Original Article

Cross-sectional study of diet and risk factors for metabolic diseases in a Ghanaian population in Sydney, Australia

Ashraf Saleh[1] M Nutr Sc, Soumela Amanatidis[1,2] MPH, Dip Nutr Diet and Samir Samman[1] PhD

[1]*Human Nutrition Unit, Department of Biochemistry, University of Sydney, NSW, Australia*
[2]*Health Promotion Unit, Central Sydney Area Health Service, NSW, Australia*

Ethnic groups in affluent environments experience higher rates of metabolic diseases than their native counterparts. Our objective was to determine the prevalence of metabolic risk factors in Ghanaians in Sydney, and to investigate the relationship with dietary and lifestyle factors. Cross-sectional design with anthropometry, blood pressure, plasma lipids, glucose and insulin concentrations were measured on two occasions on each subject. Dietary information was obtained by three 24-h dietary recalls. Adults (45 male, 35 female) were recruited from a local association in Sydney, Australia. Overweight was observed in a large proportion of subjects (71% and 66% of men and women, respectively), with 18% of men and 26% of women classified as obese. Abdominal overweight was seen in 63% and 74% of men and women, respectively. Abdominal obesity was seen in 20% of men and 49% of women. Hypertension was detected in 40% of men and 17% of women, 16% of men and 6% of women were diagnosed with definite hypertension. Seventy-one per cent of men and 29% of women were classified as hypercholesterolaemic and 67% of men and 23% of women had elevated low-density lipoprotein cholesterol. In men, low high-density lipoprotein cholesterol and hypertriacylglycerolaemia affected 18% and 13%, respectively. Fasting hyperinsulinaemia was observed in 14% and 9% of men and women, respectively. The majority of subjects (73%) sustained one or more metabolic risk factors. Dietary fat contributed 33% and 35% of total energy intake in men and women, respectively, saturated fat contributing 11% in both sexes. A high prevalence of overweight, diabetes, hypertension and dyslipidaemia exists in this population, particularly in men, highlighting the need for intervention.

Key words: Australia, cholesterol, diabetes, diet, Ghana, hypertension, lifestyle, metabolic risk factors, migration, obesity, Sydney.

Introduction

Insulin resistance and hyperinsulinaemia expressed in West Africans[1–5] are exacerbated by increasing Westernization and duration of migration.[1,5–7] It has been reported that West Africans residing in the USA, whether of remote (African-American) or direct relation (Ghanaians), have significantly lower insulin sensitivity than Caucasians living in the same environment.[5] A combination of poor nutrition practices, sedentary lifestyle, genetic predisposition and cultural attitudes has been suggested as the cause of increased risk of developing metabolic disease among susceptible populations, such as African-Americans.[8]

Insulin resistance is accepted as a major contributor to the development of hypertension,[3,9] obesity[10] and type 2 diabetes mellitus (DM).[11] Despite the ample data available for African-Americans, no studies for type 2 DM, hypertension or obesity have been undertaken in Australia for West African immigrants. It is hypothesized that the prevalence of metabolic risk factors and their sequelae in Ghanaians who have migrated to Australia have increased significantly to levels exceeding the Australian-born Caucasian population. This is due to a combination of dietary and lifestyle factors. Specifically, this study aimed to: (i) assess the prevalence of obesity, type 2 DM, hypertension and hyperlipidaemia in adult Ghanaians living in Sydney; and (ii) determine the relationships between dietary/lifestyle changes since migration from Ghana to Sydney and the development of risk factors to metabolic disease.

Methods

The study population was the adult Ghanaian migrant community of metropolitan Sydney. Recruitment of subjects was carried out by convenience sampling from member listings of the Ghanaian Association of New South Wales and local churches in the Sydney metropolitan area. Free living Ghanaian-born adults who are more than 25 years-old and residing in Sydney were recruited and interviewed in a Migrant Resource Centre, during March to August 2000. In total, 82 subjects were recruited (45 male, 37 female); however, two women were excluded from the study due to being pregnant at the time of recruitment. The study was approved by the University of Sydney Human Ethics Committee.

Correspondence address: Dr Samir Samman, Human Nutrition Unit, University of Sydney, NSW 2006, Australia.
Tel: +61 2 9351 2476; Fax: +61 2 9351 6022
Email: s.samman@biochem.usyd.edu.au
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Subjects reported to their local Migrant Resource Centre on two separate occasions after fasting for 10–12 h. The weight of each subject was measured to the nearest 0.1 kg by an electronic scale. Height was measured to the nearest 0.1 cm by a portable stadiometer. Body mass index (BMI) was calculated as mass (kg)/height^2 (m). Waist and hip circumferences were measured to the nearest 0.5 cm by a flexible, indistensible plastic tape measure. Physical activity was estimated by using The Concord Physical Activity Survey (Marketing Research Associates, North Sydney, Australia). This questionnaire was produced and validated by the Central Sydney Health Promotion Unit, Sydney.[12] Subjects were categorized into rural, semiurban and urban according to their town of residence in Ghana, and categorized into primary, secondary or tertiary according to their educational attainment. Awareness of metabolic disease and knowledge of relevant family history of metabolic disease was obtained from each subject, and if appropriate, subjects were asked if they were taking any medication to treat their condition.

