waist circumference and hip measurements using inverse-probability weighting and controlled for sex and age.

Sensitivity analysis using cut-offs ≥102 cm in males and ≥88cm in females

In unadjusted analysis, PLWH had a trend towards higher prevalence of central obesity by WHR, 47.8% (95% CI 43.7% to 51.8%) compared with those living without HIV, 41.6% (95% CI 37.7% to 45.7%) (online supplemental table 3). However, after fully adjusting for age, sex and missing waist and hip measurements, there was no significant difference in the prevalence of central obesity by WHR between PLWH and people living without HIV (online supplemental table 2).

Both the unadjusted (online supplemental table 3) and fully adjusted analysis (online supplemental table 4) showed no significant difference in the prevalence of central obesity by WC with respect to HIV status.

DISCUSSION

This cross-sectional study in a large sample of adults residing in 22 rural and peri-urban communities in Botswana found a high prevalence of central obesity using both the WHR and WC measurements, particularly in females and in older adults. There was no difference in the prevalence of central obesity by HIV status. Consistent with previous studies,[22] a higher prevalence was found in older adults and females than younger adults and males.

Defining thresholds for abdominal obesity is complicated by the varying relationships between abdominal obesity and other metabolic risk factors. Additionally, these relationships differ across sexes and ethnic groups. Consequently, different organisations have established different thresholds for defining central obesity, particularly when using WC. For instance, the WHO[19] defines two levels of central obesity based on risk of metabolic complication, with different WC cut-offs for increased and substantially increased risk. Therefore, our cut-offs follow the recommendations for sub-Saharan Africans, and our comparisons are based on this framework.

Our overall prevalence of central obesity defined by the WHR, 43.5% (46.9% females, 36.7% males), was almost similar to two previous local (Botswana) studies by Omech *et al*[23] and Mosepele *et al*,[24] with prevalences of 48.8% (51.9% females, 34.7% males), and 49% in females, 33% in males, respectively. A study in Uganda[25] also reported an almost similar prevalence of 40.6% (48.7% females, 26.5% males). On the other hand, a study in South Africa[26] reported a higher prevalence of 57.8% (60.7% females, 51.7% males). Of note, the South African study[26] had an older population (mean age of 42.6 years) than that of our study (median age 35.3 years), and was facility-based, while our population was community-based, meaning that their participants could have already had chronic illnesses, including obesity, bringing them to health facilities. Differences in the socio-economic status of the studied populations and the associated unhealthy lifestyle and central obesity may also partly explain the higher obesity prevalence in South Africa compared with Botswana.

Our overall high central obesity prevalence by WC, 50.8%, was similar to a large global systematic review and meta-analysis, which reported a prevalence of 49.6% among Africans,[22] but was higher than a pooled analysis of Sub-Saharan Africa (SSA) studies that reported a prevalence of 35% using similar WC (≥94 cm in males, ≥80 cm in females) cut-offs.[22] A large study from Burkina-Faso[27] also reported a lower prevalence (22.5%) than our study using the same WC cut-offs. Possible reasons for this

Table 4 Sex-stratified analysis of central obesity by HIV status

Waist hip ratio for PLWH vs those living without HIV			Waist circumference for PLWH vs those living without HIV	
	Females		Females	
	aPR	P value	aPR	P value
Fully adjusted model	1.03 (0.92, 1.15)	0.606	0.55 (0.38, 0.78)	0.017
	Males		Males	
Fully adjusted model	0.84 (0.67, 1.04)	0.130	0.87 (0.80, 0.96)	0.017

Model adjusted for missing data on waist circumference and hip measurements using inverse-probability weighting and controlled for age.
aPR, adjusted prevalence ratio; PLWH, people living with HIV.

include a lower proportion of females in the Burkina-Faso study (52.4%) compared with ours, a low prevalence of overweight/obesity in Burkina-Faso (18.7%)[28] than Botswana (47.3%),[29] due to their low socio-economic status. Other unmeasured confounders in our study such as level of physical activity and diet may also account for these differences.

