

Ethnic differences in resting metabolic rate, respiratory quotient and body temperature: a comparison of Africans and European Australians

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Abstract

Purpose A comparison of resting metabolic rate (RMR), respiratory quotient (RQ) and body temperature between adults of African and European descent.

Method Twenty-nine sub-Saharan Africans (SSA; 13 men and 16 women) and thirty-two Australians of European descent (EUR; eight men and 24 women) had RMR and RQ measured by indirect calorimetry. Dual-energy X-ray absorptiometry was used to determine fat mass (FM), fat-free mass, bone mineral content (BMC), appendicular lean tissue mass and non-appendicular lean tissue mass. Total skeletal muscle mass (SMM) was predicted. Residual mass (RM) was the difference between body weight and the sum of FM, SMM and BMC. The short form of the International Physical Activity Questionnaire was used to determine habitual physical activity (PA). Tympanic in the ear temperature (IET) and forearm to fingertip temperature gradients (FFG) were monitored throughout the protocol.

Results The unadjusted RMR of SSA was significantly lower compared to EUR. Adjusted for age, sex, season, PA, FM, BMC, SMM and RM, this difference in RMR was still evident (mean $\pm$ SE, SSA: 4880 ± 161 kJ/d vs. EUR: 5979 ± 111 , $P < 0.005$). The same model of adjustment also uncovered a significantly lower adjusted IET (SSA: $35.26^\circ\text{C} \pm 0.133$ vs. EUR: 35.60 ± 0.091 , $P < 0.05$), a higher adjusted RQ (SSA: 0.86 ± 0.014 vs. EUR: 0.83 ± 0.010 , $P < 0.05$) but no difference in adjusted FFG.

Conclusions In this study, SSA had a lower RMR, higher RQ and lower IET relative to EUR Australians.

Keywords African · Resting metabolic rate · Muscle mass · Obesity · Body temperature · Thermoneutral zone

Abbreviations

AA	African-Americans
AG ratio	Android-to-gynoid ratio
ASMI	Appendicular skeletal mass index
ALTM	Appendicular lean tissue mass
ATPase	Adenosine triphosphatase
BMC	Bone mineral content
BMI	Body mass index
DEXA	Dual-energy X-ray absorptiometry
EUR	Europeans
FFG	Forearm to fingertip gradient
FFM	Fat-free mass
FM	Fat mass
FMI	Fat mass index
%FM	Percentage fat mass
HR	Human research
IET	In the ear temperature
IPAQ	International Physical Activity Questionnaire
NALTM	Non-appendicular lean tissue mass
PA	Physical activity
QA	Quality assurance
QC	Quality control
RM	Residual mass
RMR	Resting metabolic rate
RQ	Respiratory quotient
SMM	Skeletal muscle mass
SNPs	Single nucleotide polymorphisms
SNS	Sympathetic nervous system
SSA	Sub-Saharan Africans

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TNZ Thermoneutral zone
UCP3 Uncoupling protein 3

Introduction

Obesity and related chronic disease are currently the most significant challenges facing public health. Like in other countries in the world, obesity is on the increase in sub-Saharan Africa. It was estimated that 20–50 percent of people residing in urban communities in Africa are either overweight or obese [1] and that the mortality rate from chronic diseases in sub-Saharan Africa will increase by 17 % within a decade if urgent measures are not put in place [1]. There are several metabolic predictors of weight gain, and they include a low relative resting metabolic rate (RMR), a high respiratory quotient (RQ), a low core body temperature and low levels of physical activity (PA) among other factors [2–4]. While these are not the sole reason for the current obesity crisis, they play a significant role in influencing weight gain.

An increase in fat mass (FM) results in an increase in RMR, but with excess FM (~40 %) RMR can decrease. This suggests a threshold effect of FM on RMR [5]. Fat-free mass (FFM), however, is the major determinant of RMR with a unit energy expenditure 3–7 times higher than FM. Within FFM, there are several tissues with varying rates of energy expenditure; the smaller weight organs (liver, lungs, brain, etc.) have a high metabolic rate per kg, while the larger compartment of skeletal muscle has a lesser unit rate of energy expenditure. Hence, when comparing groups which differ in body composition and particularly in the composition of FFM, it is imperative to have some handle on these compartments in order to arrive at meaningful conclusions on RMR [5–8]. It has been the observation of many that African-Americans (AA) had a lower adjusted RMR compared to white/European Americans [9–15]. However, other authors were of the opinion that this conclusion arose from the use of incomplete models of body composition [8, 16] and that there was no difference in RMR between AA and their white counterparts. It was important to clarify such views since small differences in energy balance may lead to long-term accrual of energy stores [17]. There were two complementary studies on ancestry and RMR. Fernandez et al. [18] found that African ancestry had a negative impact on RMR (slope of relationship = -1.78 kcal/d, $P = 0.093$), but this did not attain significance. More recently, Manini et al. [19] examined RMR of older AA and determined the contribution of European ancestry (EA) through single nucleotide polymorphisms (SNPs). European ancestry in AA was

