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Physical activity energy expenditure and cardiometabolic health in three rural Kenyan populations

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Abstract

Objectives: Physical activity is beneficial for metabolic health but the extent to which this may differ by ethnicity is still unclear. Here, the objective was to characterize the association between physical activity energy expenditure (PAEE) and cardiometabolic risk among the Luo, Kamba, and Maasai ethnic groups of rural Kenya.

Methods: In a cross-sectional study of 1084 rural Kenyans, free-living PAEE was objectively measured using individually-calibrated heart rate and movement sensing. A clustered metabolic syndrome risk score (zMS) was developed by averaging the sex-specific z-scores of five risk components measuring central adiposity, blood pressure, lipid levels, glucose tolerance, and insulin resistance.

Results: zMS was 0.08 (−0.09; −0.06) SD lower for every 10 kJ/kg/day difference in PAEE after adjustment for age and sex; this association was modified by ethnicity (interaction with PAEE $P < 0.05$). When adjusted for adiposity, each 10 kJ/kg/day difference in PAEE was predicted to lower zMS by 0.04 (−0.05, −0.03) SD, without evidence of interaction by ethnicity. The Maasai were predicted to have higher cardiometabolic risk than the Kamba and Luo at every quintile of PAEE, with a strong dose-dependent decreasing trend among all ethnicities.

Conclusion: Free-living PAEE is strongly inversely associated with cardiometabolic risk in rural Kenyans. Differences between ethnic groups in this association were observed but were explained by differences in central adiposity. Therefore, targeted interventions to increase PAEE are more likely to be effective in subgroups with high central adiposity, such as Maasai with low levels of PAEE.

1 | INTRODUCTION

The highest age-standardized mortality rates for cardiovascular diseases (CVD) occur in developing countries (World Health Organization, 2014), in large part because of the epidemiological transition, the subsequent rise of CVD risk factors, and the relative inability of these countries to prevent and treat chronic disease. Though research on cardiometabolic risk factors in these communities is generally sparse, low risk factor levels have been reported for ethnic groups

practicing subsistence living (Kaplan et al., 2017; Raichlen et al., 2017). In addition, studies examining risk factor clustering (metabolic syndrome) is especially limited (Okafor, 2012), even though epidemiological studies continue to indicate its strong association with incident CVD and CVD mortality (Galassi, Reynolds, & He, 2006; Gami et al., 2007). In Africa, where levels of cardiometabolic risk have begun to rise, estimates have differed widely between populations (Fezeu, Balkau, Kengne, Sobngwi, & Mbanya, 2007; Longo-Mbenza, Kasiam Lasi On'kin, Nge Okwe, & Kangola

Kabangu, 2011; Tran et al., 2011; Vorster, 2002; Wahab, Sani, Gbadamosi, & Yandutse, 2008). Taken together, this suggests a population-dependent etiology for CVDs, with possible complex interactions between components of biological origin and cultural practices, that is, an ethnicity-specific risk profile.

Earlier cross-sectional studies in Europe found inverse associations between free-living physical activity and cardiometabolic risk (Franks, Ekelund, Brage, Wong, & Wareham, 2004; Lakka et al., 2003; Rennie, McCarthy, Yazdgerdi, Marmot, & Brunner, 2003), and prospective studies more robustly concluded that men engaging in recommended levels of activity were less likely to develop clustering of cardiometabolic risk even if they were considered high-risk (Laaksonen et al., 2002; Laaksonen et al., 2003). Most of these studies assessed physical activity via subjective means, but, recently, more studies have used objective measures of activity (Ekelund et al., 2005; Karelis et al., 2008; Tucker, Welk, Beyler, & Kim, 2016). Among research examining the association between physical activity and cardiometabolic risk in Africa, however, there are only a few studies that have used an objective method of assessing physical activity (Assah et al., 2011a; Atiase et al., 2015; Dickie, Micklesfield, Chantler, Lambert, & Goedecke, 2016), none from the East African region other than our own (Christensen et al., 2009) and only one quantifying the association using physical activity energy expenditure (PAEE) as the exposure. Assah et al. (2011a) reported inverse associations between PAEE and clustered metabolic risk, amounting to a 6.5 kJ/kg/day higher PAEE and a 13.7% lower prevalence in metabolic syndrome between rural and urban Cameroonians, respectively.

The influence of ethnicity as a potential modifier of the association between PAEE and cardiometabolic risk has not been well documented. Among African ethnicities, we have previously reported strong inverse relationships between physical activity and glucose intolerance (from a standard 75 g glucose tolerance test) in both men and women from Kenya using individually calibrated combined heart rate (HR) and movement sensing (Christensen et al., 2009). Using waist-mounted accelerometry, Atiase et al. (2015) reported diverse and often unexpected relationships with glucose indices in five African populations, although the sample size in each of the examined strata was small, and the method of activity assessment may still have been subject to site-specific biases such as inability to capture upper-body activity. Recently we reported that ethnicity and sex did not modify the traditional cardiovascular risk factors of hypertension and dyslipidemia among Luo, Kamba, and Maasai in rural Kenya, when adjusted for overall obesity and PAEE (Christensen et al., 2016). However, the ethnic specificity of the PAEE-cardiometabolic risk factor relationship was not examined. In this report, we examine the potential mediating and/or effect-modifying role of ethnicity in the relationship between PAEE and clustered cardiometabolic risk among three rural Kenyan populations.