Using a WC cut-off ≥102 cm for males and ≥88 cm for females,[19] we established a prevalence of 32.9% (46.1% females, 6.1% males), which was similar to a global systematic review and meta-analysis of 13.2 million subjects (using this same cut-off), including African participants, which reported a prevalence of 35.9% (95% CI 33.6% to 38.3%).[22] Meanwhile, three cross-sectional studies in Tanzania,[30] n=454, Burkina-Faso,[27] n=4308, Uganda,[25] n=1962, and a population-based survey in Uganda,[31] n=3676, reported lower prevalence of 16.9%, 10.2% (16.9% females, 1.6% males), 15.2% (14.8% females, 0.4% males) and 11.8% (19.5% females, 1.3% males), respectively. Our higher WC prevalence compared with the two Ugandan studies[25 31] may be attributable to similar reasons outlined for WHR above, while the difference with studies in Burkina-Faso[27] and Tanzanian[30] could partly be due to relatively higher physical activity rates, as well as low socio-economic status in both of these as low-income countries, compared with Botswana.[32] The differences in the central obesity prevalence by the two criteria highlight the high sensitivity of the lower cut-offs, which, however, has a low specificity for adults at a substantial increased risk of metabolic complications.[19]

Our study reported a prevalence of central obesity defined by the WHR among PLWH of 47.7% (50.4% females, 39.2% males), which was comparable to studies in Uganda[25] and the prior local study by Mosepele *et al*,[24] with prevalences of 42% (48% females, 31% males), and 49% females, 33% males, respectively.

We reported the prevalence of central obesity defined by WC among PLWH to be 55.3% (68.4% females, 13.6% males), which was comparable to some studies with PLWH on ART in Tanzania[30] and Ethiopia,[33] with a prevalence of 48.0% and 52.7%, respectively. On the other hand, a study from South Africa[34] reported higher rates than us, 64.2%, among PLWH on ART. The possible reasons for a high prevalence in the South African study[34] than ours was a high proportion of females in their study, 87.2%, than our study. Meanwhile, an Ethiopian study[35] reported a slightly lower prevalence than us (41.3%).

Using a WC cut-off ≥102 cm for males and ≥88 cm for females, we reported a 35.0% (46.1% females, 6.1% males) prevalence of central obesity by WC among PLWH. Some studies in Ethiopia,[36] Uganda[25] and Tanzania[30] of 268, 990 and 150 PLWH reported lower prevalence, 18.7% (17.9% females, 0.7% males), 12% (19% females, 1% males) and 25.3%, respectively, while those reporting higher prevalence included an Ethiopian study of 407 PLWH, 41.3%.[35] On the other hand, a study of 2230 PLWH from South Africa[37] reported similar rates of central obesity, 30.9% (44.6% females and 3.9% males), which was associated with an increasing age, just like in our current report.

Among studies reporting a lower prevalence than our study, we noted a small number of PLWH, ≤150, in the Tanzanian[30] and Ethiopian[36] studies compared with our study with 586 PLWH. High physical activity rates, >95%, being in a predominantly rural area and having a low prevalence of central obesity in the general population[31] in the Ugandan study[25] may also account for differences with our study. A low socio-economic status across all the above three countries compared with Botswana[32] may also contribute to their lower prevalence than our study.

Consistent with our current study, there was no significant difference in the WHR between PLWH and those without HIV in both the previous Botswana study[24] and the Ugandan study.[25] Furthermore, while we reported no difference in WC between PLWH and those without HIV in our study, the Tanzanian[30] and South African[34] studies reported higher central obesity rates by WC among PLWH on ART compared with ART-naïve PLWH and those without HIV. Of note, there was a gender imbalance in both these studies, with the PLWH on ART group having a significantly greater proportion of females than the other two groups.