significantly related to a higher RMR, and for every percent increase in EA, the model predicted an increase in the RMR of AA by 1.6 kcal/d.

The measurement of RQ in the fasted and postprandial state denotes the relative proportion of the type of fuel being utilized in that period. A high absolute RQ (closer to 1) is indicative of greater carbohydrate utilization, and 24-h RQ is a significant predictor of weight gain even on accounting for potential confounders [2, 3]. There are not many studies that have evaluated the RQ of AA in comparison with white individuals. Three studies reported that there was no difference in fasting RQ between these ethnic groups [20–22]. However, Nicklas et al. [10] found that fasting RQ was higher in AA compared to white postmenopausal women, and Weyer et al. [23] showed that a greater 24-h RQ in AA accounted for their significantly lower 24-h fat oxidation rate. Clearly, some additional evidence was needed in this area.

RMR is always measured in the thermoneutral zone (TNZ) so that energy production equals energy expended. Determining the TNZ for every individual is difficult since there are many factors that control thermoregulation [24, 25]. A potential way forward is to monitor skin surface temperature gradients (forearm to fingertip temperature gradients, FFG) during RMR measurements [25]. In essence, FFG reflects the peripheral vasoconstriction/vasodilation that serves to either conserve or dissipate heat [24, 26]. It follows that in the TNZ, FFG should not show net vasoconstriction or vasodilatation. It has been proposed for a while now that a lower core body temperature could be a major risk factor for weight gain in both animals and humans [4]. However, 24-h monitoring of core temperatures in lean and obese did not show any difference in body temperatures between these groups [27]. One explanation for these outcomes was that obesity is an adapted state, and any defects in core temperature may only be observed in the pre-obese condition [4]. Nevertheless, we felt it was important to monitor body temperature due to its close relationship to FFG and because a significant proportion of RMR, in both sedentary and active individuals, is directed toward maintaining homeothermy [4].

The primary aim of this study was to minimize the confounding by European ancestry by studying sub-Saharan Africans (SSA) with regard to their RMR and compartments of FFM. This has not been done to date, to the best of our knowledge. In addition, we also investigated the contribution of fasting RQ, monitored FFG as a marker of the thermoneutral state and recorded in the ear temperature (IET) as a surrogate for core body temperature. Our main hypothesis was that relative to EUR Australians, SSA would show at least a 160 kcal/d (~670 kJ/d) lower RMR after adjustment for various models of body composition.

Materials and methods

Sample size

Based on detecting an effect size of 0.40 in RMR, two groups (SSA vs. EUR), eight covariates, $\alpha = 0.05$ and power of 80 %, an analysis of covariance (ANCOVA) indicated the sample size required was $n = 52$ (GPower version 3.1.9.2) [28].

Participant selection

Twenty-nine SSA (13 males and 16 females) and thirty-two EUR adult residents (eight males and 24 females) participated in the study. Ancestry was determined through self-report as was done by other authors [9, 13, 15, 16, 19]. EUR confirmed that at least both parents were of European origin, and SSA confirmed both parents and four grandparents were of African origin. All Africans hailed from sub-Saharan Africa and had been resident in Perth between 1 and 15 years. Inclusion criteria included aged between 25 and 70 years, non-smokers, not consuming more than two standard alcohol drinks per day, a stable weight for at least 6 months (<2 kg fluctuation), non-pregnant, normal menstrual cycle or postmenopause. All selected participants also had no reported metabolic disorders that interfered with their metabolic rate, such as type 2 diabetes, thyroid disease, kidney disease, hypertension, and were not taking weight loss pills or hormone replacement therapy. The study was approved by the Human Research Ethics Committee of Curtin University via approval numbers HR 103/2012 and HR 108/2014. All participants signed an informed consent form prior to participation in the study.