Dietary intake data were collected from subjects by three 24-h food recalls for two weekdays and one weekend day. Details of macro- and micronutrients were obtained (DIET-1 database; Xyris Software, Brisbane, Australia) and adjusted for estimated energy intake (EEI). The EEI was calculated for each subject incorporating an activity factor based on reported physical activity.[13] The ratio of EEI to measured crude energy intake was then used to adjust all crude nutrient intakes. The EEI/basal metabolic rate (BMR) cut-off for underreporting for each subject was taken as 1.10 (95% confidence interval for 3 days dietary recall).[14] Subjects with an EEI/BMR ratio < 1.10 were to be excluded from the analyses; however, none of the subjects were in this category.[14]

Blood pressure measurements were taken by the same investigator, following a standardized protocol that was used in an international comparative study.[15] After sufficient time to rest, blood pressure was measured with a double-headed ('Y' terminal) stethoscope (Littman Classic II SE; 3M, St Paul, MN, USA) and dial sphygmomanometer (Tycos Silver-Ring Hand Aeroid; Welch-Allyn, Arden, CA, USA). Hypertension was defined as a systolic blood pressure ≥140 mmHg and/or a diastolic blood pressure ≥90 mmHg[15] or current use of antihypertensive agents.

Capillary blood samples were taken from each subject in the fasted state. Duplicate samples were obtained, one at each visit, for the determination of fasting whole blood glucose (FBG) concentrations (Precision Q.I.D. glucometer, MediSense; Abbott Laboratories, Columbus, Ohio, USA). A third FBG measurement was taken in the case of variance by more than 10% in the duplicate FBG values for each subject. Fasting whole blood glucose correlated positively with fasting plasma glucose values ($r = 0.93$, $P < 0.001$) and the coefficient of variation was < 2%. Using American Diabetes Association (ADA) and World Health Organization (WHO) criteria, type 2 DM was defined as a mean FBG ≥6.1 mmol/L and impaired fasting glucose (IFG) was defined as a mean FBG of 5.6–6.1 mmol/L.[16] Fasting plasma insulin (FPI) was measured by radioimmunoassay (Coat-A-Count Insulin solid-phase radioimmunoassay, Diagnostic Products Corporation, Box Hill, Victoria, Australia). Fasting hyperinsulinaemia was defined as a mean FPI ≥ 15 mIU/mL according to WHO criteria.[16] Matching pairs of duplicate FPI and FBG values were used to produce a homeostatic model assessment of insulin resistance (HOMA-IR) indices as a measure of insulin sensitivity for each subject.[17]

Capillary blood samples were also collected into ethylenediaminetetraacetate (EDTA) coated tubes. Duplicate samples were taken 3–4 days apart to minimize intraindividual variation in blood cholesterol levels.[18] Plasma samples were stored at –80°C, for the subsequent analysis of plasma total cholesterol, high-density lipoprotein-cholesterol (HDL-c), and triacylglycerol concentrations by an automated enzymatic method (Hitachi 912; Roche Diagnostics, Castle Hill, NSW, Australia). Low-density lipoprotein-cholesterol (LDL-c) was calculated by using the Friedewald equation modified for SI units.[19] Blood lipid levels were categorized according to the National Cholesterol Education Program criteria.[20]

All statistical analysis was carried out using SPSS-7.5 for Windows (SPSS, Chicago, IL, USA). Pearson's correlation coefficient (r) was computed to test correlations between selected pairs of measured variables, such as macro- and micronutrients, physical activity and other risk factors for metabolic syndromes. Student's t-test was used to compare differences between groups.

Results

Anthropometry

The anthropometric measurements of the subjects in this study are presented in Table 1. The BMI correlated positively with waist circumference ($r = 0.85$ and 0.92, $P < 0.001$), and with waist-to-hip ratio (WHR; $r = 0.58$ and 0.57, $P < 0.001$) for men and women, respectively. Women were more obese than men, especially in the abdomen (BMI ≥30; Table 1). No relationship existed between town of origin or education and BMI, waist circumference, or WHR. However, a significant positive correlation ($P < 0.05$) was observed between duration of migration and subsequent change in BMI, particularly among women ($P < 0.01$).

