

# Urbanization, Physical Activity, and Metabolic Health in Sub-Saharan Africa

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**OBJECTIVE**—We examined the independent associations between objectively measured free-living physical activity energy expenditure (PAEE) and the metabolic syndrome in adults in rural and urban Cameroon.

**RESEARCH DESIGN AND METHODS**—PAEE was measured in 552 rural and urban dwellers using combined heart rate and movement sensing over 7 continuous days. The metabolic syndrome was defined using the National Cholesterol Education Program-Adult Treatment Panel III criteria.

**RESULTS**—Urban dwellers had a significantly lower PAEE than rural dwellers ( $44.2 \pm 21.0$  vs.  $59.6 \pm 23.7$  kJ/kg/day,  $P < 0.001$ ) and a higher prevalence of the metabolic syndrome (17.7 vs. 3.5%,  $P < 0.001$ ). In multivariate regression models adjusted for possible confounders, each kJ/kg/day of PAEE was associated with a 2.1% lower risk of prevalent metabolic syndrome (odds ratio 0.98,  $P = 0.03$ ). This implies a 6.5 kJ/kg/day difference in PAEE, equivalent to 30 min/day of brisk walking, corresponds to a 13.7% lower risk of prevalent metabolic syndrome. The population attributable fraction of prevalent metabolic syndrome due to being in the lowest quartile of PAEE was 26.3% (25.3% in women and 35.7% in men).

**CONCLUSIONS**—Urban compared with rural residence is associated with lower PAEE and higher prevalence of metabolic syndrome. PAEE is strongly independently associated with prevalent metabolic syndrome in adult Cameroonians. Modest population-wide changes in PAEE may have significant benefits in terms of reducing the emerging burden of metabolic diseases in sub-Saharan Africa.

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The burden of cardiovascular and other noncommunicable diseases is rising rapidly in developing countries (1). This epidemiological transition is characterized by the increasing prevalence of etiological risk factors such as obesity, diabetes, and high blood pressure (2). The clustering of these factors, termed the metabolic syndrome, confers a greater risk of premature morbidity and mortality (3). Epidemiological studies suggest that a significant part of the cardiovascular disease (CVD) epidemic is attributable to changes in lifestyle risk factors, exemplified by reduction in physical activity and

increased consumption of high-energy processed foods (2).

Rapid urbanization in many sub-Saharan African countries may contribute to the epidemiological transition in the region. Previous studies demonstrate a positive rural-urban gradient in terms of the prevalence of risk factors (4,5). Etiological risk factors such as obesity, glucose intolerance, and high blood pressure are measured precisely with standardized methods in epidemiological studies. Therefore, these data are relatively valid and comparable across studies and different populations. However, lifestyle factors

such as physical activity and diet are complex behaviors that are difficult to measure in free-living individuals. Few studies examining the association between physical activity with CVD risk factors in sub-Saharan African countries have used an objective measure of free-living physical activity (6,7), and none of these included both urban and rural dwellers. Because of the complexity of measuring physical activity objectively, most previous studies have used self-report questionnaires to estimate free-living physical activity (8,9).

The use of physical activity questionnaires in free-living population studies introduces the potential for error and recall bias, as well as other differential misclassifications that may result from the dimension or domain of activity assessed by the questionnaire (10,11). Objective measurement of free-living physical activity and its association with health outcomes is therefore imperative for quantifying dose-response associations and accurately monitoring longitudinal trends or the effects of public health interventions. Only a few studies (12,13) have reported on objectively measured free-living physical activity and its association with the metabolic syndrome—none of them were in an African population.

The aim of this cross-sectional study was to examine the independent associations between free-living physical activity energy expenditure (PAEE) and the metabolic syndrome in adult Cameroonians.

## RESEARCH DESIGN AND METHODS

Participants for this study were recruited from two urban areas (Yaoundé, the capital city of Cameroon, and Bamenda, the capital city of the North Western region) and two rural areas (Mbankomo and Bafut) of Cameroon. The inhabitants of the urban sites are mainly middle-class public or private sector workers or businessmen. Residential houses in the urban areas are grouped closely together and arranged in blocks with paved or graded road access. In the rural areas, the inhabitants are mainly farmers and/or petty traders. The rural habitation pattern is sparse, with most of the homes and farms accessed only via foot paths.