Resting metabolic rate

The protocol consisted of two visits to the laboratory. On the first visit, participants were orientated to the laboratory setting, equipment and the procedure to allay anxiety on the day of data collection. RMR was measured on their second visit, between 7 a.m. and 9 a.m. under standard conditions of an overnight fast of 10–12 h, prior abstinence from vigorous physical activity for 36 h and sleep of at least 8 h prior to the measurements. All participants were provided with a low-fat, low-calcium frozen dinner meal of their choice, to minimize effects of uncontrolled dinner on next morning's nutrient oxidation [29]. The commercial dinner meals we offered were similar in energy content. Participants were instructed to eat till they were comfortably full, and any unused portions were returned the next morning to the investigators.

On arrival at the laboratory in the morning, volunteers emptied their bladder and changed into a cotton gown.

All clothing worn by each participant was standardized to within 0.5 clo. They were provided with a gown (~ 0.46 clo) for their body size, females wore their own bra and underwear (~ 0.04 clo), and males wore their own briefs (~ 0.04 clo). Following anthropometric measurements, an iButton (Dallas semiconductor R, Maxim, CA, USA) for recording skin surface temperature was secured to the middle of their forearm and on the tip of their middle finger with Fixomull tape. The forearm to fingertip skin temperature gradient (FFG) was used as an index of peripheral vasoconstriction/vasodilation [26]. Participants then entered an environmental chamber set to 25 °C the previous evening. The chamber is a purpose-built structure within a large room of the building. It has a volume of 57.75 m³, an insulated roof and walls and is independently controlled for a temperature range from 4 to 50 °C.

Following a mandatory rest in bed at 25 °C for 30 min in the supine position, oxygen consumption and carbon dioxide production were monitored over a further 30 min by a canopy-based indirect calorimeter (Deltatrac II, Datex, Finland). RMR was calculated from the last 25 min of data using the Weir equation [30]. The continuous data from iButtons were extracted for the same 25 min of RMR recordings and used for calculating FFG. We did not have a gold-standard method for core temperature and instead used in the ear tympanic temperatures (IET, Omron, model MC-510, Japan). While the method is considered valid in comparison with more invasive techniques for core body temperature [31–33], there are studies that do not support this conclusion [34, 35]. However, IET is very practical and we provide an assessment of its repeatability in our hands. Duplicate recordings of IET were made on entering the chamber, before measurement of RMR and soon after the end of RMR measurement. The value of IET used for analysis was the average of two readings just before placing the canopy of the Deltatrac II over participant's head. On completion of the trial, volunteers were escorted to another building on campus for a dual-energy X-ray absorptiometry measurement (DEXA, Prodigy™, Lunar Corp. Madison USA). DEXA provided an assessment of FM, lean tissue mass (LTM) and bone mineral content (BMC) for the whole body and for each arm and leg. Whole-body LTM plus whole-body BMC gave fat-free mass (FFM). The sum of LTM of each arm and leg was used to calculate appendicular lean tissue mass (ALTM). Whole-body LTM minus ALTM provided non-appendicular lean tissue mass (NALTM). Total body skeletal muscle mass (SMM) was predicted from a mixed race equation of Kim et al. [36] as follows:

$$\text{SMM(kg)} = [(1.13 \times \text{ALTM}) - (0.02 \times \text{age}) + (0.61 \times \text{sex}) + 0.97] \quad (1)$$

Table 1 Body composition compartments of Europeans and sub-Saharan Africans

Variable	Europeans (<i>n</i> = 32)	Sub-Saharan Africans (<i>n</i> = 29)
Age (year)	47 ± 18.2	35 ± 6.2 [†]
BMI (kg/m ²)	27.3 ± 4.8	25.3 ± 4.8
Physical activity (MET min/week)	3710 ± 4626	2954 ± 3627
FFM (kg)	46.9 ± 9.2	50.3 ± 10.3
FM (kg)	29.8 ± 11.7	22.8 ± 9.8**
%FM	38.9 ± 8.8	31.3 ± 0.1 [†]
Waist (cm)	92.0 ± 13.1	82.7 ± 19.8**
AG ratio	1.0 ± 0.2	1.0 ± 0.4
BMC (kg)	2.6 ± 0.4	2.7 ± 0.5
LTM (kg)	44.3 ± 8.8	48.3 ± 10.3
ALTM (kg)	19.4 ± 4.5	21.7 ± 5.3
SMM (kg)	22.1 ± 5.42	25.1 ± 6.31*
NALTM (kg)	24.9 ± 4.8	26.6 ± 5.7
RM (kg)	22.3 ± 4.13	22.6 ± 3.84
FMI (kg/m ²)	10.5 ± 4.0	8.0 ± 3.6**
ASMI (kg/m ²)	6.8 ± 1.2	7.5 ± 1.5**