2 | METHODS

The study population and selection procedure for the study have been extensively documented (Christensen et al., 2008; Christensen et al., 2009; Christensen et al., 2012; Christensen et al., 2014). In brief, a cross-sectional study was conducted among rural Kenyans, comprising Luo, Kamba, and Maasai ethnicities, which represent the traditional societies of agro-fishing, agriculture, and agro-pastoralism. The Luo primarily subsist on cereal foods and fish, and reside in western Kenya near Lake Victoria; the Kamba rely on diets dominated by maize and farmed crops from semi-arid eastern Kenyan highlands; and the Maasai are agro-pastoralists from southern Kenya whose diet primarily consists of animal products as a major source of dietary energy, protein, and fat intake (Hansen et al., 2011).

Volunteers were initially selected at village meetings, and recruited at several community centers located in former Bondo, Kitui, and Transmara districts. Initial data were collected in August to September of 2005 for Luo, September to October for Kamba, and October to November for Maasai to coincide with the end of the harvest season in each geographical area. Inclusion criteria for the study were self-identification with Luo, Kamba, or Maasai ethnicity by paternal lineage, rural residency, and ≥17 years of age. Exclusion criteria included pregnancy and illness such as malaria, psychosis, and locomotor disability. In total, 1170 participants were eligible for the study.

All participants were presented with a standard statement by a local social mobilizer, describing the current study as a diabetes survey including a general health check. Participants gave written (including thumb print for illiterate individuals) and oral informed consent, as approved by the National Ethical Review Committee in Kenya and the National Committee on Biomedical Research Ethics in Denmark. Participants were excluded from analysis if data were missing on age, sex, ethnicity, cardio-metabolic risk, and objective physical activity. In total, 1084 participants were included in the final analytical sample (Luo: $n = 375$, Kamba: $n = 378$, Maasai: $n = 331$).

2.1 | Clinical and biochemical measurements

Measurements have been described in detail in previous publications (Christensen et al., 2008; Christensen et al., 2009; Christensen et al., 2012; Christensen et al., 2016; Hansen et al., 2011). Various anthropometric measurements were collected, including height, weight, and waist circumference, and derived variables such as BMI (kg/m^2). Ultrasonography was used to measure abdominal visceral and

subcutaneous fat thickness following a standard protocol (Stolk et al., 2001).

Clinical and metabolic measures were carried out. Systolic and diastolic blood pressures in the seated position were determined based on the mean of two measurements after 15 minutes of rest. A standard 75 g oral glucose challenge test was performed after an overnight fast, and testing was completed using venous blood. Fasting plasma and serum samples were centrifuged and aliquoted for transport to Gentofte, Denmark at −80°C. Assays were performed by the Steno Diabetes Center Copenhagen. Total cholesterol, high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C), triglycerides, insulin, and other biomarkers were analyzed or derived according to standard protocols.

Questionnaires for health assessment and 24-hour recall dietary intake were translated from English into Kiswahili and three local languages: Dholuo, Kikamba, and Ol-maa, which were verified for accuracy by back-translation into English. Structured interviews were conducted in Kiswahili, English, or the local language by a team investigator or trained assistant. Information obtained included age, smoking status, alcohol consumption, diet, and macronutrient intake.

2.2 | Physical activity measurement

Free-living physical activity was assessed using a combined HR and uniaxial acceleration sensor (Actiheart, CamNtech, Cambridge, UK) as described in detail elsewhere (Brage, Brage, Franks, Ekelund, & Wareham, 2005). For individual calibration, the monitor was placed on two ECG electrodes at the sternal level to record HR response to an 8-minute incremental step test as described by prior protocols (Brage et al., 2006; Brage et al., 2007). Participants were then instructed to wear the monitor continuously over 5 days with data collected in 30-second epochs. HR data were preprocessed using a Bayesian robust regression method (Stegle, Fallert, MacKay, & Brage, 2008) and individually calibrated using parameters derived from the step test. Nonwear was identified as the combination of long (>90 minute) periods of no movement combined with nonphysiological HR. Free-living PAEE was estimated using branched equation modeling (Brage et al., 2004), an approach that combines HR with movement registration to calculate physical activity intensity, which was integrated over time to produce an estimate of average daily PAEE while minimizing diurnal bias caused by nonrandom patterning of nonwear (Brage et al., 2013); this approach compares favorably with gold-standard isotopic assessment of PAEE (Assah et al., 2011b; Brage et al., 2015). Participants were excluded if objective activity data were missing ($n = 48$), if total monitor wear time was shorter than 48 hours in total ($n = 37$), or if there were less than 8 hours of data in each diurnal time quadrant (3 AM-9 AM; 9 AM-3 PM; 3 PM-9 PM; 9 PM-3 AM) during the wear period ($n = 1$). Physical activity data were additionally assessed for monitor corruption and misclassification via manual verification of free-living plots.