Plasma insulin and homeostatic model assessment of insulin resistance analysis

Men exhibited higher mean FPI and HOMA-IR index values than women (Table 2), although the differences were not significant due to large variation in FPI. When these indices were analysed by FBG status, increasing trends in both parameters were observed from IFG to normoglycaemic to type 2 DM diabetic subjects. Nevertheless, fasting hyperinsulinaemia (FPI >15 μIU/mL or 104 pmol/L) was not highly prevalent in this group, with 14.3% and 9.4% of men and women, respectively, sustaining this condition. Further analysis revealed a dissociation of FPI and HOMA-IR from lipids, blood pressure, exercise and dietary fibre. There was a positive trend of FPI and HOMA-IR with fasting triglycerides ($P < 0.05$ and $P < 0.01$, respectively).

Hypertension

Awareness of hypertension in this group was 29%. However, the prevalence rate was found to be 40.0% and 17.1% for men and women, respectively, with 17.1% of men and 5.7% of women sustaining definite hypertension. Men had significantly higher values than women in both systolic (133.6 ± 8.4 cf. 124.9 ± 14.8, $P < 0.05$) and diastolic blood pressure (86.7 ± 10.7 cf. 78.1 ± 9.9, $P < 0.005$). There were significant relationships observed between waist circumference and systolic blood pressure in women ($r = 0.56$, $P < 0.001$) and the total population ($r = 0.40$, $P < 0.001$), but not in men. Similarly, waist circumference predicted diastolic blood pressure in women ($r = 0.58$, $P < 0.001$) and the total population ($r = 0.48$, $P < 0.001$), but not in men.

Table 1. Subject characteristics and anthropometric measurements of the Ghanaian migrant population, with percentages within overweight and obese ranges

Characteristic	Men	Women
Age (years)	40.4 ± 6.4	34.8 ± 7.9
Height (m)	1.73 ± 0.05	1.61 ± 0.04
Weight (kg)	81.0 ± 9.9	70.9 ± 12.5
BMI (kg/m^2)	27.2 ± 2.8	27.4 ± 4.7
Healthy (18.5–24.9)†	28.9%	34.3%
Overweight (25.0–29.9)†	53.3%	40.0%
Obese (≥30.0)†	17.8%	25.7%
Waist (cm)	94.9 ± 8.4	86.3 ± 10.9
Healthy‡	46.7%	25.7%
Abdominal overweight§	33.3%	25.7%
Abdominal obesity¶	20.0%	48.6%
Hip (cm)	103.3 ± 5.8	106.8 ± 9.1
Waist-to-hip ratio	0.92 ± 0.05	0.81 ± 0.05
Skinfold measurements (mm)		
Tricep††	14.6 ± 3.6	26.0 ± 7.6
Subscapular††	24.1 ± 5.3	28.0 ± 10.5
Suprailiac††	19.5 ± 6.5	22.2 ± 8.4

Values are given as mean ± SD and percentages of total population; $n = 45$ for men, $n = 35$ for women. †Criteria set by World Health Organization.[21] ‡Waist circumference less than 94 cm for men, 80 cm for women.[22] §Waist circumference greater than 94 cm for men, 80 cm for women.[22] ¶Waist circumference greater than 102 cm for men, 88 cm for women.[22] ††Skinfold measurements of tricep, subscapular and suprailiac sites.

Dyslipidaemia

Significant differences were observed between men and women in total cholesterol, HDL-c, LDL-c and fasting triacylglycerol concentrations (Table 3). Lipid concentrations in men exceeded those of women in each case except for HDL-c which was significantly higher in women (Table 3). Furthermore, both LDL-c:HDL-c ratios and total cholesterol:HDL-c ratios were significantly higher in men than in women ($P < 0.001$). High-density lipoprotein-cholesterol decreased with increasing waist circumference in women ($r = 0.46$, $P < 0.005$) and in the total population ($r = 0.42$, $P < 0.001$). Fasting whole blood glucose was negatively correlated to HDL-c in women ($r = 0.46$, $P < 0.005$).