BMI body mass index, *FFM* fat-free mass, *FM* fat mass, *%FM* percentage fat mass, *AG ratio* android/gynoid ratio, *BMC* bone mineral content, *LTM* lean tissue mass, *ALTM* appendicular lean tissue mass, *SMM* skeletal muscle mass predicted from equation of Kim et al. [11], *NALTM* non-appendicular lean tissue mass, *RM* residual mass, *FMI* fat mass index, *ASMI* appendicular skeletal muscle index

Data are mean ± SD. Independent *t* test: * *P* = 0.051; ** *P* < 0.05 [†] *P* < 0.005

Residual mass (RM) was calculated as follows [13, 15]:

$$\text{RM(kg)} = \text{body weight} - (\text{FM} + \text{SMM} + \text{BMC}) \quad (2)$$

Appendicular skeletal muscle mass index (ASMI) and fat mass index (FMI) were calculated as the ratio of ALTM/height² (kg/m²) and FM/height² (kg/m²), respectively [37]. Participants also completed the short version of the self-administered International Physical Activity Questionnaire (IPAQ) [38], which inquired about their level and duration of activity over the last 7 days. Data were analyzed as per the guidelines of IPAQ, to determine their physical activity levels (Table 1). All participants were served a light brunch before they left the premises.

The Deltatrac II machine was calibrated every morning with a mixture of gases (95 % oxygen and 5 % carbon dioxide). Combustion of absolute alcohol has a theoretical RQ of 0.66. We conducted alcohol burns throughout the study period to validate the performance of the oxygen and carbon dioxide sensors. The mean RQ (±SD) of 13 burns was 0.66 ± 0.012, with a CV of 1.88 %. The DEXA machine underwent a mandatory quality assurance (QA) and quality control (QC) run every morning of its operation.

Repeatability trial

Ten participants (six SSA and four EUR) repeated the entire trial on a second visit between 2 and 12 weeks of the first.

Data analysis

All analyses were performed using the Statistical Package for Social Science (SPSS) for Windows software (version 22; SPSS Inc., Chicago, IL, USA). A two-tailed *P* value <0.05 was used for statistical significance. For comparison of metabolic variables in SSA and EUR, multivariate linear regression was employed with and without adjusting for covariates. Initial analyses used a complete model that included all possible two-way interactions. We did not observe any significant interactions for any outcome variable tested. The models reported here include the main effect of ethnicity after adjustment for covariates as per the model. Data from the repeatability trial were analyzed by paired *t* tests. Within-subject error was calculated for the total group (*n* = 10), and for each ethnic group, as the standard deviation of the difference score divided by $\sqrt{2}$ [39].

Results

The demographic, anthropometric and body composition data are presented in Table 1. SSA were younger by 12 years and had significantly lower fat mass, %FM and waist circumferences. Predicted SMM and ASMI were higher in SSA compared to EUR (Table 1). There were large variations in physical activity in both groups, but total physical activity in MET min/week was similar between

Table 2 Within-subject variation in body composition and metabolic measurements in this study

Variable	Visit 1	Visit 2	Difference	Paired <i>t</i> test	Typical error ^a
Weight (kg)	65.8 ± 22.10	65.6 ± 22.30	0.2 ± 1.06	<i>P</i> = 0.557	±0.75
FM (kg)	18.6 ± 16.35	19.0 ± 17.78	−0.34 ± 1.64	<i>P</i> = 0.523	±1.16
FFM (kg)	47.1 ± 9.66	45.8 ± 8.66	1.26 ± 3.76	<i>P</i> = 0.316	±2.66
RMR (kJ/d)	4690 ± 1112	4683 ± 108	7 ± 268	<i>P</i> = 0.935	±190
RQ	0.84 ± 0.03	0.83 ± 0.03	0.0138 ± 0.044	<i>P</i> = 0.346	±0.031
IET (°C)	35.5 ± 0.45	35.7 ± 0.60	−0.185 ± 0.503	<i>P</i> = 0.275	±0.355
FFG (°C)	+1.64 ± 2.57	+1.54 ± 2.31	+0.097 ± 3.23	<i>P</i> = 0.926	±2.28

