

Free-Living Physical Activity Energy Expenditure Is Strongly Related to Glucose Intolerance in Cameroonian Adults Independently of Obesity

FELIX K. ASSAH, MD¹
ULF EKELUND, PHD¹
SOREN BRAGE, PHD¹

JEAN CLAUDE MBANYA, PHD²
NICHOLAS J. WAREHAM, PHD¹

OBJECTIVE — We examined the cross-sectional association between objectively measured free-living physical activity energy expenditure (PAEE) and glucose tolerance in adult Cameroonians without known diabetes.

RESEARCH DESIGN AND METHODS — PAEE was measured in 34 volunteers using the doubly labeled water method and indirect calorimetry (resting). Fasting blood glucose and 2-h postload blood glucose were measured during a standard 75-g oral glucose tolerance test.

RESULTS — There was a significant negative correlation between PAEE and 2-h glucose ($r = -0.43$; $P = 0.01$) but not fasting glucose ($r = 0.1$; $P = 0.57$). The inverse association between PAEE and 2-h glucose remained after adjustment for age, sex, smoking, alcohol consumption, and BMI ($\beta = -0.017$ [95% CI -0.033 to -0.002]) and was unchanged after further adjustment for waist circumference, body fat percentage, or aerobic fitness.

CONCLUSIONS — PAEE is inversely associated with 2-h glucose independently of adiposity or fitness. Interventions aimed at increasing PAEE could play an important role in diabetes prevention in developing countries.

Diabetes Care 32:367–369, 2009

Developing countries are undergoing a rapid epidemiologic transition characterized by rising prevalence of obesity, diabetes, and cardiovascular diseases (CVDs). Recent changes in diet and physical activity patterns have been suggested as possible risk factors (1). It is important to understand the association between modifiable exposure variables and risk factors for chronic diseases in order to design appropriate intervention strategies. We examined the cross-sectional association between physical activity energy expenditure (PAEE) and glucose tolerance in nondiabetic adult Cameroonians.

RESEARCH DESIGN AND METHODS

A cross-sectional study of 17 men and 17 women recruited from an urban and a rural residential area of Cameroon was conducted. Ethical approval for the study was obtained from the Cameroon National Ethics Committee, and all participants provided signed informed consent.

Height and waist circumference were measured using standard clinical procedures. Body weight was measured using an electronic scale (Tanita TBF-531; Tanita U.K., Middlesex, U.K.). Total body water (TBW) was measured by deuterium dilution. Fat-free mass was calculated

from TBW, assuming a hydration factor of 73%, and fat mass derived as the difference between body weight and fat-free mass.

Resting energy expenditure (REE) was measured using the MedGem hand-held indirect calorimeter (HealthTech Inc., Golden, CO). Total energy expenditure (TEE) was measured by the doubly labeled water (DLW) method. Two baseline urine samples were collected on separate days before the administration of a standard dose of DLW (174 mg/kg body wt of oxygen-18 and 70 mg/kg body wt of deuterium). Postdose urine samples were collected daily for the next 6 days. TEE was calculated using Schoeller's estimation of CO₂ production, assuming a respiratory quotient of 0.85. PAEE ($\text{kJ} \cdot \text{day}^{-1} \cdot \text{kg}^{-1}$) was calculated as $0.9 \times \text{TEE} - \text{REE}$, taking the thermogenic effect of food into account. Aerobic fitness was estimated by linear extrapolation of the individually observed heart-rate response to a standardized step test up to the age-predicted maximum heart rate.

Capillary blood glucose was measured after an overnight fast (fasting blood glucose [FBG]) and then 2 h after ingestion of 75 g glucose dissolved in 250–300 ml water (2-h postload blood glucose [2-h BG]). This was done using a HemoCue B-Glucose Analyzer (HemoCue, Ängelholm, Sweden).

Statistical analyses were done using STATA (version 10 special edition; StataCorp, College Station, TX). Student's *t* test was used to assess differences in the descriptive variables. Independent associations of PAEE with FBG and 2-h BG were assessed by multiple linear regression analyses.

RESULTS — The mean \pm SD age of the study participants was 34.5 ± 7.5 years. Body fat was significantly lower in men than in women (18.5 ± 9.3 vs. $34.6 \pm 7.2\%$; $P < 0.001$); however, the difference in BMI was of borderline significance (25.4 ± 3.6 vs. $28.4 \pm 5.1 \text{ kg/m}^2$; $P = 0.05$).

