

Interethnic physiological and pathological diversities in Southern African populations

ABSTRACT—The African continent accommodates a diversity of races—Arabs, Berbers, Nilo-Saharan, Bantu, Pygmies, as well as immigrant white and Indian populations, especially in Central, East and Southern Africa. In these populations and sub-populations, differences prevail in physiological variables, in biological disorders, in measurements made by laboratory and associated means, and in disease patterns. A salient question is: which differences will persist, and which become modified or even disappear as a result of progressive urbanisation, a rise in prosperity, and changes in lifestyle, particularly when these occur in populations previously poor or who have lived in a rural traditional manner.

Differences in the growth of Indian children

The growth of children in India differs from that of white children [1]. Among upper-class Indian children, growth is approximately 'normal' until around puberty; it then slows down and ceases at an age earlier than in white children [2,3]. This pattern is also seen in Indian immigrants in other parts of the world—Southern Africa, West Indies, and the UK. Thus, in the East End of London (UK), Indian boys aged 17 years are on average 7 cm shorter and 13 kg lighter than British boys [4].

To learn more of the bearing of genetic, socio-economic and other factors on the growth of Indian pupils, heights and weights were measured of 4,594 pupils who attended schools representative of upper, intermediate, and lower socio-economic classes [5]. Upper-class children were significantly bigger at all ages than children in the lower or poor classes, but differences tended to lessen by the age of 17 years. In the affluent group, under the 5th percentile of the National Center for Health Statistics (NCHS) reference values, height-for-age ranged from 4.7% to 22.0% in boys, and from 5.08% to 20.7% in girls; weight-for-age ranged from 20.3% to 45.0% in boys and from 9.3% to 37.7% in girls. At full growth at 17 years, mean height and weight were significantly less, roughly by 7 cm and

10 kg respectively, in the affluent group than in white children in NCHS reference data. The lower attainment of ultimate height in Indian children appears to be largely genetically determined and not due to simple dietary insufficiency.

Less adverse significance of obesity in black women

Severe obesity with a body-mass index (kg/m^2) (BMI) of 30 or over is fairly common in developed populations. They are more prone to hypertension, hyperlipidaemia, and hyperglycaemia, and at increased risk of hypertension-related diseases—diabetes, gallstones, coronary heart disease (CHD)—and certain types of cancer [6]. Obese people not characterised by adverse factors (hypertension, hyperlipidaemia, hyperglycaemia) are regarded by some as having 'healthy' obesity, in which weight-loss is not mandatory [7].

In South African black people, obesity is common among young and middle-aged women, particularly among urban dwellers. In Johannesburg, even in severely obese women, adverse stigmata were uncommon [8]. In a rural area, only five of 40 (12.5%) very obese black women (BMI ≥ 30) had significantly abnormal findings: hypertension ($\geq 160/95$ mm Hg) was present in three, hyperglycaemia (fasting blood glucose > 7.8 mmol/l) in one, hypercholesterolaemia (> 5.2 mmol/l) in three, and hyperglyceridaemia (fasting serum triglyceride > 5.2 mmol/l) in three [9]. Their diet was low in fat and high in carbohydrate, providing 21% and 65% respectively of the total energy intake. Accordingly, in 35 women (87.5%), obesity could be regarded as 'healthy'. This phenomenon is not an isolated occurrence. In a group of very obese poor women living on a low-fat diet in Santiago, Chile, obesity was not associated with hyperlipidaemia [10]. Even among the present-day black population in the US, obesity is less often associated with hypertension and diabetes than is the case with the white population [11]. Accordingly, obesity's noxiousness is regulated both by socio-economic state and by race.

Hypertension common in urban black adults

Some primitive or traditionally living rural populations experience little or no rise in blood pressure with age [12]. Even at present, elevated blood pressure is relatively uncommon in rural black adults. In two recent studies in Eastern Transvaal on persons aged 20–65 years, hypertension ($\geq 160/95$ mm Hg) was present in only 7% and 13% [9, 13].