The prevalence of central obesity increased with age and was higher in females than males. Previous studies have reported that older adults have a low basal metabolic rate than young adults, contributing to excess fat accumulation.[38 39] Ageing is also linked with fat redistribution from subcutaneous areas to the abdominal region.[40] In women, menopause contributes to an increase in truncal obesity and visceral fat deposition.[41] Furthermore, the high central obesity prevalence in females than males has been attributed to several factors, including socio-cultural factors such as the perception of overweight as a sign of good health, beauty or affluence, particularly among females, in some black African communities, whereas thinness may be associated with advanced HIV disease.[42–44] Other reasons include low education levels and its association with housework and a sedentary lifestyle.[45] Additionally, central obesity is associated with low testosterone levels as the hormone promotes fat consumption and reduces central obesity.[46]

The high prevalence of central obesity in our study is a concerning public health challenge as it represents a high risk of cardiometabolic disease that clinicians and policymakers must address so as to prevent or delay progression to disease. Given the resource constraint in our setting and SSA in general, there is a need to use low-cost tools to identify individuals at increased risk of cardiovascular disease and strengthen interventions on healthy lifestyle modifications in those at highest risk, females and older adults. A comparable prevalence between PLWH and the general population could be due to good HIV control by ART resulting in the return to health phenomenon; however, considering that a subset of BCPP participants started ART within 5 years of BCPP, they were few (n=586) and without baseline/serial weight obtained in the home

setting, the duration of ART may have been insufficient to cause significant weight gain and central obesity. Thus, the comparable prevalences may reflect population-level lifestyle factors such as unhealthy dietary habits and insufficient physical activity levels.

Strengths and limitations of the study

To our knowledge, this is the first community-based study to directly measure central obesity by HIV status in Botswana overall and by two criteria for central obesity. Our study had several limitations; we had a high refusal rate for the central obesity assessment among BCPP survey participants, with 49% of approached individuals declining obesity measurements, which may introduce selection bias. Since our substudy was nested within an HIV prevention project, participant motivation for involvement in the main project may not have been carried over to this central obesity substudy, as shown by the reasons for refusal given in figure 1. Females were more likely to provide consent for waist and hip measurements than males. To address this potential selection bias, complete case IPW methods were used to adjust PRs for missing measurements. In controlling for this potential bias, our results were grossly unchanged, and we arrived at the same conclusions that were generalisable to this population despite the missing data. Another limitation was the use of a tape measure, which is prone to measurement errors, to assess WC and the WHR. More accurate techniques, such as CT or MRI,[47] were impractical for our community-based study. Assessing central obesity at one point in time prevented establishing a temporal relationship among HIV, ART duration and central obesity. Additionally, we did not assess other potential confounders such as dietary habits and physical activity levels. We also did not assess the different ART combinations and their association with central obesity or lipodystrophy. Finally, only 28.7% (586) of our participants were PLWH, with male under-representation at 24%. This could have biased results in favour of female people living without HIV.

In conclusion, our study highlights the significant burden of central obesity in both PLWH and people without HIV in rural and peri-urban communities in Botswana. Of note, over two-thirds of adult females in Botswana had central obesity; however, living with HIV was not significantly associated with central obesity by WC or WHR. Future prospective research should investigate anthropometric measures and their associations with health outcomes by HIV status and the acceptability of such measures for use given the high rate of refusal in our study population. Evidence-based interventions are required to reduce the prevalence of central obesity and the associated risks to health and well-being.

Author affiliations
[1]Department of Internal Medicine, University of Botswana Faculty of Medicine, Gaborone, Botswana
[2]Department of Statistics, University of Botswana, Gaborone, Botswana
[3]Botswana Harvard Health Partnership, Gaborone, Botswana
[4]Department of Family Medicine and Public Health, University of Botswana Faculty of Medicine, Gaborone, Botswana
[5]Brigham and Women's Hospital, Boston, Massachusetts, USA
[6]Infectious Disease, London School of Hygiene & Tropical Medicine, London, UK
[7]Immunology & Infectious Diseases, Harvard T H Chan School of Public Health, Boston, Massachusetts, USA

Acknowledgements We would like to thank all our study participants, healthcare workers and community leaders who assisted us in conducting this community-based central obesity assessment. We are also grateful to the Centers for Disease Control (CDC), USA, Harvard T. H. Chan School of Public Health, PEPFAR and NIH RCRE (P20CA210283) for their support in conducting this study.