Values are mean ± SD; *N* = 10

FM fat mass, FFM fat-free mass, RMR resting metabolic rate, RQ respiration quotient, IET in the ear temperature, FFG forearm to fingertip temperature gradient

^a Typical error = (SD of difference/√2) [26]

the two groups (Table 1). These values remained nonsignificant even after adjustment for age, gender, season and the body composition models employed (data not shown).

We present a series of analyses of RMR, RQ, IET and FFG that adjusted for different compartments of body composition (Table 2). In model 1, we adjusted for age, gender, season, PA, FM and FFM. Since ALTM and NALTM are approximations for skeletal muscle mass and organ tissue mass, respectively, we split FFM into its components, BMC, ALTM and NALTM in model 2 (Table 2). A final model 3 replaced ALTM and NALTM with predicted SMM and RM. We observed a significant relationship between unadjusted NALTM and RM in our sample ($r = 0.88$, $P = 0.001$). The overall analysis indicated a significantly lower adjusted RMR in SSA by 1000–1150 kJ/d across the models employed. Adjusted RQ was higher, and adjusted IET was significantly lower in SSA compared to EUR (Table 2). There were no differences in FFG between groups. Partial correlations indicated a direct relationship between IET and RMR that ranged from $r = 0.33$ – 0.35 (All $P < 0.02$), depending on the models 1–3 in Table 2.

Repeatability trial

There were no significant differences between visits in body composition or in metabolic measurements. The results of the two visits and an estimate of the typical within-subject error for each outcome is provided for the ten subjects in Table 2. Typical error in each ethnic group for main outcome variables is presented as SSA versus EUR and was IET (0.38 vs. 0.32 °C), FFG (2.7 vs. 1.5 °C), RQ (0.018 vs. 0.014) and RMR (172 vs. 191 kJ/d).

Discussion

There has been much research on the RMR of African-Americans compared to white Americans. While the

majority of studies indicate a significantly lower value in AA, there is a range of outcome from nonsignificantly lower by 133 kJ/d [16] to significantly lower by 889 kJ/d [9]. In part, this may arise from the different models of body composition used to partition FFM into its metabolically active components [8], but also from the degree of European ancestry in the AA group studied. The study of Manini et al. [19] sheds some light on the latter issue by predicting that every percentage point increase in EA would increase the conditional mean RMR by 1.6 kcal/d (95 % CI: 0.42, 2.7 kcal/d). The large CI of the slope indicates that those of pure African ancestry could have an RMR between 42 and 270 kcal/d lower, when compared to a pure European person. The present study is the first to the best of our knowledge that attempts to negate the influence of European admixture on the RMR of Africans.

RMR and body composition

There were significant differences in body composition with the younger SSA having a lower FM, %FM, FMI, but higher ASMI compared to EUR, the latter a reflection of greater muscle mass adjusted for height (Table 1). Predicted SMM was just significant, but NALTM and RM were similar between the two groups. Habitual PA did not differ between the two groups (Table 3), but is known to influence RMR even if the acute effect of PA is excluded prior to RMR measurement [40]. We hence included PA as a covariate in our analysis. As the participants were recruited throughout the year, we also included season in our model to control for any potential influence on RMR [41, 42]. Our analyses hence adjusted for all traditional factors affecting RMR and then modeled different compartments of body composition, in particular the effect of changes in the composition of FFM (Table 2, models 1–3).