2.3 | Clustered metabolic risk score

A continuous clustered metabolic risk score (zMS) was created by averaging sex-specific z-scores of five equally weighted indicators for metabolic risk, including abdominal obesity, measured by averaged z-scores of waist circumference (cm) and visceral fat (cm); blood pressure, derived from average z-scores of systolic and diastolic blood pressures (mmHg); blood glucose, captured by average z-scores of measurements taken in the fasted state as well as at 30 and 120 minutes after glucose challenge (mmol/L); fasting insulin (ρmol/L); and lipid levels, assessed by average z-scores of triglycerides (mmol/L) and negative HDL-C (mmol/L). These measurements were standardized (z-scored) by subtracting the sample mean from the measured value and then dividing by the SD of the sample mean. To meet z-standardization normality assumptions, insulin and triglyceride levels were log-transformed. The average of these five components resulted in a sex-stratified score for clustered metabolic risk, or zMS. A second 4-component score was also created which did not contain the abdominal obesity component, hence denoted non-Ob zMS.

2.4 | Statistical analysis

Stata/IC 13.1 was used for all statistical analyses (StataCorp, College Station, TX). Descriptive statistics for the sample were presented as means (SD), medians (IQR) for nonnormally distributed data, or numbers with proportions, stratified by ethnicity and sex.

Least squares multiple linear regressions were used to describe and predict the association between zMS and PAEE. To systematically explore each potential confounder's effect on this association, adjustments were progressively added to successive regression models. Models were stratified by ethnicity and adjusted for age, sex and monitor wear-time only (Model A), then additionally adjusted for smoking status, alcohol consumption status, and total fat intake (Model B), and finally further adjusted for waist circumference and visceral fat (Model C). Pooled analyses of all ethnic groups together were further adjusted by ethnic group. All linear regressions were checked for influential points using Cook's D, DFBETA, and leverage-vs-residual-squared plots.

Evidence for interaction by ethnicity was assessed using likelihood ratio test comparing nested models with and without an interaction term between ethnicity and PAEE. For these models, we also fitted an interaction between age and PAEE. In the postestimation analysis, predictive margins were obtained by accounting for the direct effects of the covariates at each adjustment level.

TABLE 1 Descriptive characteristics of the study sample, stratified by sex and ethnicity

	Women			Men		
	Luo (n = 207)	Kamba (n = 279)	Maasai (n = 170)	Luo (n = 168)	Kamba (n = 99)	Maasai (n = 161)
Age (yrs)	39.2 (9.5)	40.2 (9.5)	35.0 (10.5)	39.7 (10.3)	42.5 (10.2)	38.6 (11.5)
BMI (kg/m^2)	21.7 (3.7)	22.5 (4.5)	22.5 (4.8)	21.1 (2.8)	20.0 (2.6)	21.0 (4.8)
Waist circumference (cm)	77.3 (8.6)	77.3 (10.2)	78.8 (11.8)	78.7 (8.3)	76.7 (7.3)	80.7 (13.5)
Visceral fat (cm)	6.2 (1.1)	6.1 (1.4)	6.4 (1.5)	6.7 (1.6)	6.2 (1.4)	7.2 (1.8)
Systolic blood pressure (mmHg)	113 (14.2)	120 (16.3)	116 (13.2)	118 (17.3)	125 (16.0)	124 (14.6)
Diastolic blood pressure (mmHg)	72 (9.4)	75 (10.4)	73 (9.3)	73 (10.3)	75 (9.3)	77 (9.8)
Blood glucose, fasting (mmol/L)	4.8 (0.6)	4.4 (0.6)	4.6 (1.3)	4.3 (0.7)	4.4 (1.1)	4.4 (1.0)
Blood glucose, 30 minutes (mmol/L)	6.1 (1.3)	6.3 (1.4)	6.1 (1.6)	6.1 (1.5)	6.5 (1.8)	6.3 (1.5)
Blood glucose, 120 minutes (mmol/L)	5.6 (1.4)	5.3 (1.5)	5.3 (1.5)	5.2 (2.0)	4.8 (2.1)	4.5 (1.7)
Insulin, fasting (ρmol/L)	19 [17]	24 [19]	26 [21]	16 [13]	14 [14]	24 [19]
Triglyceride (mmol/L)	0.9 [0.5]	0.8 [0.4]	0.8 [0.5]	0.92 [0.5]	0.8 [0.4]	0.9 [0.5]
Total cholesterol (mmol/L)	3.6 (0.9)	3.8 (0.9)	3.8 (0.9)	3.7 (1.0)	3.8 (0.9)	4.0 (1.1)
HDL cholesterol (mmol/L)	0.9 (0.4)	1.2 (0.3)	1.2 (0.3)	1.0 (0.4)	1.1 (0.3)	1.1 (0.32)
LDL cholesterol (mmol/L)	2.2 (0.7)	2.2 (0.8)	2.2 (0.7)	2.3 (0.8)	2.2 (0.7)	2.4 (0.9)
Total fat intake (kJ/day)	1320 (637)	882 (614)	1820 (763)	1890 (1020)	1290 (806)	2670 (1080)
Smoking status (% yes)	1.5 (0.5; 4.7)	5.3 (3.2; 8.8)	10.8 (6.9; 16.6)	15.7 (10.8; 22.3)	35.4 (26.5; 45.5)	15.8 (10.9; 22.4)
Alcohol consumption (% yes)	4.4 (2.3; 8.2)	3.9 (2.2; 7.0)	3.5 (1.6; 7.7)	30.4 (23.9; 37.8)	33.3 (24.7; 43.2)	14.9 (10.2; 21.3)
PAEE (kJ/kg/day)	57.3 (19.8)	63.1 (23.5)	73.5 (23.1)	73.4 (26.4)	77.0 (29.3)	76.6 (25.8)