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As a result of the absence of complete population registers, a sampling frame was established following enumeration of eligible adults in houses in delimited areas of the study sites. Eligible participants were adults aged between 25 and 55 years without previously diagnosed diabetes or any known cardiovascular disease. All 3,854 eligible participants (rural:  $N = 2,238$ , mean age  $35.3 \pm 7.7$  years; urban:  $N = 1,616$ , mean age  $35.4 \pm 8.3$  years) in the sampling frame were approached through door-to-door recruitment and invited to participate in the study. A total of 651 volunteers (rural:  $N = 303$ , mean age  $38.5 \pm 8.3$  years; urban:  $N = 348$ , mean age  $37.9 \pm 9.1$  years) took part in this study. Volunteers were provided detailed information about the study verbally and in writing, and invited to attend a testing session at their local hospital. Volunteers were asked to refrain from eating, drinking or smoking starting at 10:00 P.M. the evening prior to testing. Personal and sociodemographic data, as well as data on smoking, alcohol consumption, and fruit and vegetable intake were collected by self-report using an adapted World Health Organization (WHO) STEPwise Approach to Surveillance questionnaire.

Ethical approval for the study was obtained from the Cameroon National Ethics Committee, and all participants provided signed informed consent. Pregnant women and other volunteers who could not take part in exercise testing according to study protocol were excluded from the study. An exercise screening questionnaire, adapted from the WHO Rose angina questionnaire was used to screen for preexisting cardiovascular disease which would contraindicate exercise testing.

### Physical activity measurement

Free-living physical activity was measured using a combined heart rate (HR) and movement sensor (Actiheart; Cambridge Neurotechnology, Cambridge, U.K.) which has been validated in this population in free-living adults with doubly labeled water method as criterion (14). The individual HR response to the mechanical work during an 8-min incremental step test was used for individual calibration of HR data (15). During the test, HR was monitored in real time and the test was aborted if HR reached 90% of the age-predicted maximum HR (16), if the subject could not keep up with the stepping protocol, or if they requested to stop.

Free-living physical activity was measured in 1-min epochs using the combined

HR and movement sensor over 7 continuous days. The participants were requested to carry on with their normal habitual lifestyle and wear the monitor at all times except while showering, bathing, or swimming. The mean  $\pm$  SD worn duration was  $6.3 \pm 1.6$  days. Participants with less than 2 full days of free-living data ( $n = 51$ ) were not included in these analyses. HR data were preprocessed (17) and converted to energy expenditure using the individual calibration derived from the step test, and the accelerometer data were converted to energy expenditure using group acceleration equations corresponding to level walking or running (15). Minute-by-minute HR and movement-derived physical activity intensity was combined in a branched equation model (18) to calculate free-living PAEE. Basal metabolic rate ( $\sim$ resting energy expenditure [REE]) was estimated using the Oxford equations (19), and total energy expenditure (TEE) was calculated by adding REE and a component for the thermic effect of food (equivalent to 10% of TEE) to the derived PAEE. Energy expenditure variables are expressed per kilogram body weight to adjust for differences in body size. The physical activity level was calculated as TEE/REE. The distribution of intensity of activity was expressed as average daily time (min/day) spent at 1 MET equal to time spent asleep or sedentary, between 1 and 3 METs equal to light physical activity, above 3 METs equal to moderate-to-vigorous physical activity. An estimate of cardiorespiratory fitness ( $VO_{2max}$  in mL of  $O_2$ /kg/min) was calculated as the intensity of work at the age-predicted maximum heart rate (15) by extrapolation of the individual heart rate response to mechanical work derived from the step test calibration.

### Anthropometry and clinical measures

Height was measured without shoes using a standard rigid stadiometer. Body weight was measured using electronic clinical scales, and body composition was measured using bio-impedance (Tanita TBF-531 scales; Tanita UK, Uxbridge, Middlesex, U.K.). BMI was calculated as the ratio of body weight (kg) to the square of height ( $m^2$ ) and categorized as normal:  $<25$ ; overweight:  $\geq 25$  to  $<30$ ; and obese:  $\geq 30$   $kg/m^2$ .