We had no direct measures of skeletal muscle mass or organ tissue mass which was a limitation. However, DEXA

Table 3 Comparison of metabolic variables between Europeans and sub-Saharan Africans adjusted for models of body composition

Variable	Europeans (<i>n</i> = 32)	Sub-Saharan Africans (<i>n</i> = 29)
RMR (kJ/d)	5862 ± 214	4858 ± 179 [†]
adj_RMR (kJ/d)—model 1	5979 ± 109	4901 ± 156 [†]
adj_RMR (kJ/d)—model 2	5987 ± 111	4860 ± 161 [†]
adj_RMR (kJ/d)—model 3	5979 ± 111	4880 ± 161 [†]
RQ	0.83 ± 0.007	0.86 ± 0.009**
adj_RQ—model 1	0.83 ± 0.010	0.86 ± 0.014*
adj_RQ—model 2	0.83 ± 0.010	0.86 ± 0.014**
adj_RQ—model 3	0.83 ± 0.010	0.86 ± 0.014**
IET (°C)	35.74 ± 0.078	35.22 ± 0.092 [†]
adj_IET (°C)—model 1	35.60 ± 0.090	35.25 ± 0.128**
adj_IET (°C)—model 2	35.61 ± 0.092	35.26 ± 0.133**
adj_IET (°C)—model 3	35.60 ± 0.091	35.26 ± 0.133**
FFG (°C)	0.70 ± 0.460	0.78 ± 0.580
adj_FFG (°C)—model 1	−0.10 ± 0.564	−0.059 ± 0.803
adj_FFG (°C)—model 2	−0.19 ± 0.557	−0.31 ± 0.809
adj_FFG(°C)—model 3	−0.18 ± 0.540	−0.51 ± 0.780

Model 1: adjusted for age, sex, season, PA, FM and FFM

Model 2: adjusted for age, sex, season, PA, FM, BMC, ALTM and NALTM

Model 3: adjusted for age, sex, season, PA, FM, BMC, SMM and RM

There was no significant interaction between sex × ethnicity; season × ethnicity; and season × sex in any model

RMR resting metabolic rate, *RQ* respiration quotient, *IET* in the ear temperature, *FFG* forearm to fingertip gradient, *PA* physical activity, *FM* fat mass, *FFM* fat-free mass, *BMC* bone mineral content, *ALTM* appendicular lean tissue mass, *NALTM* non-appendicular lean tissue mass, *RM* residual mass

Data are mean ± SE. * *P* = 0.051; ** *P* < 0.05; [†] *P* < 0.005

measurements allow the division of FFM into ALTM and NALTM. Except for a small amount of skin and connective tissue, ALTM represents skeletal muscle mass, while NALTM (or trunk fat-free soft tissue) can be assumed to reflect organ mass [7]. Total skeletal muscle mass derived from multi-slice magnetic resonance imaging (MRI) is closely related to ALTM, and prediction equations developed on a mixed race population group exploit this close relationship [36]. NALTM and RM (Table 1) are conceptually similar in that they reflect MRI-measured organ tissue mass [13, 43], though the former includes some skeletal muscle of the trunk. We found a good correlation between

NALTM and RM in this dataset (*r* = 0.88. *P* = 0.001), and other authors have used trunk fat-free soft tissue (NALTM in this paper) to adjust for organ tissue mass as well [19]. Thus, both models 2 and 3 of our analysis allowed for functional comparisons in RMR between the groups studied [5].

Overall, we found that the RMR of SSA was significantly lower by ~1000–1150 kJ/d (~240–275 kcal/d) across all models tested. Such differences at a group mean level are far greater than generally reported for AA and are at the upper 95 % CI of the slope relating ancestry and RMR [19]. We are confident of our measurements since the test–retest data indicated good repeatability in RMR, and other variables studied. If we had the technology to measure brain weights, we may have explained a further 2 % of the variance in RMR [16]. However, it is unlikely that the inclusion of brain weights would account for the differences in RMR observed here.

Respiratory quotient

A high fasting RQ indicates carbohydrate as a major source of fuel and relatively greater carbohydrate oxidation predisposes to weight gain [2]. In a study of AA and whites, Ellis et al. [44] established that a high non-sleeping RQ predicted an increase in FM, independent of energy balance and insulin status. Conceptually, similar observations had been made in Pima Indians, where a greater carbohydrate oxidation rate was an independent predictor of weight gain [3]. In this study, adjusted fasting RQ was higher in SSA compared to EUR across all models of body composition tested (Table 3). This suggests that SSA have a relatively lower rate of fasting fat oxidation compared to EUR Australians. Nicklas et al. [10] also demonstrated a lower fasting fat oxidation in AA compared to white Americans, both as absolute values and as a percentage of RMR. Weyer et al. [23] measured their participants in a whole-body calorimeter and observed a higher 24-h RQ in AA compared to white Americans. They report a gender-specific effect wherein AA women were similar to white women in 24-h RQ, but AA men had a significantly higher 24-h RQ than white men which accounted for ~90 kJ less fat oxidation over the entire day. These outcomes are in contrast to results of some early studies, where no difference was observed in fasting RQ between AA and white Americans [20–22]. So, the balance of available evidence is equivocal at present. Future prospective studies on RMR could relate measurements of substrate oxidation to prediction of FM in SSA and hence confirm or refute these observations.