Data are means (SD), medians [IQR], or proportions (95% CI).

3 | RESULTS

The characteristics of the 1084 participants analyzed in this study are provided in Table 1. A total of 656 (60.5%) women and 428 men with valid physical activity data were assessed. Mean ± SD age of the sample was 39.1 ± 10.3 years; women averaged 38.6 ± 10.0 years and men 39.9 ± 10.8 years. The median duration of monitor wear was 95.0 ± 9.1 hours (4 days) without significant differences between ethnic subgroups. On average, participants engaged in a mean PAEE of 68.2 ± 25.6 kJ/kg/day. Women registered a lower PAEE than men (63.6 ± 23.5 kJ/kg/day vs 75.4 ± 27.0 kJ/kg/day, $P < 0.001$). Across the three ethnic populations, the Maasai registered the highest mean PAEE overall (74.9 ± 24.6 kJ/kg/day; Luo: 64.2 ± 24.6 kJ/kg/day; Kamba: 66.5 ± 26.2 kJ/kg/day). Maasai women had significantly higher daily PAEE levels (73.5 ± 23.1 kJ/kg/day, $P < 0.01$) compared with Luo (57.3 ± 19.8) and Kamba (63.1 ± 23.5) women, but men were more similar across ethnic groups.

Among the components that constitute the metabolic syndrome, mean values between the sexes and ethnic groups were similar. Mean waist circumference overall was 78.2 ± 10.3 cm and visceral fat 6.4 ± 1.5 cm, though Maasai men had higher central obesity compared with Kamba (80.7 ± 13.5 cm v. 76.7 ± 7.3 cm waist circumference, 7.2 ± 1.8 cm v. 6.2 ± 1.4 cm visceral fat; $P < 0.001$), and men in general possessed greater visceral fat than women (6.8 ± 1.7 v. 6.2 ± 1.3 cm respectively, $P < 0.001$). Overall, systolic and diastolic blood pressure means were 119 ± 16

and 74 ± 10 mmHg respectively. Mean venous blood glucose at fasting, 30-minutes, and 120-minutes were recorded at 4.4 ± 0.9, 6.2 ± 1.5, and 5.2 ± 1.7 mmol/L; blood glucose levels were lower in men compared to women at fasting ($P < 0.05$) and 2 hours ($P < 0.001$). Fasting serum insulin (overall log mean 3.1 ± 0.68 ρmol/L, $P < 0.01$) was lower in men, and plasma triglyceride levels (overall mean 0.98 ± 0.5 mmol/L, $P < 0.01$) were lower in women. Plasma HDL-C was 1.1 ± 0.34 mmol/L on average.

In the multiple linear regression analyses of the association between PAEE and metabolic risk factors (Table 2), clustered cardiometabolic risk (zMS) was 0.08 ± 0.01 SD lower for every 10 kJ/kg/day difference in PAEE, after adjustment for age, sex, ethnicity, and monitor wear time (Model A); this difference was 0.07 ± 0.01 SD after additionally adjusting for smoking status, alcohol consumption status, and total fat intake (Model B). Adiposity, blood pressure, blood glucose, fasting insulin, and triglycerides were all negatively associated with PAEE. There was significant interaction between ethnicity and PAEE on waist circumference, visceral fat, fasting glucose, HDL-C, and overall clustered cardiometabolic risk (Model B). In the subgroup analysis, PAEE was significantly associated with central adiposity, 2-hour glucose, insulin, lipid measures, and clustered cardiometabolic risk among each of the three groups; however, blood pressure was only significantly associated with PAEE in Kamba and Maasai. In these analyses which were additionally adjusted for interaction between age and PAEE, associations were stronger (interaction significant) in older vs younger adults with respect to adiposity measures,

TABLE 2 Linear associations between physical activity energy expenditure and metabolic risk factors in three rural Kenyan populations ($n = 1084$)