With urbanisation, however, blood pressure rises

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markedly. In Durban, one study found 28% of black men with severe hypertension ($\geq 160/95$ mm Hg) [14], higher than the 20–25% in white men of the same age [11]. Early observers attributed the excessive rise in black people to maladaptation exacerbated by apartheid. However, in large city populations in other parts of Africa, eg in Dakar, Lagos, Benin City, Nairobi, where there is no apartheid, a rise in the frequency of hypertension is a recognised accompaniment of urbanisation [15], reaching levels as high as those noted in Durban and Johannesburg. In the US, hypertension is more common in the black than in the white population [11]. The explanation for their susceptibility is not clear. Local studies have shown that level of salt intake is not a serious contributing risk factor.

Sequelae of osteoporosis uncommon in black women

Over 99% of body calcium is contained in bone. In poor populations, especially developing populations, daily intake of the element is a half or less than that in the prosperous [16].

In South Africa, early studies on ribs and vertebral bodies in Indian, black, and white subjects indicated no significant differences in mean calcium concentration [16]. Bone-dimension studies on the second metacarpal were undertaken in series of black schoolchildren and elderly people, both in rural and in urban areas. The black children had slightly lower, but statistically significant, mean scores than white children, although in the elderly the differences were not significant [17]. Subsequently, studies were made on black and white women, subdivided into those having had (a) none to two, or (b) seven or more children [18]. In black or white mothers of large, compared with small, families, there were no significant differences in mean dimensions of bone (second metacarpal and humerus). Black mothers had significantly lower mean values for most variables compared with those of white mothers. Undoubtedly, the smaller dietary calcium intake by black mothers is offset by its greater absorption [19]. In this context the rare occurrence of hip fracture and of collapsed vertebral bodies in black women is remarkable. In Johannesburg a decade ago, the frequency of hip fracture in black women was only one-tenth of that in white women [20]. A recent unpublished study by ourselves revealed an equally wide differential. In the US, hip fractures are less common in black than in white women [21]. The explanation for the more favourable situation in black compared with white women is not immediately obvious.

Abnormal iron retention or siderosis in black people

In 1929, postmortem studies on black adults frequently revealed abnormally high iron deposits in the liver and other organs and tissues [22]. This was at first ascribed to malnutrition or to pellagra. But because

iron balance studies on white subjects had shown that abnormal deposition can result from high iron intake [23], a number of everyday foodstuffs eaten by black people in rural and urban areas were analysed. A high iron intake, reaching 50–100 mg daily, was found to be quite common and was the probable cause of the iron overload [24]. The iron came from food prepared in iron utensils, including the ubiquitous iron tripod 'kaffir pot', paraffin cans, and other vessels. Adventitious uptake was especially high in foodstuffs of low pH value, such as fermented porridge and 'kaffir beer'. With habitually high intakes of such foods, concentrations of iron can reach 5% in the liver and 10% in the spleen [24]. Excessively high deposits, although present in only a small minority of people, promote liver fibrosis and cirrhosis, diabetes, osteoporotic changes, and also prejudice vitamin C metabolism, although clinical scurvy was not reported [25]. Iron deficiency anaemia was relatively rare but other haematological indices were often raised. The traditional iron pots are nowadays less often used for food preparation, and consequently high levels of serum iron and abnormal iron deposition are less common than in 1950–1960. Clearly, in this case the responsible factor was wholly environmental.

Abnormalities in electrocardiograms of young black men and women

In the 1960s, electrocardiogram tracings of black people living in America [26], and of South, East, and West African blacks, revealed a number of abnormalities. They mimicked pericarditis, myocardial infarction, left ventricular hypertrophy, and acute cor pulmonale. Differences in tracings between black and white subjects included, in the former, persistence of the 'juvenile' pattern, greater prevalence of ST segment elevation with or without tall T-waves, and high QRS voltage. Abnormalities were variously attributed to race, to malnutrition, or simply to being 'normal variants' [27].