Waist circumference was measured at the level of the midpoint between the lower costal margin and the anterior superior iliac crests. Measurement was made to the

nearest 0.1 cm using a nonstretch fiberglass tape measure with participants wearing light indoor clothing. Central obesity was defined as waist circumference  $\geq 80$  cm in women or  $\geq 94$  cm in men, or using the cut points of the National Cholesterol Education Program-Adult Treatment Panel III (NCEP-ATP III) (20) when included to define the metabolic syndrome.

Blood pressure was measured using the Omron M4-1 automatic blood pressure monitor. Measurements were made on the dominant arm after 5 min of rest with the subject seated. Three measurements were taken at 1-min intervals, and the average of the three constituted the final blood pressure value.

A standard oral glucose tolerance test was carried out in all participants. Capillary blood glucose readings were measured after an overnight fast of at least 8 h, and then 2 h after ingestion of 75 g of glucose dissolved in 300 mL of water. Blood glucose measurements were done on whole fresh capillary blood using a Hemocue B-Glucose Analyzer (HemoCue AB, Ängelholm, Sweden).

### Metabolic syndrome and clustered metabolic risk score

The metabolic syndrome was defined using the NCEP-ATP III criteria (20). To demonstrate the population distribution of global metabolic health, we computed a continuous summary metabolic risk score ( $z$ -score) largely on the basis of the components of the metabolic syndrome. This variable was derived by standardizing and then summing waist circumference, blood pressure (average of systolic and diastolic), blood glucose (average of fasting, 30-min, and 2-h glucose), triglycerides, body fat, fasting insulin, and inverse HDL cholesterol. The standardization of these continuous variables was computed by subtracting the sample mean from the individual value and then dividing by the SD of the sample mean. Sex-specific calculations were made for waist circumference, body fat, fasting insulin, and HDL cholesterol.

### Biochemical assays

Fasting blood samples were collected in the mornings between 7:30 and 9:30 A.M. Plasma and serum samples were separated upon collection in a refrigerated centrifuge at 4°C. After centrifugation at  $\sim 1,400g$ , samples were aliquoted and stored at  $-80^\circ C$ . Samples were transported on dry ice by air to Cambridge (U.K.)

and stored at  $-80^{\circ}\text{C}$  until further analyses. Assays were performed by the National Institute for Health Research Cambridge Biomedical Research Centre, Core Biochemical Assay Laboratory. HDL cholesterol and triglycerides were measured using automated assays on the Dade Behring Dimension RxL analyzer (Siemens Healthcare, Camberley, U.K.). HDL was measured using a homogenous accelerator selective detergent assay and triglycerides were measured using an enzymatic assay according to manufacturers' specifications. LDL was calculated by the Friedwald formula ( $\text{LDL} = \text{Cholesterol} - \text{HDL} - [\text{Triglycerides}/2.2]$ ). Fasting insulin was assayed in singleton on a 1235 AutoDELFIA automatic immunoassay system using a two-step time resolved fluorometric assay kit manufactured by Perkin Elmer Life Sciences (Wallac Oy, Turku, Finland). Cross-reactivity with intact proinsulin was  $<0.5\%$  at 2,736 pmol/L, 32–33 split proinsulin 1% at 2,800 pmol/L, and C-peptide  $<0.1\%$  at 5,280 pmol/L.

### Calculations and statistical analyses

Analyses were carried out using STATA version 10.1 (StataCorp, College Station, TX). Where applicable, the level of statistical significance was considered at  $P < 0.05$ .

Descriptive characteristics of the study sample are presented as means with SD, adjusted means with SE, or as numbers with proportions, stratified by sex-specific PAEE quartiles. For dichotomous

grouping variables, difference in means was assessed using the Student *t* test and difference in proportion using the  $\chi^2$  test or Fisher exact test. Linear trends across PAEE quartiles were assessed by linear regression.

Independent associations between PAEE and the metabolic syndrome were examined in multivariate logistic regression models. There was no PAEE interaction by residential site or sex on its association with the metabolic syndrome. Models were either unadjusted or adjusted for age, sex, residential site, smoking, alcohol consumption, fruit and vegetable consumption, and educational level (as a surrogate for socioeconomic status). To illustrate the population impact of physical inactivity on metabolic health, we calculated the population attributable fraction of prevalent metabolic syndrome due to being in the lowest quartile of PAEE (Q1-PAEE). The population attributable fraction was computed as  $\frac{\text{prevalence of Q1-PAEE in case subjects} - \text{prevalence of Q1-PAEE in control subjects}}{1 - \text{prevalence of Q1-PAEE in control subjects}}$ .