Group	Outcome	Model A β [95% CI]	Model B β [95% CI]	Model C β [95% CI]
Luo	Waist circumference (cm)	−0.43 [−0.79, −0.08]	−0.37 [−0.74, −0.01]	–
	Visceral fat (cm)	−0.09 [−0.15, −0.03]	−0.09 [−0.15, −0.02]	–
	SBP (mmHg)	−0.39 [−1.09, 0.32]	−0.41 [−1.14, 0.32]	−0.21 [−0.94, 0.52]
	DBP (mmHg)	−0.44 [−0.88, 0.00]	−0.45 [−0.91, 0.00]	−0.29 [−0.74, 0.16]
	Fasting glucose (mmol/L)	−0.02 [−0.05, 0.01]	−0.02 [−0.05, 0.01]	−0.01 [−0.04, 0.02]
	30-min glucose (mmol/L)	−0.06 [−0.13, 0.00]	−0.06 [−0.13, 0.00]	−0.05 [−0.12, 0.01]
	2-hr glucose (mmol/L)	−0.14 [−0.22, −0.07]	−0.13 [−0.20, −0.05]	−0.10 [−0.17, −0.03]
	Fasting insulin (ρmol/L)	−0.29 [−0.44, −0.14]	−0.25 [−0.39, −0.11]	−0.21 [−0.34, −0.07]
	Triglycerides (mmol/L)	−0.03 [−0.05, −0.01]	−0.03 [−0.05, −0.01]	−0.02 [−0.04, 0.00]
	HDL cholesterol (mmol/L)	0.04 [0.02, 0.05]	0.04 [0.02, 0.06]	0.03 [0.02, 0.05]
	zMS	−0.06 [−0.08, −0.04]	−0.06 [−0.08, −0.03]	–
	Non-Ob zMS	−0.06 [−0.09, −0.04]	−0.06 [−0.08, −0.04]	−0.04 [−0.07, −0.02]
Kamba	Waist circumference (cm)	−1.05 [−1.40, −0.70]	−0.91 [−1.27, −0.54]	–
	Visceral fat (cm)	−0.13 [−0.18, −0.07]	−0.12 [−0.18, −0.06]	–
	SBP (mmHg)	−1.30 [−1.93, −0.66]	−1.22 [−1.91, −0.53]	−0.90 [−1.60, −0.21]
	DBP (mmHg)	−1.12 [−1.51, −0.74]	−1.05 [−1.47, −0.63]	−0.85 [−1.27, −0.43]
	Fasting glucose (mmol/L)	−0.04 [−0.07, −0.01]	−0.03 [−0.06, 0.00]	−0.02 [−0.05, 0.01]
	30-min glucose (mmol/L)	−0.05 [−0.11, 0.00]	−0.05 [−0.11, 0.02]	−0.04 [−0.10, 0.03]
	2-hr glucose (mmol/L)	−0.12 [−0.19, −0.06]	−0.10 [−0.17, −0.03]	−0.08 [−0.15, −0.01]
	Fasting insulin (ρmol/L)	−0.32 [−0.45, −0.18]	−0.26 [−0.40, −0.11]	−0.12 [−0.26, 0.02]
	Triglycerides (mmol/L)	−0.04 [−0.06, −0.02]	−0.04 [−0.06, −0.02]	−0.02 [−0.04, 0.00]
	HDL cholesterol (mmol/L)	0.01 [0.00, 0.03]	0.01 [0.00, 0.02]	0.01 [−0.01, 0.02]
	zMS	−0.08 [−0.10, −0.06]	−0.07 [−0.09, −0.05]	–
	Non-Ob zMS	−0.07 [−0.10, −0.05]	−0.06 [−0.08, −0.04]	−0.04 [−0.06, −0.01]
Maasai	Waist circumference (cm)	−1.84 [−2.37, −1.30]	−1.81 [−2.36, −1.26]	–
	Visceral fat (cm)	−0.17 [−0.24, −0.09]	−0.15 [−0.23, −0.07]	–
	SBP (mmHg)	−1.70 [−2.35, −1.04]	−1.54 [−2.21, −0.87]	−0.65 [−1.31, 0.00]
	DBP (mmHg)	−1.15 [−1.60, −0.70]	−1.00 [−1.47, −0.54]	−0.46 [−0.93, −0.00]
	Fasting glucose (mmol/L)	−0.08 [−0.14, −0.03]	−0.07 [−0.13, −0.01]	−0.07 [−0.13, 0.00]
	30-min glucose (mmol/L)	−0.12 [−0.19, −0.04]	−0.13 [−0.20, −0.05]	−0.09 [−0.18, −0.01]
	2-hr glucose (mmol/L)	−0.16 [−0.24, −0.08]	−0.17 [−0.25, −0.09]	−0.11 [−0.20, −0.03]
	Fasting insulin (ρmol/L)	−0.29 [−0.44, −0.15]	−0.27 [−0.42, −0.13]	−0.06 [−0.19, 0.08]
	Triglycerides (mmol/L)	−0.03 [−0.06, −0.01]	−0.03 [−0.06, −0.01]	−0.01 [−0.03, 0.02]
	HDL cholesterol (mmol/L)	0.01 [0.00, 0.03]	0.01 [0.00, 0.03]	0.01 [−0.01, 0.02]
	zMS	−0.10 [−0.13, −0.07]	−0.09 [−0.12, −0.06]	–
	Non-Ob zMS	−0.09 [−0.12, −0.06]	−0.08 [−0.11, −0.05]	−0.04 [−0.06, −0.01]
All	Waist circumference (cm)	−1.10 [−1.33, −0.86]*	−1.02 [−1.26, −0.77]*	–
	Visceral fat (cm)	−0.13 [−0.17, −0.10]*	−0.12 [−0.16, −0.09]*	–
	SBP (mmHg)	−1.10 [−1.48, −0.73]	−1.01 [−1.40, −0.61]	−0.55 [−0.94, −0.16]
	DBP (mmHg)	−0.92 [−1.16, −0.68]	−0.84 [−1.09, −0.59]	−0.55 [−0.80, −0.29]
	Fasting glucose (mmol/L)	−0.04 [−0.07, −0.02]*	−0.04 [−0.06, −0.02]*	−0.03 [−0.05, −0.01]*
	30-min glucose (mmol/L)	−0.07 [−0.11, −0.04]	−0.07 [−0.11, −0.03]	−0.06 [−0.10, −0.02]
	2-hr glucose (mmol/L)	−0.14 [−0.18, −0.10]	−0.13 [−0.17, −0.09]	−0.09 [−0.14, −0.05]
	Fasting insulin (ρmol/L)	−0.31 [−0.39, −0.23]	−0.27 [−0.35, −0.19]	−0.14 [−0.22, −0.07]
	Triglycerides (mmol/L)	−0.04 [−0.05, −0.02]	−0.04 [−0.05, −0.02]	−0.02 [−0.03, −0.00]
	HDL cholesterol (mmol/L)	0.02 [0.01, 0.03]*	0.02 [0.01, 0.03]*	0.02 [0.01, 0.02]*
	zMS	−0.08 [−0.09, −0.07]*	−0.07 [−0.09, −0.06]*	–
	Non-Ob zMS	−0.08 [−0.09, −0.06]	−0.07 [−0.08, −0.05]	−0.04 [−0.05, −0.03]