Prevalence of metabolic risk factors

Numerous subjects were found to express various combinations of metabolic syndrome components with 14.8, 23.0, 20.3, 12.2 and 1.4% of subjects sustaining 1, 2, 3, 4 and 5 risk factors, respectively. Approximately half (48.8%) were found to have both normal blood pressure and fasting blood

Table 3. Plasma lipid levels and percentage of individuals with low, borderline or high values

Plasma lipid levels (mmol/L)	Men	Women
Total cholesterol†	5.6 ± 0.8	4.7 ± 1.0*
Borderline to high (5.2–6.1)	51.1%	17.1%
High (≥6.2)	20.0%	11.4%
LDL-cholesterol†	3.8 ± 0.9	2.9 ± 0.9*
Borderline to high (3.4–4.0)	37.8%	8.6%
High (≥4.1)	28.9%	14.3%
HDL-cholesterol†	1.2 ± 0.3	1.4 ± 0.3**
Low (≤0.9)	17.8%	2.9%
Triglycerides‡	1.4 ± 0.7	0.8 ± 0.3*
High (≥1.7)	22.2%	2.9%
Borderline to high (2.3–4.4)	13.3%	0.0%
High (≥4.5)	0.0%	0.0%

Values are mean ± SD. $n = 45$ for men, $n = 35$ for women. P-value denotes level of significance in comparing difference between men and women. Values are significantly different *$P < 0.001$; **$P < 0.002$. Criteria for low, borderline and high are based on the †National Cholesterol Education Program[20] or ‡World Health Organization[16] guidelines. HDL, high-density lipoprotein; LDL, low-density lipoprotein.

Table 2. Fasting blood glucose, fasting plasma insulin and homeostatic model index of insulin resistance, classified by gender and fasting blood glucose categories, in Ghanaians living in Sydney, Australia

	n	FBG (mmol/L)	FPI (μIU/mL)	HOMA-IR index
Men	45	5.7 ± 1.3	10.43 ± 8.52	2.66 ± 2.49
Women	35	5.4 ± 0.6	8.24 ± 5.50	1.85 ± 1.31
Totals				
Normoglycaemic	50	5.1 ± 0.4	9.37 ± 7.56	2.10 ± 1.85
IFG	17	5.8 ± 0.3	8.40 ± 7.36	2.03 ± 1.86
Type II diabetic	13	7.0 ± 1.0	11.45 ± 7.13	3.49 ± 2.91

Values are given as mean ± SD. FBG, fasting blood glucose; FPI, fasting plasma insulin; HOMA-IR, homeostatic model index of insulin resistance; IFG, impaired fasting glucose.

glucose values. However, 7.5% of subjects had both hypertension and type 2 DM, therefore indicating a high proportion of the population with either elevated blood pressure or FBG values. The presence of these disorders was not related to self-reported family history of metabolic disease. A considerable number of subjects reporting no known family history of metabolic disease (36.4%) were found to have hypertension and/or elevated FBG values.

Dietary/lifestyle studies

The mean EEI over reported energy intake value was 1.29 ± 0.28, indicating a moderate degree of underreporting. No significant differences were observed between men and women in the adjusted macronutrient intakes (Table 4). However, men reported higher daily intake of beer ($P < 0.05$) and other alcohol ($P < 0.002$). The ratio of polyunsaturated : mononunsaturated : saturated fatty acids (P:M:S) for this group was 0.51:1.04:1.00 and 0.58:1.10:1.00 for men and women, respectively.

Salt intake was related to diastolic blood pressure in this group ($P < 0.02$); however, this relationship was lost when controlled for waist circumference and age. The dietary Na : K ratio was 1.00:1.23 and 1.00:1.52 for men and women, respectively. The intake of protein, fat, dietary fibre, sodium and potassium was not significantly related to the development of metabolic diseases in this group.

The Concord Physical Activity Survey measured both self-reported recreational and incidental exercise levels. Leisure exercise was minimal while incidental exercise was estimated at 18.9 ± 20.7 and 22.8 ± 24.3 min/day for men and women, respectively. Two subjects reported participating in regular sporting activities. Physical activity levels reported in this group were not related to the development of metabolic risk factors.

Discussion

The Ghanaians in this study showed an increased prevalence of metabolic disease with increasing Westernization, particularly in men.[6] Women exhibited more favourable biochemical profiles and had lower prevalence rates of diabetes and hypertension than men. When data from the present survey are compared with other populations, a clear dichotomy is seen between men and women and prevalence rates for obesity, type 2 DM and hypertension. The prevalence rates do not conform to the trend indicated in the figures for West African men and women with increasing Westernization (Figs 1,2). The prevalence of type 2 DM observed in Ghanaian men in the present study exceeded that of any other West African population, in contrast with a relatively low prevalence in women which was comparable to that of their urban West African counterparts.[1,23]