**RESULTS**—Descriptive characteristics of the study sample stratified by sex and rural or urban residence are presented in Supplementary Table 1. Clinical and metabolic characteristics of the study sample stratified by sex-specific quartiles of PAEE are presented in Table 1. Most of the individual metabolic and clinical parameters, as well as the clustered metabolic

risk score or prevalence of the metabolic syndrome showed a significant linear trend across quartiles of PAEE. Blood lipids were generally low in this sample with no sex or rural-urban differences. Prevalence of metabolic syndrome was almost fourfold lower in the highest compared with the lowest quartile of PAEE.

Urban dwellers had a significantly lower PAEE than rural dwellers ( $44.2 \pm 21.0$  vs.  $59.6 \pm 23.7$  kJ/kg/day,  $P < 0.001$ ) and a higher prevalence of the metabolic syndrome ( $17.7$  vs.  $3.5\%$ ,  $P < 0.001$ ) (Supplementary Tables 1 and 2). There was no significant difference between the sexes in metabolic risk expressed as a continuous clustered metabolic score, but urban dwellers had a significantly higher mean score than rural dwellers ( $0.80 \pm 3.67$  vs.  $-1.11 \pm 2.90$ ,  $P < 0.001$ ). This difference was observed to correspond to a rural-to-urban right shift in the population density distribution of the clustered metabolic *z*-score when stratified by residential area (Fig. 1). A similar but reverse difference was observed with the population distribution of PAEE, with a rural-to-urban left shift in the distribution.

Table 2 presents independent associations between PAEE quartiles and prevalent metabolic syndrome. With the lowest quartile of PAEE as reference, there was a strong inverse association between PAEE and prevalent metabolic syndrome. Compared with the lowest quartile of PAEE, being in the 2nd quartile of PAEE was associated with a reduction in the risk