In South Africa several workers, especially Grusin [28] in 1954, drew particular attention to the frequent finding in black subjects of inverted T-waves with ST depression, and of tall T-waves with or without ST depression. Fifteen years later we confirmed these findings in our studies [29], but were unable to explain the difference on the basis of variations in anthropometric measurements, ponderal index, nutritional state, infections, activity, or from variations in cultural levels. These ECG differences in the black subjects had no prognostic significance. Recent reports have indicated that the differences in ECG tracings are becoming less common.

Quicker bowel transit time in black children

The great physician-philosophers of old—Hippocrates, Galen, Moses Maimonides, and others—were well

aware that the composition of the diet, especially its content and nature of plant foods, regulates the passage of digested food through the body.

In South Africa, black schoolchildren and adults living in rural and urban areas had shorter food transit times, defaecated more often, and daily passed larger quantities of faeces than did white subjects [30]. In black people this pattern of bowel behaviour was associated [31] with a far lower incidence of chronic bowel diseases and colorectal cancer. It was originally attributed to their higher intake of fibre-containing foods and their lower intake of fat [32]. This was perhaps one of the earliest propositions that particular patterns of diet are associated with particular patterns of diseases.

Recent studies on bowel behaviour in preschool children showed that, despite the smaller fibre intake associated with urbanisation, the urban black group had a median food transit time (first appearance of marker) of 7 hours, far shorter than the 27 hours of a comparable white group [33]. In a rural group and a better-class urban group of black adolescents, and a comparable white group, respective median times for men were 18, 22, and 28.5 hours, and for women 23, 28, and 34.5 hours [34]. Median times in these groups of black people are somewhat longer than in earlier data. While median times of subjects in upper and lower thirds of fibre intake differed as expected, the difference was less than expected if fibre intake had been the primary determinant of transit time. The wide ranges in transit time in all groups were conspicuous. Interestingly, white schoolchildren in a Salvation Army home, given a supplement of bran 'crunchies' to raise their fibre intake to that of rural black children, still had longer transit times.

The shorter transit time in black people is the result of a different *milieu interieur* in the bowel which includes:

- i a greater excretion of 'resistant' starch into the colon;
- ii higher production of breath methane [35];
- iii greater surface area of the colon [36];
- iv lower frequency of polyps [37];
- v lower faecal pH values [38];
- vi a higher level of everyday physical activity, a factor known to affect bowel motility and behaviour.

Interethnic differences in breath methane excretion

Methane is produced in the large intestine and excreted in the breath. Methanogenesis in relation to health and disease has been little studied, although one study noted high excretion in colon cancer patients [39]. In South Africa, proportions of methane producers were: rural black 84%; urban black 72%; white 52%; and Indian 41%. Bowel cancer risk was least in populations with the highest proportion of methane producers. Accordingly, methanogenesis appears not to be a risk

factor of bowel cancer but an indication of healthy colonic function [35]. A recent study on a small unpublished series of local Chinese students revealed frequency of methane production to be even lower than that in Indians. This diversity of breath methane excretion is not currently explicable.

Hyperinsulinaemia in young Indians after glucose dose

It is well known that Indians, especially when they are prosperous emigrants, are excessively prone to diabetes. Although mean fasting blood glucose levels of Indian students are the same as those of whites one hour after a 50 g glucose dose, their serum insulin levels are significantly higher than those of the white students [40]. In our investigations on Indian school pupils, mean serum insulin values at fasting and one hour after a 50 g glucose dose were 16 and 67 $\mu\text{u/ml}$, and on local white pupils 15 and 42 $\mu\text{u/ml}$ [41]; blood glucose values were 3.5 and 4.1 mmol/l and 4.1 and 4.9 mmol/l. This phenomenon of hyperinsulinaemia and excessive diabetes is presumably of genetic origin, and becomes increasingly manifest with rise in prosperity.

Interethnic differences in coronary heart disease

In South Africa, the occurrence of coronary heart disease (CHD) is highest in the Indian population, less high in the white, and lowest in the black population [42-44]. In the white population mortality rates are high, being higher for Afrikaans than for English-speaking people [45]. The rate for Jews is particularly high.