Contributors Conceptualisation: MMo, SL and CGW. Methodology: MMo and TG. Formal analysis: MMa and KMo. Investigation: KMa. Writing—original draft preparation: TMo, CGW and MMo. Writing—review and editing: TMo, MMo, MMa, AL and OJM-B. Supervision and funding: MMo. All authors have read and agreed to the published version of the manuscript. MMo is the guarantor.

Funding The parent study was supported by the President's Emergency Plan for AIDS Relief (PEPFAR) through the Centers for Disease Control and Prevention (CDC) (cooperative agreements U01 GH000447 and U2G GH001911). The funding agencies had no role in the design, decision to publish, or preparation of the manuscript. The contents of this article are solely the responsibility of the authors and do not necessarily represent the official views of the funder.

Competing interests None declared.

Patient and public involvement Patients and/or the public were involved in the design, or conduct, or reporting, or dissemination plans of this research. Refer to the Methods section for further details.

Patient consent for publication Not applicable.

Ethics approval This study involves human participants and was approved by the Botswana Ministry of Health Human Research Development Committee (HRDC), Gaborone, Botswana HPDME 13/18/1 X12. Centers for Disease Control (CDC), Atlanta, USA. Participants gave informed consent to participate in the study before taking part.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Data are available upon reasonable request.

ORCID iDs
Tiny Masupe http://orcid.org/0000-0001-8366-6585
Onkabetse Julia Molefe-Baikai http://orcid.org/0000-0002-3371-4611
Mosepele Mosepele http://orcid.org/0009-0008-5089-7449

REFERENCES

1 Afshin A, Forouzanfar MH, Reitsma MB, *et al*. Health effects of overweight and obesity in 195 countries over 25 years. *N Engl J Med* 2017;377:13–27.
2 Sahakyan KR, Somers VK, Rodriguez-Escudero JP, *et al*. Normal-weight central obesity: implications for total and cardiovascular mortality. *Ann Intern Med* 2015;163:827–35.
3 Powell-Wiley TM, Poirier P, Burke LE, *et al*. Obesity and cardiovascular disease: a scientific statement from the american heart association. *Circulation* 2021;143:e984–1010.