**Table 1—Clinical and metabolic characteristics by quartiles of PAEE in adult Cameroonians (n = 552)**

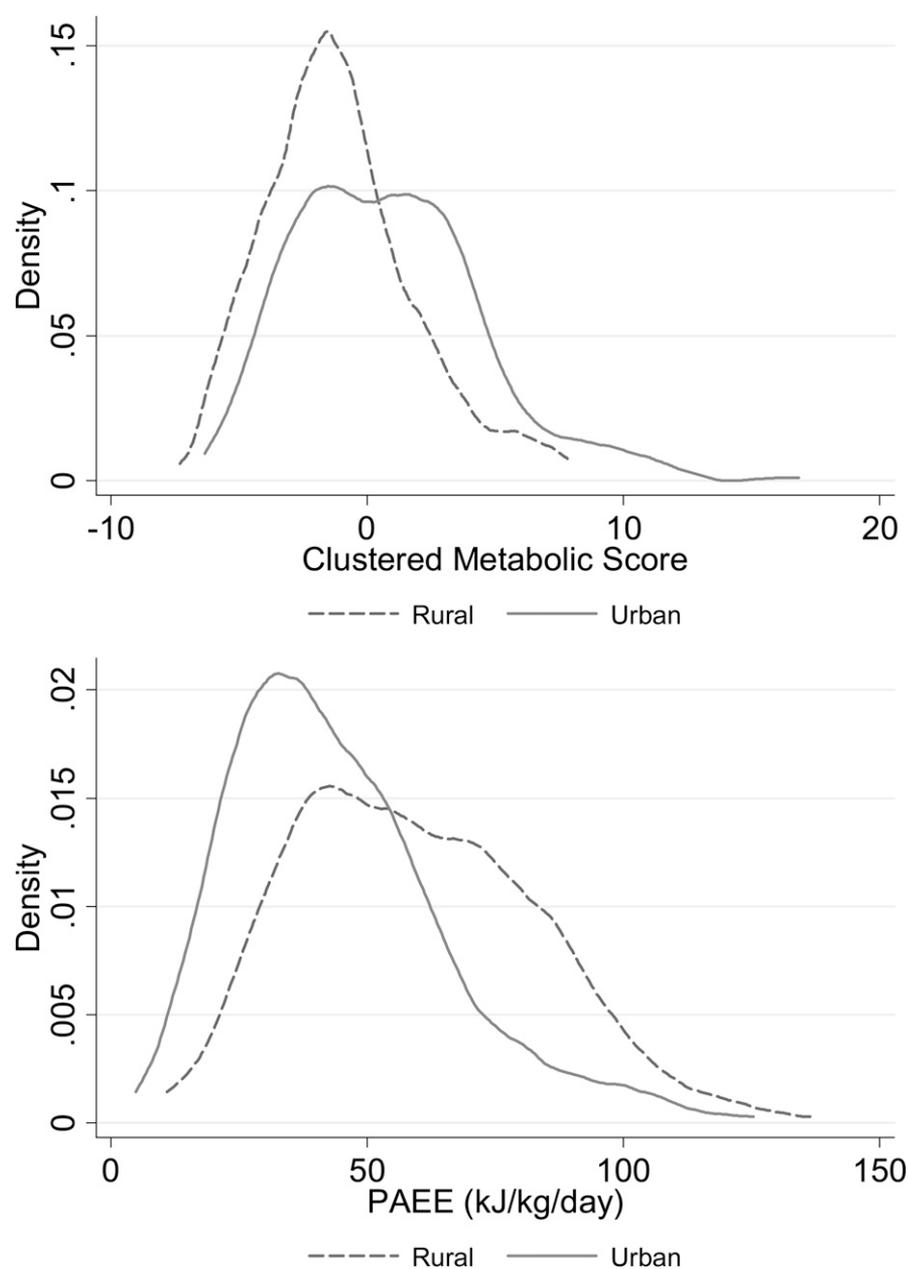
	Quartiles of PAEE				P
	Q1	Q2	Q3	Q4	
PAEE (kJ/kg/day)	25.8 $\pm$ 0.9	40.8 $\pm$ 0.9	54.9 $\pm$ 0.9	82.7 $\pm$ 0.9	<0.001
Age (years)	41.6 $\pm$ 0.7	38.7 $\pm$ 0.8	36.1 $\pm$ 0.7	37.7 $\pm$ 0.7	<0.001
Education (years)	12.6 $\pm$ 0.4	11.1 $\pm$ 0.4	10.1 $\pm$ 0.4	8.2 $\pm$ 0.4	<0.001
BMI (kg/m <sup>2</sup> )	28.0 $\pm$ 0.4	27.3 $\pm$ 0.4	25.3 $\pm$ 0.4	23.4 $\pm$ 0.4	<0.001
WC (cm)	93.3 $\pm$ 0.9	90.9 $\pm$ 0.9	86.8 $\pm$ 0.9	82.3 $\pm$ 0.9	<0.001
Body fat (%)	31.0 $\pm$ 0.9	30.0 $\pm$ 0.9	27.7 $\pm$ 0.9	23.7 $\pm$ 0.9	<0.001
Diastolic BP (mmHg)	81.8 $\pm$ 1.1	77.7 $\pm$ 1.0	74.0 $\pm$ 1.0	71.4 $\pm$ 1.0	<0.001
Systolic BP (mmHg)	129.1 $\pm$ 1.6	124.3 $\pm$ 1.6	119.7 $\pm$ 1.6	117.1 $\pm$ 1.6	<0.001
Fasting BG (mmol/L)	5.0 $\pm$ 0.1	4.7 $\pm$ 0.1	4.6 $\pm$ 0.1	4.7 $\pm$ 0.1	0.01
2-h BG (mmol/L)	6.5 $\pm$ 0.2	6.3 $\pm$ 0.2	6.2 $\pm$ 0.2	6.2 $\pm$ 0.2	0.1
HDL cholesterol (mmol/L)	1.23 $\pm$ 0.03	1.23 $\pm$ 0.03	1.25 $\pm$ 0.03	1.19 $\pm$ 0.03	0.4
Triglycerides (mmol/L)	0.93 $\pm$ 0.04	0.83 $\pm$ 0.04	0.82 $\pm$ 0.04	0.80 $\pm$ 0.04	0.09
Fasting insulin (pmol/L)	34.1 $\pm$ 2.8	35.9 $\pm$ 2.7	24.9 $\pm$ 2.7	17.7 $\pm$ 2.6	<0.001
<i>z</i> MS ( <i>z</i> -score)	1.78 $\pm$ 0.27	0.65 $\pm$ 0.27	-0.81 $\pm$ 0.27	-1.77 $\pm$ 0.26	<0.001
MS	23 (20)	17 (14)	9 (7)	6 (5)	<0.001