For the international comparison of the rates of diabetes, the present data were recalculated according to the older (1985) WHO criteria[22] where a FBG ≥6.7 mmol/L is diagnostic of type 2 DM rather than the 1998 ADA/WHO criteria.[16] This resulted in an apparent decrease in the high prevalence of type 2 DM in Ghanaian women but not men (Fig. 2). This suggests that type 2 DM is either more severe and/or poorly controlled in Ghanaian men compared with women. The major determinant of IFG is hypothesized to be defective insulin action rather than impaired β-cell function, thus explaining the higher HOMA-IR indices observed in IFG compared to those in normoglycaemia.[11] Progression to type 2 DM was marked by a considerable increase in HOMA-IR beyond that of IFG. The current study has revealed that the HOMA-IR did not differentiate between Ghanaians with normoglycaemia or IFG, although it tended to increase in Ghanaians with type 2 DM, mainly due to the raised FBG. The mean FPI for Ghanaians in the current

Table 4. Adjusted daily intakes and percentage contribution to total energy intake of macronutrients in Ghanaians living in Sydney, Australia

	Men (n = 40)	Women (n = 32)
Estimated energy intake† (MJ/day)	11.32 ± 0.70	9.03 ± 0.65
Protein (% energy)	18.1 ± 2.8	18.7 ± 3.2
Total fat (% energy)	32.9 ± 7.2	34.7 ± 5.6
Saturated fat (% energy)	11.1 ± 2.7	11.2 ± 3.4
Monounsaturated fat (% energy)	12.1 ± 2.9	12.9 ± 2.4
Polyunsaturated fat (% energy)	6.7 ± 3.3	7.5 ± 2.3
Carbohydrate (% energy)	46.6 ± 6.8	45.2 ± 6.9
Sugars (% energy)	10.7 ± 5.6	10.9 ± 4.8
Starch (% energy)	35.9 ± 5.9	34.2 ± 7.0
Alcohol (% energy)	1.0 ± 2.4	0.1 ± 0.5
Fibre (g/day)	30.1 ± 10.0	27.0 ± 8.6
Cholesterol (mg/day)	381.4 ± 125.1	326.6 ± 93.1
Sodium (mg/day)	3754 ± 1297	2644 ± 925
Potassium (mg/day)	3989 ± 1125	3488 ± 1141

†Ratio of estimated daily energy intake, % energy : % contribution of nutrient to total energy intake. Values are mean ± SD.

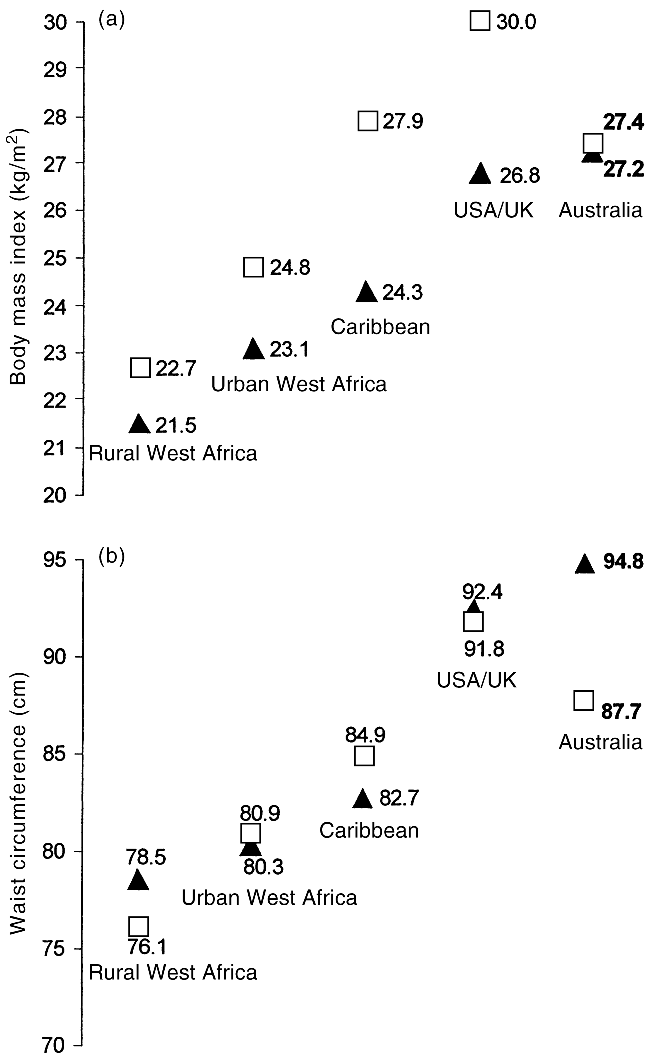

Figure 1. Summary of mean (a) body mass index (BMI) and (b) waist circumference values in West African populations, including data from the present study and from published data.[6,21] The trend of increasing overweight and obesity with increasing Westernization followed in the case of men but not women in the present study. ▲, Men; □, women.