4 Lee CMY, Huxley RR, Wildman RP, *et al*. Indices of abdominal obesity are better discriminators of cardiovascular risk factors than BMI: a meta-analysis. *J Clin Epidemiol* 2008;61:646–53.
5 Janssen I, Katzmarzyk PT, Ross R. Waist circumference and not body mass index explains obesity-related health risk. *Am J Clin Nutr* 2004;79:379–84.
6 Barberio AM, Alareeki A, Viner B, *et al*. Central body fatness is a stronger predictor of cancer risk than overall body size. *Nat Commun* 2019;10:383.
7 Bailin SS, Gabriel CL, Wanjalla CN, *et al*. Obesity and weight gain in persons with HIV. *Curr HIV/AIDS Rep* 2020;17:138–50.
8 van der Kooy K, Leenen R, Seidell JC, *et al*. Abdominal diameters as indicators of visceral fat: comparison between magnetic resonance imaging and anthropometry. *Br J Nutr* 1993;70:47–58.
9 Piqueras P, Ballester A, Durá-Gil JV, *et al*. Anthropometric indicators as a tool for diagnosis of obesity and other health risk factors: a literature review. *Front Psychol* 2021;12:631179.
10 Lake JE. The fat of the matter: obesity and visceral adiposity in treated HIV infection. *Curr HIV/AIDS Rep* 2017;14:211–9.
11 Bakal DR, Coelho LE, Luz PM, *et al*. Obesity following ART initiation is common and influenced by both traditional and HIV-/ART-specific risk factors. *J Antimicrob Chemother* 2018;73:2177–85.
12 Lake JE, Trevillyan J. Impact of Integrase inhibitors and tenofovir alafenamide on weight gain in people with HIV. *Curr Opin HIV AIDS* 2021;16:148–51.
13 Venter WDF, Moorhouse M, Sokhela S, *et al*. Dolutegravir plus two different prodrugs of tenofovir to treat HIV. *N Engl J Med* 2019;381:803–15.
14 Ministry of Health and Wellness. Handbook of the Botswana 2016 integrated HIV clinical care guideline. Republic of Botswana, 2016. Available: www.moh.gov.bw/Publications/Handbook_HIV_treatment_guidelines.pdf
15 Todowede OO, Mianda SZ, Sartorius B. Prevalence of metabolic syndrome among HIV-positive and HIV-negative populations in sub-Saharan Africa-a systematic review and meta-analysis. *Syst Rev* 2019;8:4.
16 Girma D, Dejene H, Geleta LA, *et al*. Metabolic syndrome among people living with HIV in Ethiopia: a systematic review and meta-analysis. *Diabetol Metab Syndr* 2023;15:61.
17 Makhema J, Wirth KE, Pretorius Holme M, *et al*. Universal testing, expanded treatment, and incidence of HIV infection in Botswana. *N Engl J Med* 2019;381:230–42.
18 Gaolathe T, Wirth KE, Holme MP, *et al*. Botswana's progress toward achieving the 2020 UNAIDS 90-90-90 antiretroviral therapy and virological suppression goals: a population-based survey. *Lancet HIV* 2016;3:e221–30.
19 WHO. Waist circumference and waist-hip ratio. Report of a World Health Organisation expert consultation, Geneva, 8-11 December 2008, Available: https://www.who.int/nutrition/publications/obesity/WHO_report_waistcircumference_and_waisthip_ratio/en
20 Alberti K, Zimmet P, Shaw J. Metabolic syndrome--a new world-wide definition. A consensus statement from the international diabetes federation. *Diabet Med* 2006;23:469–80.
21 Alberti KGMM, Eckel RH, Grundy SM, *et al*. Harmonizing the metabolic syndrome. *Circulation* 2009;120:1640–5.
22 Wong MCS, Huang J, Wang J, *et al*. Global, regional and time-trend prevalence of central obesity: a systematic review and meta-analysis of 13.2 million subjects. *Eur J Epidemiol* 2020;35:673–83.
23 Omech B, Tshikuka J-G, Mwita JC, *et al*. Prevalence and determinants of metabolic syndrome: a cross-sectional survey of general medical outpatient clinics using National Cholesterol Education Program-Adult Treatment Panel III criteria in Botswana. *Diabetes Metab Syndr Obes* 2016;9:273–9.
24 Mosepele M, Hemphill LC, Moloi W, *et al*. Pre-clinical carotid atherosclerosis and sCD163 among virally suppressed HIV patients in Botswana compared with uninfected controls. *PLoS One* 2017;12:e0179994.
25 Enriquez R, Ssekubugu R, Ndyanabo A, *et al*. Prevalence of cardiovascular risk factors by HIV status in a population-based cohort in South Central Uganda: a cross-sectional survey. *J Int AIDS Soc* 2022;25:e25901.
26 Owolabi EO, Ter Goon D, Adeniyi OV. Central obesity and normal-weight central obesity among adults attending healthcare facilities in Buffalo City Metropolitan Municipality, South Africa: a cross-sectional study. *J Health Popul Nutr* 2017;36:54.