The mortality rate for Indians is higher than that for whites, and is increasing. In a recent study in Bombay, CHD was found to be common even in fully vegetarian, low-fat eating, non-smoking, non-hypertensive, non-obese labourers. Among Indian migrants in London, the incidence of CHD in middle-aged men was 50% higher than in white men [46]. In Birmingham, Asian patients matched with white patients for age, sex, blood pressure, and duration of symptoms had more severe coronary artery disease; yet Asian patients smoked less, were less obese, and had lower mean serum cholesterol levels [47].

Coronary heart disease remains uncommon in urban blacks and is nearly absent in rural dwellers [42]. In Soweto, at Baragwanath Hospital, only 70 or so patients with CHD are admitted annually from a population of 1.5-2 million. This low rate is unexpected since a rising proportion of prosperous black people has all the risk factors for CHD. How prevalent is it elsewhere in Africa? In Nigeria, CHD is virtually unknown. At Enugu, over a 4-year period, not one patient out of 348 with cardiac disease had CHD [48]. Investigations in the Ivory Coast, Zaire, Zambia, Botswana, and other African countries have indicated the disease to be extremely rare, if not entirely absent.

An editorial on 'British and African hearts' underlined the tremendous contrast between the experience of CHD in the two types of population [49]. In 1973, in the East End of London, immigrant black people from the Caribbean had 'one-tenth of the average attack rate' [46]. In Birmingham, England, the disparity between frequency of CHD in blacks and whites was even wider [47].

Interethnic diversities in the occurrence of cancer

Cancer occurrence data are derived from incidence rates, from records of admissions to hospitals, and death rates. The South African Cancer Registry for 1986 [50] revealed that in the white population most rates, standardised to 'world' population, are slightly lower than those of many Western populations. However, whites have exceptionally high rates for basal cell and squamous cell cancers of the skin.

Total rates for black people are less than half of those for whites. Rates are excessively high for cervical and oesophageal cancers, yet low for stomach and colorectal cancers and, to a lesser extent, for breast cancer. Very low rates prevail for skin cancer.

In the Indian population overall rates, especially for men, are lower than those for white people. Compared with data on Indians in Bombay [51], men in South Africa have higher rates for stomach, prostate, and bladder cancers, but lower rates for cancers of the oesophagus and pancreas. Indian women in South Africa have higher rates for oesophageal and cervical cancers. The extremely low occurrence of skin cancer is conspicuous, as it is with African populations [52]; protection is presumably due to skin pigmentation.

Discussion and conclusions

The presentations given illustrate some of the physiological and pathological differences existing between black, Indian, and white people in South Africa. Which differences will persist and which will become modified, or even disappear, with a rise in urbanisation and prosperity?

First, in all populations and in any community, well-recognised differences will persist in the behaviour and responses between individuals. In the case of the black population, three characteristics are likely to be sustained:

- i their greater frequency of hypertension;
- ii their lesser susceptibility to severe osteoporosis and to hip fractures;
- iii their far smaller proneness to skin cancer.

In the Indian population, differences likely to persist are:

- i children's lower attainment in height;
- ii the excessive susceptibility of children and adults to hyperinsulinaemia, and in the case of adults to diabetes and coronary heart disease;
- iii their lesser susceptibility to skin cancer.

In the case of the white population, because of the environmental factor, the exceptionally high incidence of skin cancer is likely to remain.