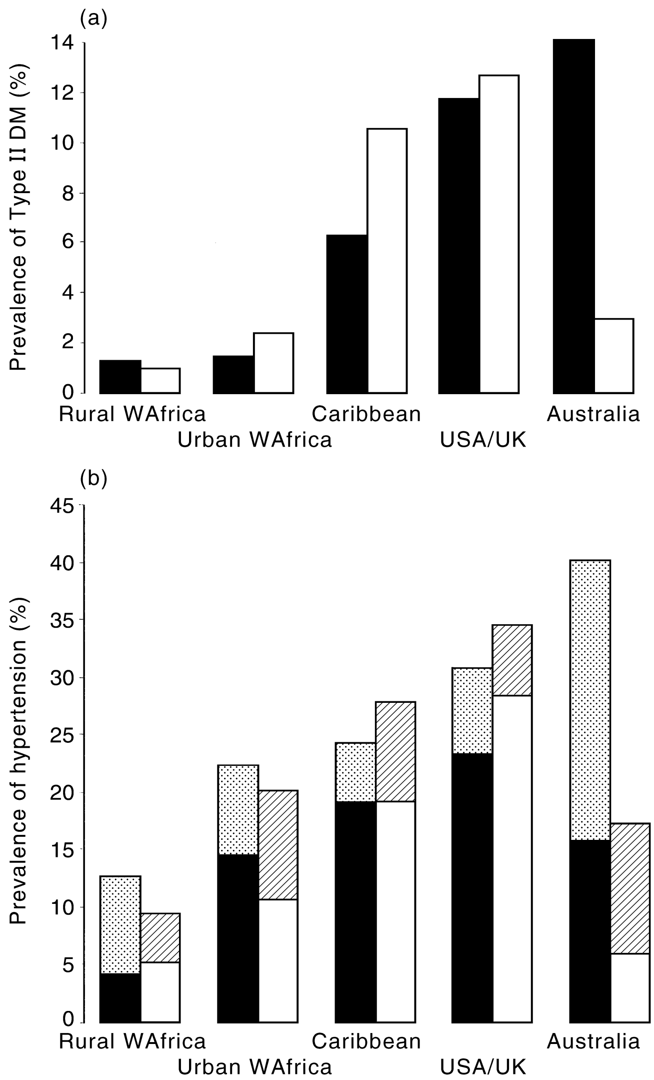

Figure 2. Prevalence rates of (a) type II diabetes mellitus (based on 1985 World Health Organization criteria),[24] and (b) hypertension (HT) in populations of West African origin, incorporating data from the present study and published data.[1,6,7,21] (░), Total HT in men; (▨), total HT in women; (■), definite HT in men; (□), definite HT in women.

study compared well to that of Ghanaians in the USA, thus illustrating the relative state of hyperinsulinaemia in this group compared to Caucasians.[2]

Previous studies have reported relationships between FPI, physical activity and dietary fibre but not lipids in West African populations.[4,25,26] Despite the correlation between FPI and blood pressure in Caucasians, the relationship was either weak or absent in African-Americans.[3,9] The absence of any significant associations between FPI and these variables in Ghanaians in the present study suggests a dissociation of plasma insulin from dietary and lifestyle factors. Dietary analysis revealed no significant correlations between nutrient intake and the development of metabolic risk factors. Similarly, it has been shown in Vietnamese refugees in Australia, that exercise, diet and alcohol are not related to BMI.[27] Furthermore, since reported physical activity for this group was low overall, physical activity levels would not be reflective of metabolic health. Thus, further studies of diet and physical activity and their association with metabolic risk factors are suggested for this population in order to decipher the significance of this relationship.

There were noteworthy differences in the prevalence rates of metabolic risk factors between genders. One in five men and one in nine women were found to be affected by type 2 DM. Similarly, significantly higher proportions of men than women experienced hypercholesterolaemia and hypertriglyceridaemia (Table 3), but not hyperinsulinaemia (Table 2). However, women in this study exhibited higher prevalence rates of abdominal overweight and obesity, and a higher mean tricep skinfold measurement than men (Table 1).