27 Cisse K, Samadoulougou S, Ouedraogo M, *et al*. Prevalence of abdominal obesity and its association with cardiovascular risk among the adult population in Burkina Faso: findings from a nationwide cross-sectional study. *BMJ Open* 2021;11:e049496.
28 Kaboré S, Millogo T, Soubeiga JK, *et al*. Prevalence and risk factors for overweight and obesity: a cross-sectional countrywide study in Burkina Faso. *BMJ Open* 2020;10:e032953.
29 Letamo G. Dual burden of underweight and overweight/obesity among adults in Botswana: prevalence, trends and sociodemographic correlates: a cross-sectional survey. *BMJ Open* 2020;10:e038614.
30 Kingery JR, Alfred Y, Smart LR, *et al*. Short-term and long-term cardiovascular risk, metabolic syndrome and HIV in Tanzania. *Heart* 2016;102:1200–5.
31 Kabwama SN, Kirunda B, Mutungi G, *et al*. Prevalence and correlates of abdominal obesity among adults in Uganda: findings from a national cross-sectional, population based survey 2014. *BMC Obes* 2018;5:40.
32 World Bank. World Bank Open Data, 2022. Available: https://data.worldbank.org
33 Tesfaye DY, Kinde S, Medhin G, *et al*. Burden of metabolic syndrome among HIV-infected patients in Southern Ethiopia. *Diabet Metab Synd Clin Res Rev* 2014;8:102–7.
34 Awotedu K, Ekpebegh C, Longo-Mbenza B, *et al*. Prevalence of metabolic syndrome assessed by IDF and NCEP ATP 111 criteria and determinants of insulin resistance among HIV patients in the Eastern Cape Province of South Africa. *Diabet Metab Synd Clin Res Rev* 2010;4:210–4.
35 Gebrie A. The burden of metabolic syndrome in patients living with HIV/AIDS receiving care at referral hospitals of Northwest Ethiopia: A hospital-based cross-sectional study, 2019. *Diabet Metab Synd Clin Res Rev* 2020;14:1551–6.
36 Bosho DD, Dube L, Mega TA, *et al*. Prevalence and predictors of metabolic syndrome among people living with human immunodeficiency virus (PLWHIV). *Diabetol Metab Syndr* 2018;10:10.
37 Huis In 't Veld D, Pengpid S, Colebunders R, *et al*. Body mass index and waist circumference in patients with HIV in South Africa and associated socio-demographic, health related and psychosocial factors. *AIDS Behav* 2018;22:1972–86.
38 Visser M, Deurenberg P, van Staveren WA, *et al*. Resting metabolic rate and diet-induced thermogenesis in young and elderly subjects: relationship with body composition, fat distribution, and physical activity level. *Am J Clin Nutr* 1995;61:772–8.
39 Buscemi S, Verga S, Caimi G, *et al*. Low relative resting metabolic rate and body weight gain in adult Caucasian Italians. *Int J Obes* 2005;29:287–91.
40 Kuk JL, Saunders TJ, Davidson LE, *et al*. Age-related changes in total and regional fat distribution. *Ageing Res Rev* 2009;8:339–48.
41 Opoku AA, Abushama M, Konje JC. Obesity and menopause. *Best Pract Res Clin Obstet Gynaecol* 2023;88:102348.
42 Micklesfield LK, Lambert EV, Hume DJ, *et al*. Socio-cultural, environmental and behavioural determinants of obesity in black South African women. *Cardiovasc J Afr* 2013;24:369–75.
43 McCormick CL, Francis AM, Iliffe K, *et al*. Increasing obesity in treated female HIV patients from Sub-Saharan Africa: potential causes and possible targets for intervention. *Front Immunol* 2014;5:507.
44 Puoane T PH, Fourie JM, Shapiro M BSN, *et al*. 'Big is beautiful' – an exploration with urban black community health workers in a South African township. *South African Journal of Clinical Nutrition* 2005;18:6–15.
45 Azizi F, Salehi P, Etemadi A, *et al*. Prevalence of metabolic syndrome in an urban population: Tehran Lipid and Glucose Study. *Diabetes Res Clin Pract* 2003;61:29–37.
46 Laaksonen DE, Niskanen L, Punnonen K, *et al*. Sex hormones, inflammation and the metabolic syndrome: a population-based study. *Eur J Endocrinol* 2003;149:601–8.
47 Klein S, Allison DB, Heymsfield SB, *et al*. Waist circumference and cardiometabolic risk: a consensus statement from Shaping America's Health: Association for Weight Management and Obesity Prevention; NAASO, The Obesity Society; the American Society for Nutrition; and the American Diabetes Association. *Am J Clin Nutr* 2007;85:1197–202.