β denotes the change in the outcome variable for every 10 kJ/kg/day change in PAEE.

Insulin and triglycerides are log-transformed.

zMS: clustered metabolic risk score with all risk factors.

Non-Ob zMS: clustered metabolic risk score with all risk factors except the adiposity component included.

Model A: adjusted for age, sex, and monitor wear time (plus ethnicity in pooled analyses).

Model B: adjusted for age, sex, monitor wear time, smoking status, alcohol consumption status, and total fat intake (plus ethnicity in pooled analyses).

Model C: adjusted for age, sex, monitor wear time, smoking status, alcohol consumption status, total fat intake, waist circumference, and visceral fat (plus ethnicity in pooled analyses).

*$P < .05$ for PAEE × ethnicity interaction.

systolic blood pressure, fasting insulin, triglycerides, and clustered cardiometabolic risk.

In exploring the effect of central obesity as a potential confounder or mediator of the association between PAEE and cardiometabolic risk factors, waist circumference and visceral fat were removed from the clustered metabolic risk score calculation and adjusted for in Model C. In this model, each 10 kJ/kg higher daily PAEE was predicted to lower nonobesity zMS (non-Ob zMS) by 0.04 ± 0.01 SD. Thus, controlling for abdominal adiposity appreciably reduced the magnitude of the PAEE effect (41.5% mediation). This was also observed for the nonclustered metabolic risk components; higher PAEE was associated with statistically significant favorable differences in all metabolic risk factors across all three ethnic groups, and interaction between ethnicity and PAEE remained significant only for fasting glucose and HDL-C in this model. The above-mentioned interaction between age and PAEE was attenuated and no longer significant in adiposity-adjusted models.

To further compare the effect of adiposity on the PAEE–zMS association, a prediction model was created based on the adjustment levels for Model B plus the inclusion of a PAEE-ethnicity interaction term. Depicting PAEE in quintiles, Figure 1 (*left panel*) predicts the Maasai at PAEE quintile 1 (<46 kJ/kg daily PAEE) to have a statistically significant zMS at 0.72 ± 0.16 SD, with the Kamba and the Luo estimates being lower at 0.30 ± 0.11 SD and 0.16 ± 0.11 SD (all P < 0.001), respectively. Whereas the interaction was significant between PAEE and ethnicity for zMS, this interaction was attenuated after accounting for

central obesity (*right panel*). In the *non-ob zMS* model, the Maasai still had the highest cardiometabolic risk in PAEE quintile 1 (0.59 ± 0.16 SD vs the Kamba: 0.28 ± 0.12 SD, P < 0.005 and the Luo: 0.17 ± 0.11 SD, P < 0.001). Linear trends in this model were similar to zMS; in general, the Maasai demonstrated a higher predicted metabolic risk than the Kamba and Luo at every quintile, with a strong dose-dependent decreasing trend among all ethnicities.

4 | DISCUSSION

In a population sample of three ethnic groups from rural Kenya, representing the traditional practices of agro-fishing, agriculture, and agro-pastoralism, the association between objectively-measured PAEE and the clustering of metabolic risk factors was determined to be inversely related in a dose-dependent fashion. PAEE remained strongly associated with the continuous clustered cardiometabolic risk score at all three levels of adjustment, including models that controlled for abdominal adiposity.