Data are age-adjusted means  $\pm$  SE or *n* (%). *P* values for linear trend across quartiles of PAEE. BG, blood glucose; BP, blood pressure; MS, metabolic syndrome; WC, waist circumference; *z*MS, clustered metabolic risk score.

amount of PAEE can be achieved by 30 min/day of brisk walking.

**CONCLUSIONS**—Our results suggest that free-living PAEE was significantly and independently associated with prevalent metabolic syndrome. This association persisted after multivariate adjustments for possible confounders and was similar in both urban and rural dwellers, even though rural dwellers were more physically active and had a more favorable metabolic profile than urban dwellers. Low levels of physical activity may be contributing significantly to the metabolic syndrome in this population. Theoretically, these data suggest a 26.3% population attributable fraction of prevalent metabolic syndrome as a result of being in the lowest quartile of PAEE. However, independent of several confounders, it is likely that small changes in PAEE may confer significant benefits in terms of lower risk of metabolic syndrome.

Because of the cross-sectional design of our study, we cannot infer causality. Further, generalization of these results to other populations should be made with caution. The study sample included only adults aged 25–55 years without known metabolic syndrome or CVD. Therefore, these results may not be directly generalizable to younger or older individuals, as well as individuals with diagnosed prevalent metabolic or cardiovascular disease. The lack of complete population registers as well as the absence of fixed house addresses or telephone numbers limited our ability to use simple random sampling of participants. Our use of volunteers from a study-established sampling frame may introduce sampling bias, which would limit the generalizability of these results even to our target population. However, our sample was similar to other larger population-based surveys with respect to BMI, blood pressure, and educational level (21,22). Separate from prevalence of risk factors, one could argue that educational level may have influenced volunteering for this study. However, the proportion of volunteers in this sample having at least a secondary education (44% of men and 35% of women) was comparable to data from the 2004 demographic and health survey in Cameroon (48% of men and 32% of women) (21). The observed inverse association between total volume of physical activity (PAEE) with the metabolic syndrome is comparable to results from previous studies which used objective measures of physical activity (12,13,23). Franks et al. (23) reported a



**Figure 1**—Population distribution of PAEE and clustered metabolic z-score in adults in rural and urban Cameroon.

of prevalent metabolic syndrome, which was not statistically significant (odds ratio 0.63 [95% CI 0.32–1.25]). The risk of prevalent metabolic syndrome was significantly lower for the 3rd and 4th quartiles of PAEE compared with the 1st quartile (0.32 [0.14–0.73] and 0.19 [0.08–0.49], respectively). Multivariate adjustments for age, sex, residential area, smoking, alcohol drinking, fruit and vegetable consumption, and educational level as potential confounders attenuated but did not change these associations.

Defining the lowest quartile of PAEE to represent sedentary or inactive lifestyle,

the population attributable fraction of prevalent metabolic syndrome due to a sedentary or inactive lifestyle was 26.3% (25.3% in women and 35.7% in men). Expressing PAEE as a continuous variable, and assuming a linear relationship with the metabolic syndrome, each kJ/kg/day of PAEE was associated with a 2.1% lower risk of prevalent metabolic syndrome (odds ratio 0.98,  $P = 0.03$ ), after adjustment for potential confounders as in Table 2, model 3. This implies that an increase of 6.5 kJ/kg/day of PAEE would correspond to approximately 13.7% risk reduction in prevalent metabolic syndrome. This