In the ear temperature and peripheral vasoconstriction

In this study, adjusted IET was significantly lower in SSA when compared to EUR (Table 2). IET was used as a surrogate for core body temperature, and we documented good precision in our measurements (0.355 °C or CV = 1 %,

Table 2). A prerequisite of RMR measurements is that it is conducted in the TNZ. We set room temperature to 25 °C which is in the middle of the TNZ for humans and standardized all clothing, so microenvironments were as similar as possible between ethnic groups. To ascertain how each person responded to the set temperature, we also monitored FFG, an index of vasoconstriction/vasodilation. We did not detect any between-group differences in FFG with values $<+2$ (Table 3). This would confirm that our participants were at thermoneutrality with no clinical vasoconstriction [26]. We labor these points since both IET and RMR will respond in each person, to deviations from TNZ. In fact, a major portion of RMR is directed toward the regulation of core body temperature [4]. To some extent, this was seen as a significant partial correlation ($r = 0.33\text{--}0.35$) between IET and RMR depending on the model of adjustment employed (Table 3). Over the years, there has always been a case for a low body temperature in the initiation of weight gain in the pre-obese state, in both animals and humans [4]. So the observations of a low adjusted IET in SSA compared to EUR could represent an additional risk factor for weight gain. More acceptable measures of core body temperature are needed in future studies in these ethnic groups.

It is tempting to speculate on the possible reasons for these differences in RMR. There are many processes that contribute to RMR, and these include Na/K ATPase activity, protein turnover, sympathetic nervous system (SNS) activity and substrate cycling. We are unaware of studies that have measured these in Africans, though theoretically a reduction in either of these would contribute to a lower RMR. Moreover, there is close relationship between mitochondrial activity and whole-body oxygen consumption at rest [45, 46]. Mitochondrial uncoupling would increase heat production at the expense of ATP production. Indeed variations in uncoupling protein 3 (UCP 3) that is strongly expressed in skeletal muscle were significantly related to RMR in AA but not in whites [11]. Those authors observed a ~582 kJ/d lower RMR in the CC genotype compared to the TT genotype in AA participants [11]. Such data propose that mitochondrial efficiency may also explain a sizeable portion of the ethnic disparity in RMR observed in this and other studies in the area.

There were some limitations to our cross-sectional study. We did not select a representative sample of the groups under study, so the outcomes are not readily generalizable to all SSA. A gold-standard method for core body temperature was unavailable to us. Instead, we used IET which was practical, repeatable and had been validated by others. A study published at the time of our investigations showed that physical fitness per se rather than moderate to vigorous PA reduced the differences in RMR between AA and whites by 25 % [15]. We could not control for this variable and are unable to judge its potential impact in this

study. Lastly, while the determination of ethnicity through self-report is a common approach in the literature in this area, it may not be considered optimal. However, there are strengths to our research as well. We recruited SSA to minimize the effect of EUR admixture on RMR and other metabolic variables in this group. We used validated tools for the measurement of RMR/body composition and have documented their precision. Our protocol of measurement and its execution was good. We controlled clothing worn, tightly regulated the ambient temperature of measurement and provide an objective confirmation through FFG that each group was in the thermoneutral zone.

In conclusion, we demonstrate that sub-Saharan African individuals are characterized by the combination of a lower adjusted RMR, a higher RQ and a lower IET compared to Australians of European descent. Each of these metabolic factors is an independent predictor of weight gain. If these outcomes are confirmed in population representative studies, their collective impact would help explain some of the greater predisposition to weight gain in this ethnic group.

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Author contributions PAAN, KP and MJS planned the study. PAAN and KP carried out the study and collated the data. MJS checked data entry, analyzed the data, co-wrote the first draft with PAAN and directed all revisions. All authors contributed to the writing of the final manuscript.

Compliance with ethical standards

Conflict of interest On behalf of the investigators, MJS declares that there is no conflict of interest.

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