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References

- 1 Hamill PVV, Drizd TA, Johnson CL, *et al.* Physical growth; National Center for Health Statistics percentiles. *Am J Clin Nutr* 1979; **32**:607-29.
- 2 Gopaldas T, Cappor I. A nutritional status study on privileged adolescent Gujarati boys. *Indian J Med Res* 1981; **73**:188-98.
- 3 Pereira P, Mehta S, Khare BB, *et al.* Physical growth characteristics in adolescent girls of upper socio-economic group in Varanasi. *Indian J Med Res* 1983; **77**:839-44.
- 4 Ulikaszek S, Evans E, Mumford P. Anthropometric survey. *Lancet* 1979; **i**:214.
- 5 Walker ARP, Walker BF, Jones J, Kadwa M. Growth of South African Indian schoolchildren in different social classes. *J R Soc Health* 1989; **109**:54-6.
- 6 Van Itallie TB. Health implications of overweight and obesity in the United States. *Ann Intern Med* 1985; **103**:983-8.
- 7 Knaap TR. A methodological critique of the 'ideal weight' concept. *J Am Med Assoc* 1983; **250**:506-10.
- 8 Joffe BI, Goldberg RB, Feinstein J, *et al.* Adipose cell size in obese Africans: evidence against the existence of insulin resistance in some patients. *J Clin Pathol* 1979; **32**:471-4.
- 9 Walker ARP, Walker BF, Walker AJ, Vorster HH. Low frequency of adverse sequelae of obesity in South African rural black women. *Int J Vitam Nutr Res* 1989; **59**:224-8.
- 10 Albala C, Olivares S, Andrade M, *et al.* Impact of the socioeconomic level on serum lipids in obese women. *Fifth International Congress on Obesity*. Jerusalem: 1986;65.
- 11 Cornoni-Huntley J, LaCroix AZ, Havlik RJ. Race and sex differentials in the impact of hypertension in the United States. *Arch Intern Med* 1989; **149**:780-8.
- 12 Walker ARP. Overweight and hypertension in emerging populations. *Am Heart J* 1964; **68**:581-5.
- 13 de Villiers EC, Albertse EC, McLachlan MH. The prevalence of obesity and hypertension among Zulu women in a remote area. *S Afr J Sci* 1988; **84**:601-2.
- 14 Seedat YK, Seedat MA, Hackland DBT. Biosocial factors and hypertension in urban and rural Zululand. *S Afr Med J* 1982; **61**:999-1002.
- 15 Azinge NO. The changing pattern of hypertension in Benin City, Nigeria. *Trop Doct* 1980; **10**:154-6.
- 16 Walker ARP, Arvidsson UB. Studies on human bone from South African Bantu subjects. Part I. Chemical composition of ribs from subjects habituated to a diet low in calcium. *Metabolism* 1954; **3**:385-91.
- 17 Walker ARP. Cortical thickness of bone in under privileged populations. *Am J Clin Nutr* 1970; **23**:244-5.
- 18 Walker ARP, Richardson BD, Walker BF. The influence of numerous pregnancies and lactations on bone dimensions in South African Bantu and Caucasian mothers. *Clin Sci* 1972; **42**:189-96.
- 19 Walker ARP, Fox FW, Irving JT. Studies in human mineral metabolism. The effect of bread rich in phytate phosphorus on the metabolism of certain mineral salts with special reference to calcium. *Biochem J* 1948; **42**:452-62.
- 20 Solomon L. Osteoporosis and fracture of the femoral neck in the South African Bantu. *J Bone Joint Surg* 1969; **50B**:2-13.
- 21 Pollitzer WS, Anderson JJB. Ethnic and genetic differences in bone mass: a review with a hereditary vs environmental perspective. *Am J Clin Nutr* 1989; **50**:1244-59.