From the ¹Medical Research Council Epidemiology Unit, Institute of Metabolic Science, Addenbrooke's Hospital, Cambridge, U.K.; and the ²Faculty of Medicine and Biomedical Sciences, University of Yaounde 1, Yaounde, Cameroon.

Corresponding author: Ulf Ekelund, ulf.ekelund@mrc-epid.cam.ac.uk.

Received 21 August 2008 and accepted 11 November 2008.

Published ahead of print at <http://care.diabetesjournals.org> on 18 November 2008. DOI: 10.2337/dc08-1538.

© 2009 by the American Diabetes Association. Readers may use this article as long as the work is properly cited, the use is educational and not for profit, and the work is not altered. See <http://creativecommons.org/licenses/by-nc-nd/3.0/> for details.

The costs of publication of this article were defrayed in part by the payment of page charges. This article must therefore be hereby marked "advertisement" in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

Table 1—Independent effect of objectively measured free-living PAEE on FBG and 2-h BG

	FBG (mmol/l)				2-h BG (mmol/l)			
	β	95% CI	P	R ²	β	95% CI	P	R ²
Model 1				0.01				0.19
PAEE (kJ · kg ⁻¹ · day ⁻¹)*	0.002	−0.004 to 0.008	0.53		−0.016	−0.028 to −0.004	0.01	
Model 2				0.12				0.30
PAEE (kJ · kg ⁻¹ · day ⁻¹)	0.004	−0.004 to 0.012	0.30		−0.017	−0.033 to −0.002	0.03	
BMI (kg/m ²)	0.026	−0.042 to 0.094	0.44		0.015	−0.117 to 0.147	0.82	
Model 3				0.19				0.30
PAEE (kJ · kg ⁻¹ · day ⁻¹)	0.005	−0.003 to 0.013	0.18		−0.018	−0.033 to −0.003	0.02	
Waist (cm)	0.024	−0.005 to 0.053	0.10		0.003	−0.055 to 0.062	0.91	
Model 4				0.20				0.30
PAEE (kJ · kg ⁻¹ · day ⁻¹)	0.008	−0.001 to 0.017	0.08		−0.018	−0.037 to 0	0.05	
Body fat (%)	0.037	−0.005 to 0.078	0.08		−0.004	−0.087 to 0.08	0.93	
Model 5				0.16				0.30
PAEE (kJ · kg ⁻¹ · day ⁻¹)	0.005	−0.003 to 0.012	0.22		−0.019	−0.034 to −0.003	0.02	
Vo _{2max} (ml · kg ⁻¹ · min ⁻¹)	−0.024	−0.061 to 0.013	0.19		0.009	−0.064 to 0.081	0.81	

All models included 34 participants. Model 1 is unadjusted. Models 2–5 are adjusted for age, sex, smoking, and alcohol consumption. *Post hoc power calculation indicated a 10% power to refute the null hypothesis of no association between PAEE and FBG (the CI of the effect estimate includes zero). The association between PAEE and 2-h BG had a 76% power, even though the CI of this association was wider than that of the PAEE vs. FBG association. β , regression coefficient; R², variance.

TEE (191.4 ± 55.9 vs. 143 ± 22.6 kJ · kg⁻¹ · day⁻¹; P = 0.002) and PAEE (73.9 ± 49.7 vs. 45.4 ± 17.4 kJ · kg⁻¹ · day⁻¹; P = 0.03) were significantly higher in men than in women. There was no difference in mean FBG (4.3 ± 0.7 vs. 4.6 ± 0.6 mmol/l; P = 0.21) or 2-h BG (5.9 ± 1.8 vs. 6.3 ± 1.1 mmol/l; P = 0.43) between men and women.

BMI, waist circumference, and body fat were not significantly correlated with FBG or 2-h BG. PAEE was not significantly correlated with FBG (r = 0.11; P = 0.53) but inversely correlated with 2-h BG (r = −0.43; P = 0.011).

In unadjusted linear regression analyses (Table 1), PAEE was significantly negatively associated with 2-h BG (β = −0.016 [95% CI −0.028 to −0.004]). This association remained largely unchanged when adjusted for age, sex, smoking, and alcohol consumption (−0.018 [−0.032 to −0.004]). Further adjustments for BMI, waist circumference, body fat, or Vo_{2max} (Table 1) did not change the results. There was no significant sex interaction.