This is the first study to date that also demonstrates an effect of ethnic origin on the association between objectively assessed PAEE and metabolic risk in the African populations. In the analyses stratified by ethnicity, the Maasai were identified to be at higher cardiometabolic risk compared to the Kamba and Luo for every quintile of daily PAEE, and this difference was statistically significant at the lowest level of PAEE (<46 kJ/kg/day) with convergence of risk at higher levels.
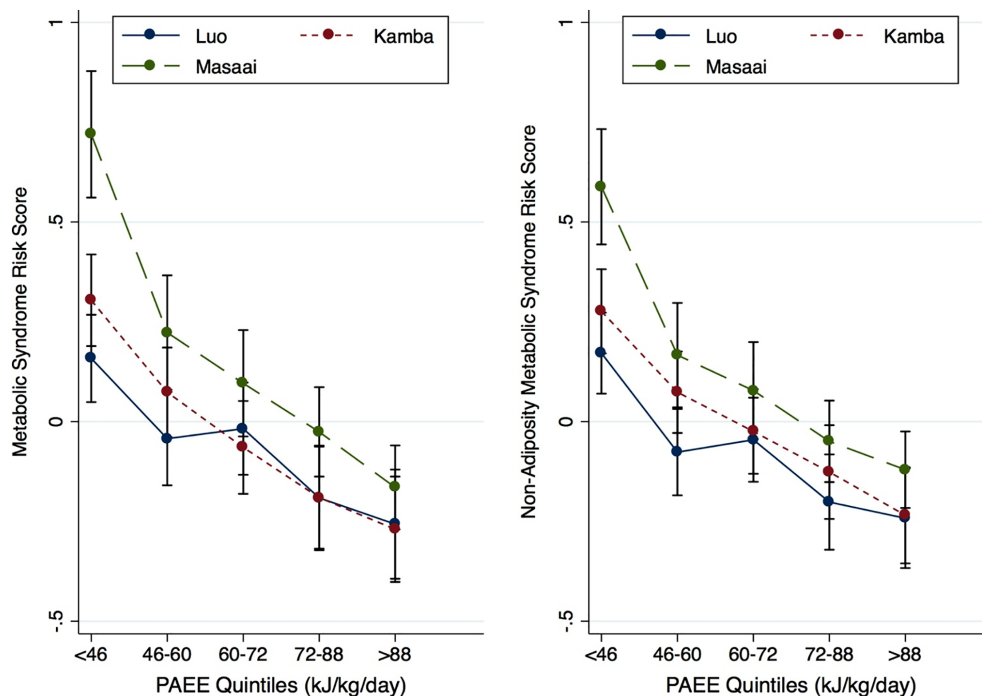

FIGURE 1 Clustered metabolic risk score (zMS) (*left panel*) and non-adiposity clustered metabolic risk score (*Non-Ob zMS*) by PAEE quintile (right panel). Estimates are marginal means from regression model including age, sex, smoking status, alcohol intake, fat intake, ethnicity, PAEE, and PAEE x ethnicity interaction

4.1 | Physical activity, central obesity and clustered metabolic risk

In the multiple linear regressions, the mediating effect of central adiposity on the associations between PAEE and clustered metabolic risk were explored with waist circumference and visceral fat as proxies. It was demonstrated to have large effects on the constituents of the metabolic syndrome as well as the clustering of these risk factors as a whole, as indicated by differences in the regression coefficients postadjustment. Central adiposity was not the sole contributor to this association, since PAEE continued to be significantly associated with all other outcome measures. However, the results indicated that adiposity reliably accounted for approximately half of this association. The difference in adjusted zMS for every 10 kJ/kg/day of additional PAEE decreased from −0.07 (−0.08; −0.05) in Model B to −0.04 (−0.05; −0.02) in Model C, or a 41.5% difference after adiposity adjustment - an observation noted in the subcomponent analyses as well, in metrics such as systolic and diastolic blood pressures, fasting insulin, and triglyceride levels. Lifestyle interventions that also target obesity reduction are therefore likely to exert major beneficial effects on multiple cardiovascular risk factors as well as clustered cardiometabolic risk overall, though notably associations with fasting glucose levels and HDL-C remain relatively unchanged.

4.2 | Physical activity, central obesity and ethnicity

Additionally, central adiposity seems to play a major role in explaining the contribution of ethnic origin on the association between PAEE and cardiometabolic risk. After adjustment for adiposity, the observed interaction between ethnicity and PAEE on clustered cardiometabolic risk was attenuated, such that the relationship between PAEE and cardiometabolic risk was more universal across ethnic groups (with the notable exceptions of fasting glucose which was more strongly associated in the Maasai, and HDL-C which was more strongly positively associated with PAEE in the Luo). As the Maasai have the highest mean waist circumference among the three ethnic groups, nonlinearity in the PAEE-metabolic risk relationship may be due to differential adiposity, which identifies a potential target for intervention—namely those with high measures of waist circumference regardless of ethnic origin.