These results are contrary to results from numerous studies on Caucasian populations demonstrating a positive effect of abdominal overweight and obesity on health risk.[28,29] This raises the argument that some anthropometric relationships implied by the use of constant BMI standards are not strong or nonexistent in populations of West African origin, a phenomenon also discussed by Long *et al.*[30]

Several researchers have reported a lipid profile in African-Americans that is consistent with protection from chronic heart disease (i.e. higher concentrations of HDL-c and lower plasma triacylglycerol compared to Caucasians).[31,32] However, as cardiovascular disease is the leading cause of death in African-Americans, the protective effect of plasma lipids is possibly moderated by the higher incidence of hypertension.[33,34] As in the cases of type 2 DM and hypertension, women in this study exhibited more favourable lipid profiles than the men. Nevertheless, as self-reported physical activity was low, and overweight and obesity were epidemic in both genders, it remains unexplained why women do not have similar prevalence rates of type 2 DM, hypertension and dyslipidaemia to men. An important consideration is the effect that psychosocial stress has on the development of metabolic risk factors. Some studies have shown that stress is significantly related to hypertension and increases in total plasma cholesterol, LDL-c and triglycerides in African-Americans.[35,36]

The results of this study reveal that despite predominantly normal HDL-c and triacylglycerol concentrations, Ghanaians are not protected from developing hypertension or type 2 DM. The widespread phenomenon of isolated metabolic risk factor development calls for the delineation of an 'African metabolic syndrome'. Active screening of metabolic risk factors is encouraged for rapid detection and intervention at the individual level, and the implementation of an ethnic-specific intervention program to promote awareness and health education at the community level.

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References

1. Mbanya J-C, Cruickshank JK, Forrester T, Balkau B, Ngogang JY, Riste L, Forhan A, Anderson NM, Bennett F, Wilks R. Standardized comparison of glucose intolerance in West African-origin populations of rural and urban Cameroon, Jamaica, and Caribbean migrants to Britain. Diabetes Care 1999; 22: 434–440.
2. Osei K, Schuster DP, Owusu SK, Amoah AGB. Race and ethnicity determine serum insulin and C-peptide concentrations and hepatic insulin extraction and insulin clearance: Comparative studies of three populations of West African ancestry and white Americans. Metabolism 1997; 46: 53–58.
3. He J, Klag MJ, Caballero B, Appel LJ, Charleston J, Whelton PK. Plasma insulin levels and incidence of hypertension in African-Americans and whites. Arch Intern Med 1999; 159: 498–503.
4. Irwin ML, Mayer-Davis EJ, Addy CL, Pate RR, Durstine JL, Stolaczyk LK, Ainsworth BE. Moderate-intensity physical activity and fasting insulin levels in women: The cross-cultural activity participation study. Diabetes Care, 2000; 23: 449–454.
5. Osei K, Schuster DP. Effects of race and ethnicity on insulin sensitivity, blood pressure, and heart rate in three ethnic populations: Comparative studies in African-Americans, African immigrants (Ghanaians), and White Americans using ambulatory blood pressure monitoring. Am J Hypertens 1996; 9: 1157–1164.
6. Cooper RS, Rotimi CN, Kaufman JS, Owoaje EE, Fraser H, Forrester T, Wilks R, Riste L, Cruickshank JK. Prevalence of NIDDM among populations of the African Diaspora. Diabetes Care 1997; 20: 343–348.
7. Rotimi CN, Cooper RS, Okosun IS, Olatunbosun ST, Bella AF, Wilks R, Bennett F, Cruickshank JK, Forrester TE. Prevalence of diabetes and impaired glucose tolerance in Nigerians, Jamaicans and US blacks. Ethn Dis 1999; 9: 190–200.
8. Kumanyika S, Adams-Campbell LL. Obesity, diet, and psychosocial factors contributing to cardiovascular disease in blacks. In: Epidemiology and Related Issues. Philadelphia: F. A. Davis, 1990: 47–73.
9. Liese AD, Mayer-Davis EJ, Chambless LE, Folsom AR, Sharrett AR, Brancati F, Heiss G. Elevated fasting insulin predicts incident hypertension: the ARIC study. J Hypertens 1999; 17: 1169–1177.
10. Seidell JC. Obesity, insulin resistance and diabetes – a worldwide epidemic. Br J Nutr 2000; 83 (Suppl. 1): S5–S8.
11. Tripathy D, Carlsson M, Almgren P, Isomaa B, Taskinen M-R, Tuomi T, Groop LC. Insulin secretion and insulin sensitivity in relation to glucose tolerance: Lessons from the Botnia Study. Diabetes 2000; 49: 975–980.
12. Booth ML, Owen N, Bauman A, Gore CJ. Active and inactive Australians. Perspectives from the supplementary policy analysis of the pilot survey of the fitness of Australians. Canberra: Department of the Environment, Sport and Territories, 1995.
13. FAO/WHO/UNU. Report of a joint expert consultation energy and protein requirements, WHO Technical Report Series no. 724. Geneva: WHO, 1985.
14. Goldberg GR, Black AE, Jebb SA, Cole TJ, Murgatroyd PR, Coward WA, Prentice AM. Critical evaluation of energy intake data using fundamental principles of energy physiology: 1. Derivation of cut-off limits to identify under-recording. Eur J Clin Nutr 1991; 45: 569–581.
15. Ataman SL, Cooper R, Rotimi C, McGee D, Osotimehin B, Kadiri S, Kingue S, Muna W, Fraser H, Forrester T, Wilks R. Standardization of blood pressure measurement in an international comparative study. J Clin Epidemiol 1996; 49: 869–877.
16. Alberti KGMM, Zimmet PZ. Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: Diagnosis and Classification of Diabetes Mellitus Provisional Report of a WHO Consultation. Diabet Med 1998; 15: 539–553.
17. Matthews DR, Hasker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostatic model assessment: insulin resistance and β-cell function from plasma glucose and insulin concentration in man. Diabetologia 1985; 28: 412–419.
18. Samman S. Nutritional considerations in the variability of plasma cholesterol measurements. Eur J Clin Nutr 1991; 45: 463–468.
19. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clin Chem 1972; 18: 499–502.
20. The Expert Panel. Summary of the second report of the National Cholesterol Education Program (NCEP) Expert Panel on detection, evaluation, treatment of high blood cholesterol in adults (Adult Treatment Panel II). JAMA 1993; 269: 3015–3023.
21. Okosun IS, Cooper RS, Prewitt E, Rotimi CN. The relation of central adiposity to components of the insulin resistance syndrome in a biracial US population sample. Ethn Dis 1999; 9: 218–229.
22. World Health Organization. Physical status: the use and interpretation of anthropometry: Report of a WHO Expert Committee. WHO Technical Report Series. Geneva: WHO, 1995; 854: 1–452.