CONCLUSIONS— In a sample of nondiabetic Cameroonian adults with BMI comparable with that of national survey data (2), we observed a strong inverse association between PAEE and 2-h plasma glucose but not fasting plasma glucose independent of obesity/adiposity. This association was unchanged after adjustment for age, sex, smoking, and alcohol consumption as

confounders and also was not affected by adjustment for cardiorespiratory fitness. There were borderline significant associations between PAEE and body fat with FBG, but no significant associations were observed among BMI, waist circumference, or cardiorespiratory fitness with FBG or 2-h BG in this study. The difference in pattern of association between PAEE and 2-h BG or FBG may relate to the fact that these biochemical parameters reflect different pathophysiological processes (3). Because the risk of future diabetes, as well as CVD outcomes, is more closely associated with impaired glucose tolerance than with impaired fasting glucose (4), our observations have public health importance for the prevention of these metabolic disorders.

The cross-sectional nature of these analyses limits any inference about the direction of causality. However, the results from trials are unequivocal about the role of physical inactivity in the development of diabetes (5). The small sample size of this study could have limited the statistical power to detect an association between PAEE and 2-h BG. However, the precise measurement of the exposure, as used in the present study, acts to increase statistical power and may be one explanation for our ability to detect this association in a small study.

Our results are in agreement with other studies in Caucasians using objective measurement of physical activity with adjustment for measurement error (6).

They are also in agreement with studies in other ethnic groups, which have used less precise measures of PAEE but larger samples (7).

This is the first report of objectively measured free-living PAEE and its association with glucose tolerance in an African population. These results suggest that low levels of PAEE may be a factor underlying the rise of diabetes in this population (8). Public health efforts to increase overall PAEE could play an important role in diabetes prevention and CVD reduction in developing countries.

Acknowledgments— No potential conflicts of interest relevant to this article were reported.

References

- Joubert J, Norman R, Lambert EV, Groenewald P, Schneider M, Bull F, Bradshaw D: Estimating the burden of disease attributable to physical inactivity in South Africa in 2000. *S Afr Med J* 97:725–731, 2007
- Fezeu LK, Assah FK, Balkau B, Mbanya DS, Kengne AP, Awah PK, Mbanya JC: Ten-year changes in central obesity and BMI in rural and urban Cameroon. *Obesity (Silver Spring)* 16:1144–1147, 2008
- Meyer C, Pimenta W, Woerle HJ, Van Haften T, Szoke E, Mitrakou A, Gerich J: Different mechanisms for impaired fast-

- ing glucose and impaired postprandial glucose tolerance in humans. *Diabetes Care* 29:1909–1914, 2006
4. Unwin N, Shaw J, Zimmet P, Alberti KG: Impaired glucose tolerance and impaired fasting glycaemia: the current status on definition and intervention. *Diabet Med* 19:708–723, 2002
 5. Gillies CL, Abrams KR, Lambert PC, Cooper NJ, Sutton AJ, Hsu RT, Khunti K: Pharmacological and lifestyle interventions to prevent or delay type 2 diabetes in people with impaired glucose tolerance: systematic review and meta-analysis. *BMJ* 334:299, 2007
 6. Wareham NJ, Wong MY, Day NE: Glucose intolerance and physical inactivity: the relative importance of low habitual energy expenditure and cardiorespiratory fitness. *Am J Epidemiol* 152:132–139, 2000
 7. Pereira MA, Kriska AM, Joswiak ML, Dowse GK, Collins VR, Zimmet PZ, Gareeboo H, Chitson P, Hemraj F, Purran A, et al.: Physical inactivity and glucose intolerance in the multiethnic island of Mauritius. *Med Sci Sports Exerc* 27:1626–1634, 1995
 8. Sobngwi E, Mbanya JC, Unwin NC, Porcher R, Kengne AP, Fezeu L, Minkoulou EM, Tournoux C, Gautier JF, Aspray TJ, Alberti K: Exposure over the life course to an urban environment and its relation with obesity, diabetes, and hypertension in rural and urban Cameroon. *Int J Epidemiol* 33:769–776, 2004