**Table 2—Independent associations between PAEE and metabolic syndrome in adult Cameroonians (n = 552)**

Sex-specific PAEE quartiles	Metabolic syndrome
Model 1* ( $P < 0.001$ )	
Q1	1.00
Q2	0.63 (0.32–1.25)
Q3	0.32 (0.14–0.73)
Q4	0.19 (0.08–0.49)
Model 2† ( $P < 0.001$ )	
Q1	1.00
Q2	0.67 (0.33–1.36)
Q3	0.35 (0.15–0.82)
Q4	0.20 (0.08–0.51)
Model 3‡ ( $P = 0.027$ )	
Q1	1.00
Q2	0.87 (0.40–1.89)
Q3	0.50 (0.20–1.24)
Q4	0.33 (0.11–1.02)

Data are odds ratio or odds ratio (95% CI).  $P$  values for linear trend across quartiles of PAEE. \*Unadjusted. †Adjusted for age and sex. ‡Adjusted for age, sex, residential area, smoking, alcohol consumption, fruit and vegetable consumption, and number of years of education.

significant inverse association between PAEE scaled for fat-free body mass and a clustered metabolic syndrome score. However, we scaled PAEE for body weight, not fat-free mass. Although researchers agree that PAEE should be expressed taking into account differences in body size, the exact scaling factor applicable to body size is still a contentious issue. During light intensity locomotion activities, which are the main component of total activity in many people, the energy expended is predominantly to move the body weight. The expression of PAEE per kilogram body weight may be more appropriate for the habitual free-living activity pattern, and is easy to interpret for clinical or public health usage.

The importance of the distribution of free-living physical activity intensities, beyond total volume of physical activity, on metabolic health is still uncertain. Many studies that have examined the effect of intensity distribution of physical activity on global metabolic risk have used self-report of physical activity (24). Studies that have used objective assessment of physical activity intensity distribution are less common (12,13). Most studies confirm the deleterious effect of increasing sedentary time or decreasing moderate-to-vigorous physical activity on the metabolic syndrome (12,24).

However, the benefits of increasing overall activity compared with shifting the intensity distribution toward more vigorous activity is still inconclusive. In this study we show that objectively measured total volume of physical activity had a significant independent association with the metabolic syndrome. Our use of total volume of activity may be more applicable to health promotion initiatives in regions with low literacy. It may be more feasible to encourage overall physical activity than to recommend specific subdimensions of activity to less educated people.

Rapid urbanization of lifestyle in sub-Saharan Africa is characterized by increasing prevalence and clustering of metabolic risk factors such as diabetes, high blood pressure, and obesity (4,25). Our data show an evident rural-urban gradient of PAEE, which mirrors progression toward western levels of habitual physical activity. This transition is not only explained by increasing frequency of high risk individuals, but by a shift in the underlying population distribution of risk factors. Our use of an aggregate variable for metabolic health (the metabolic syndrome) is relevant for assessing the effects of physical activity level on multiple risk factors in the whole population. Further, the metabolic syndrome is associated with a higher morbidity and mortality (3). We did not collect data on energy intake in this study because of the difficulties of collecting objective dietary data in a population-based study. However, we adjusted for self-reported fruit and vegetable intake in the analyses, which would capture part of the variance associated with healthy or unhealthy dietary habits.

These data suggest a significant population burden of prevalent metabolic syndrome associated with being in the lowest quartile of PAEE, or a mean PAEE of  $26 \pm 7$  kJ/kg/day. This level of physical activity corresponds to an average of 45 MET-hours/week. Typically, these individuals would be spending more than 80% of their time in sedentary activities with very little physical effort. Encouraging people to increase their physical activity, even through reduction of sedentary time, may confer considerable health benefits especially in the least active individuals.

In conclusion, PAEE is independently associated with the metabolic syndrome in this population. Reduction in physically active lifestyles as a result of urbanization may be critical in driving the current transition toward a higher burden of metabolic diseases in this region. Public

health interventions to increase physical activity may be expected to significantly curb the burden of metabolic syndrome in sub-Saharan Africa.

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F.K.A. designed the project, collected the data, conceived the hypothesis for this manuscript, performed data analyses, and drafted the manuscript. U.E. and S.B. provided critical input for the conception of this manuscript and assisted with the editing. U.E., S.B., J.C.M., and N.J.W. assisted with the project design and critically appraised the manuscript. All authors participated in the interpretation of the results and approved the final version of the manuscript.

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