- 22 Strachan AS. Haemosiderosis and haemochromatosis in South African natives, with a comment on the aetiology of haemochromatosis. Glasgow: MD thesis, 1929.
- 23 Brock JF, Hunter D. Fate of large doses of iron administered by mouth. *Q J Med* 1937;6:5-16.
- 24 Walker ARP, Arvidsson UB. Iron 'overload' in the South African Bantu. *Trans R Soc Trop Med Hyg* 1953;47:536-48.
- 25 Bothwell TH, Charlton RW. Historical overview of hemochromatosis. *Ann NY Acad Sci* 1988;526:1-10.
- 26 Thomas J, Harris E, Lasiter G. Observations on the T-wave and S-T segment changes in the precordial electrocardiograms of 320 young Negro adults. *Am J Cardiol* 1960;5:468-72.
- 27 Blackman NS, Kuskin L. Inverted T-waves in the precordial electrocardiogram of normal adolescents. *Am Heart J* 1964;67:304-12.
- 28 Grusin H. Peculiarities of the African's electrocardiogram and the changes observed in serial studies. *Circulation* 1954; 9: 860-7.
- 29 Walker ARP, Walker BF. The bearing of race, age, sex and nutritional state on the precordial electrocardiogram of young South African Bantu and Caucasian subjects. *Am Heart J* 1969;77:441-59.
- 30 Burkett DP, Walker ARP, Painter NS. Effect of dietary fibre on stools and transit times and its role in the causation of disease. *Lancet* 1972;ii:1408-12.
- 31 Segal I, Walker ARP. Low-fat intake with falling fibre intake commensurate with rarity of non-infective bowel diseases in Blacks in Soweto, Johannesburg, South Africa. *Nutr Cancer* 1986;8:185-91.
- 32 Walker ARP. Diet and atherosclerosis. *Lancet* 1985;ii:565-6.
- 33 Walker ARP, Walker BF. Bowel behaviour in young black and white children. *Arch Dis Child* 1985;60:967-70.
- 34 Walker ARP, Walker BF, Lelake A, et al. Transit time and fibre intake in black and white adolescents in South Africa. *Digestion* (in press).
- 35 Segal I, Walker ARP, Lord S, Cummings JH. Breath methane and large-bowel cancer in contrasting populations. *Gut* 1988;29:608-13.
- 36 Melissas J, Segal I, Leries M, Moar JJ. The anatomical length of the colon in black adults: differences between cadaver specimens and living subjects. *S Afr Med J* 1985;68:523.
- 37 Segal I, Cooke SAR, Hamilton DB, Ou Tim L. Polyps and colorectal cancer in South African blacks. *Gut* 1981;22:653-7.
- 38 Walker ARP, Walker BF, Walker AJ. Faecal pH, dietary fibre intake, and proneness to colon cancer in four South African populations. *Br J Cancer* 1987;53:489-95.
- 39 Pique JM, Pallares M, Cuso E, Vilar-bonet J, Gassull MA. Methane production and colon cancer. *Gastroenterology* 1984;87:601-5.
- 40 Keller P, Schatz L, Jackson WPU. Immunoreactive insulin in various South African population groups. *S Afr Med J* 1972;46:152-7.
- 41 Walker ARP, Bernstein RE, du Plessis I. Hyperinsulinaemia from glucose dose in South African Indian children. *S Afr Med J* 1972;47:1916.
- 42 Walker ARP, Walker BF. Coronary heart disease in blacks in underdeveloped populations. *Am Heart J* 1985; 109: 1410.
- 43 Sewdarsen M, Vythilingum S, Jialal I, Moodley T, Mitha AS. Risk factors in young Indian males with myocardial infarction. *S Afr Med J* 1987;71:261-2.
- 44 Rossouw JE, Weich HFH, Steyn K, Kotze JP, Kotze TJW. The prevalence of ischaemic heart disease in three rural South African communities. *J Chron Dis* 1984;37:97-106.
- 45 Walker ARP. Epidemiology of ischaemic heart disease in different populations in Johannesburg. *S Afr Med J* 1980;57:748-52.
- 46 Pedoe HT, Clayton D, Morris JN, et al. Coronary heart attacks in East London. *Lancet* 1975;ii:833-8.
- 47 Lowry PJ, Glover DRG, Mace PJE, Littler WA. The pattern and severity of coronary artery disease in Asians and whites living in Birmingham. *Postgrad Med J* 1983;59:634-5.
- 48 Uzodike VO, Anidi AI, Ekpechi LVO. The pattern of heart disease in Enugu, Nigeria. *Nigerian Med J* 1977;7:315-9.
- 49 Leading article. British and African hearts. *Lancet* 1983;i:1256-7.
- 50 *South African cancer registry 1986*. Johannesburg: South African Institute for Medical Research, 1988.
- 51 Jussawalla DJ, Yeole BB, Netekar MV. Cancer in Indian Moslems. *Cancer* 1985;55:1149-58.
- 52 Parkin DM. *Cancer occurrence in developing countries*. Lyon: International Agency for Research on Cancer, IARC Scientific Publications, No. 75, 1986.

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