The influence of cultural activities and diet may also play large roles in cardiometabolic risk susceptibility. As the Maasai generally have higher levels of physical activity, presumably influenced by herding culture and to some extent traditional jumping-dance activities (in men only), the Maasai at the lowest quintile of PAEE nevertheless have highest cardiometabolic risk, whereas at the highest PAEE quintile the Maasai have a metabolic risk equal to the Kamba and Luo. The presumption is that higher physical activity levels may normalize metabolic dysfunction in Maasai who would otherwise be at high risk due to high central obesity or dietary intake—especially fat intake—more so than in other ethnic groups. Furthermore, those who do not engage in physical activity may exacerbate existing genetic or socio-cultural predispositions and aggravate their CVD risk factors.

4.3 | Strengths of the study

There are several strengths of our analysis. While the clustered cardiometabolic z-score, zMS, may have limited clinical utility, since it is not a biologically defined variable but one that is statistically-derived, the advantage of using clustered risk scores is that they are statistically more sensitive and less error prone (Ragland, 1992). In comparison with categorical data, zMS captures more variance in cardiometabolic risk. Since its subcomponents represent multiple ways to measure the "latent variable" of metabolic risk clustering, this is essentially analogous to a repeated measures approach, and the implication is that the creation of a risk score is more robust than using a single measure. Additionally, objective measurement of physical activity is likely to benefit from greater precision than traditional subjective methods, as these instruments are not prone to recall bias, and algorithms for PAEE have been validated under both controlled conditions (Brage et al., 2004; Brage et al., 2007; Strath, Brage, & Ekelund, 2005) and in the field among population samples of UK adults and rural and urban Cameroonians using doubly labeled water as the criterion standard (Stegle et al., 2008; Assah et al., 2011b; Brage et al., 2015). Finally, although the categorization of PAEE into quintiles attenuates statistical power, it has decent representation to prevent exposure misclassification, but we acknowledge that the observed association between activity and cardiometabolic risk is likely still attenuated by some measurement error, for example, owing to between-seasonal variation in physical activity.

Identifying appropriate interventions on cardiometabolic risk clustering remains up for debate. First, an area of disagreement lies in the relevance of the metabolic syndrome as a condition. The fact that there is an association between metabolic risk clustering, incident CVD, and CVD mortality is not in doubt—several meta-analyses have indicated that the increase in CVD risk from having concomitantly raised levels across multiple metabolic risk factors, syndrome or not, is over 50% (Gallasi et al., 2006; Gami et al., 2007). However, quantifying the precise degree of this association has been problematic. There is the underlying assumption that the metabolic syndrome confers greater CVD risk than the sum of its components, which remains unverified (Mente et al., 2010), and the pathophysiological basis for the metabolic syndrome has also not been well-characterized.

Second, recommendations for reducing cardiometabolic risk based on PAEE are limited to only a few studies that specify the most appropriate duration and type of PAEE engaged. For instance, it is unknown if physical activity recommendations should be to increase total energy expenditure, or to raise fitness overall by engaging in shorter periods

of vigorous activity (Wareham et al., 1998). Additional analyses of our data (eg, using PAEE intensity patterns) can be conducted to shed further light on these questions.

4.4 | Implications for lifestyle interventions

Despite these concerns, reducing cardiometabolic risk clustering, either across the board or by targeting only some of its constituent parts, has been shown effective in randomized controlled trials for the primary prevention of CVDs (Andrews et al., 2011; Ebrahim et al., 2011). Usually, these reductions are achieved through lifestyle interventions in the form of healthier diets and/or increased physical activity. Although it is the topic of much heated debate, which lifestyle intervention is most risk-neutralizing, increasing physical activity may confer some unique advantages, especially in developing countries where changes in diet, for example, reductions in energy intake as often advocated in Western interventions, may not be possible or even appropriate. Physical activity as a lifestyle intervention may also be more effective in communities that have traditionally engaged in more physical activity, as the intervention calls for a continuation in established behavior rather than a behavioral change.

What should the public health approach be toward reducing CVD, the metabolic syndrome, and cardiometabolic risk factors in developing countries? Screening for the metabolic syndrome is one method to identify those at high risk for CVD; however, identifying high-risk groups does not seem to be substantially more effective than current population-based approaches, and likely advocating lifestyle modifications, such as increasing PAEE, would be characteristically "too little too late." Facilities must exist that can diagnose and treat disease; in developing countries and especially in rural areas, this may not be feasible. The preliminary evidence here indicates that advocating a population-based increase in general PAEE may be more appropriate than more targeted measures, yet the approach can still be targeted in some ways—emphasizing PAEE among those with high central adiposity, for instance, which would exert a differential effect on CVD risk reduction. More studies to establish the causal relationship between PAEE and cardiometabolic risk clustering need to be conducted, as well as studies to evaluate the impact on incident CVD in developing countries. With continued research, effective strategies for reversing the increase in CVD prevalence may be implemented in the developing world.

CONFLICT OF INTEREST

We declare no competing interests.

AUTHOR CONTRIBUTIONS

SB and JCL conceived the hypothesis for the manuscript and designed the present project. MKB, FKK, and DLC collected the data and KW processed the activity data. JCL performed the data analysis and drafted the manuscript. All authors discussed the results, commented on the manuscript, and approved the final version. DLC, HF, IT, DLM, MKB, and SB contributed to the overall design of The Kenya Diabetes Study.

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