23. Owoaje EE, Rotimi CN, Kaufman JS, Tracy J, Cooper RS. Prevalence of adult diabetes in Ibadan. E Afr Med J 1997; 74: 299–302.
24. World Health Organization. Diabetes Mellitus: Report of a WHO Study Group. WHO Technical Report Series no. 727. Geneva: WHO, 1985.
25. Ludwig DS, Pereira MA, Kroenke CH, Hilner JE, van Horn L, Slattery ML, Jacobs DR. Dietary fiber, weight gain, and cardiovascular disease risk factors in young adults. JAMA 1999; 282: 1539–1546.
26. Zoratti R, Godsland IF, Chaturvedi N, Crook D, Stevenson JC, McKeigue PM. Relation of plasma lipids to insulin resistance, nonesterified fatty acid levels, and body fat in men from three ethnic groups. Relevance to variation in risk to diabetes and coronary disease. Metab Clin Exp 2000; 49: 245–252.
27. Bermingham M, Brock K, Nguyen D, Tran-Dinh H. Body mass index and body fat distribution in newly-arrived Vietnamese refugees in Sydney, Australia. Eur J Clin Nutr 1996; 50: 698–700.
28. Bjorntorp P. Abdominal fat distribution and disease: an overview of epidemiological data. Ann Med 1992; 24: 15–18.
29. Lind L, Lithell H. Hypertension, hyperlipidaemia, insulin resistance and obesity: parts of a metabolic syndrome. Blood Press 1992; 1 (Suppl. 4): 49–54.
30. Long AE, Prewitt TE, Kaufman JS, Rotimi CN, Cooper RS, McGee DL. Weight–height relationships among eight populations of West African origin: the case against constant BMI standards. Int J Obesity Relat Metab Disord 1998; 22: 842–846.
31. McDonough JR, Garrison GE, Hames CG. Blood pressure and hypertensive disease among Negroes and whites. Am J Intern Med 1964; 65: 271–315.
32. Linn S, Fulwood R, Rifkin B, Carroll M, Muesing R, Williams OD, Johnson C. High density lipoprotein cholesterol levels among US adults by selected demographic and socioeconomic variables. The Second National Health and Nutrition Examination Survey 1979–1980. Am J Epidemiol 1989; 129: 281–294.
33. Harris-Hooker S, Sanford GL. Lipids, lipoproteins and coronary heart disease in minority populations. Atherosclerosis 1994; 108 (Suppl.): S83–S104.
34. Lackland DT, Orchard TJ, Keil JE, Saunders DE Jr, Wheeler FC, Adams-Campbell LL, McDonald RH, Knapp RG. Are race differences in the prevalence of hypertension explained by body mass index and fat distribution? A survey in a biracial population. Int J Epidemiol 1992; 21: 236–245.
35. Barnes V, Schneider R, Alexander C, Staggers F. Stress, stress reduction, and hypertension in African Americans: An updated review. J Natl Med Assoc 1997; 89: 464–476.
36. Calderon R, Schneider RH, Alexander CN, Myers HF, Nidich SI, Haney C. Stress, stress reduction and hypercholesterolemia in African Americans: a review. Ethn Dis 1999; 9: 451–462.