Occasional Reviews

Male procreative superiority index (MPSI): the missing coefficient in African anthropogenetics

FELIX I D KONOTEY-AHULU

Summary and conclusions

The adult man in Africa, unlike the average European man, can have a biological fitness exceeding that of his wife. Sociocultural factors allow, and indeed encourage, this state of affairs, which may have far-reaching genetic consequences. The male procreative superiority index (MPSI) of any man is easily worked out by dividing the total number of a man's children by the average number of children born to each wife. The country-wide mean MPSI for 3095 fathers contacted throughout Ghana was 2.03, indicating that the Ghanaian father on the average has twice as many children as the mother. The genetic consequences of this phenomenon are discussed, bringing out effects on such diverse genes as those for abnormal haemoglobins, twins, and extra digits. African anthropogenetics needs rethinking more on factual lines than on theoretical evolutionary concepts.

Introduction

A statement like, "The male in Africa has more children than the female" sounds like a genetic heresy, but this is exactly what happens in most of tropical Africa. Go to any African village, collect 100 children, identify their parents, and you will find that they have about 20 mothers but only 10 fathers between them. Repeating the experiment in Scotland would give a different result—the 100 children would most probably point to 40 fathers and 40 mothers. The first obvious consequence of this phenomenon is that the African children have more genes in common than the Scottish children. The genetic "likeness" of the African children (hence of the African population) considerably exceeds that of the Scottish children (and population) in a way that cannot be satisfactorily explained by their different in-breeding coefficients alone. If biological fitness is defined as "the number of genes one contributes to the offspring generation"2 then the adult man in Africa, unlike the man in Scotland, has a greater fitness than the wife. This male procreative superiority in Africa has been quantified in the Ghanaian population by using a convenient index—the male procreative superiority index (MPSI). One would expect that in a population with high values of MPSI the marker genes would proliferate in a way unusual in European populations. It is postulated for debate that the high rates of genetic markers in African populations (using, for example, extra digits, twinning, and abnormal haemoglobins in West Africa) cannot be dissociated from high values of MPSI.

Ghana Institute of Clinical Genetics, Korle Bu Teaching Hospital, Accra, Ghana

FELIX I D KONOTEY-AHULU, MD, FRCP, honorary Volta Aluminium Company visiting research physician consultant

How MPSI is worked out

- 1 Total number of man's children: N_m
- 2 Number of women (wives) man had children by: W
- 3 Total number of children born to these women including, possibly, other men's children: N_w
 - 4 Average number of children born to each wife: $\frac{N_w}{W}$

5 MPSI =
$$\frac{\text{Total number of man's children}}{\text{Average number of children born to each wife}} = \frac{\frac{N_m}{N_w}}{W}$$

$$= \frac{N_m \times W}{W}$$

Thus MPSI=total man's children multiplied by number of wives divided by total wives' children.

EXAMPLES

1 A Ghanaian man has 94 children by 23 wives, four of whom had a total of six children before marriage to him.

Man's children $N_m = 94$

Number of women the man had children by, W = 23

Total number of children born to wives, $N_w = 94 + 6 = 100$

MPSI of man:
$$\frac{N_m \times W}{N_w} = \frac{94 \times 23}{100} = 21.62$$

2 A thrice-divorced American film star has three children by his fourth wife giving him a total of eight children. Two of his previous wives have since married again and had two more children.

MPSI of this man:
$$\frac{N_m \times W}{N_w} = \frac{8 \times 4}{10} = 3.2$$

3 Where a man has only one wife and has had all his children by her and she has had no children by any other man as in most mono- $N_m \times W$

gamous set-ups,
$$N_m = N_w$$
, and $W = 1$, so that $MPSI = \frac{N_m \times W}{N_w} = 1$

and the gene contributions from each parent to the offspring are equal.

Using this formula to quantify differential procreation in the nine regions of Ghana¹ through 5708 successful interviews of fathers and mothers the results show that the average Ghanaian father has twice as many children as the mother (table II).

Only Greater Accra and Central and Western Regions have MPSI values much below the national average. These are also the more advanced regions where procreation is less likely to be on traditional African lines. Even so, the father in sophisticated Accra has one-and-a-half times more children than the mother.

Prevalence of some common genetic markers in Africa

EXTRA DIGITS

This Mendelian dominant birth defect is quite common in Africa but the incidence at birth had never been determined until Bonney and colleagues, at the Second International Congress on Twin Studies in Washington, DC, 29 August-1 September 1977, reported it in "1% of single births and 2% of twin births"; but observing the defect in only one of each twin pair they computed that the "incidence

among twins does not differ significantly from that among single births." The actual number of babies with extra digits in $13\,231$ consecutive live births at the Korle Bu Teaching Hospital in Ghana³ between 1 January and 31 December 1975 was 123, an at-birth incidence of 0.93%. Thus 1% of children born in the Greater Accra Region of Ghana would be expected to have extra digits. This is a high frequency for any genetic marker in any population.

TABLE I—Chances that successive wives carry haemoglobin trait

| Number | Trait chance |
|-----------------------------|-------------------------------|
| st wife of trait man | 1 in 3 of being AS or AC |
| st two wives of trait man | 1 in 9 of both being AS or AC |
| st three wives of trait man | 1 in 27 of all being AS or AC |
| st four wives of trait man | 1 in 81 of all being AS or AC |

more wives is eliminated in his case. Indeed, there is a high (1 in 27) chance that the first three wives of a husband with sickle-cell trait will each carry an abnormal haemoglobin trait (table I).

One way to prove this hypothesis is to show that generally speaking more fathers with abnormal haemoglobins will tend to have greater MPSI values than homozygous AA fathers in populations where religious and other traditions allow many wives. The highest haemoglobin C trait incidence in the world⁵ 6 12 is in the Upper Region of Ghana, which also has the highest average MPSI values in the country. Already, Roberts and Boyo¹³ have found that marriages between partners with sickle-cell trait produced more children than those with nontrait fathers; and from Lebanon Dabbous and Firzli¹⁴ have found a predominance of the sickle-cell trait among Mohammedans. The Christians, they said, inhabited the mountainous

TABLE II-Average MPSI per region in Ghana

| | Upper | Northern | Brong Ahafo | Ashanti | Western | Central | Eastern | Volta | Greater Accra | Whole country |
|------|-------|----------|----------------|---------|---------|---------|---------|-------|------------------|---------------|
| MPSI | 2.82 | 2.01 | 2.53 | 2.21 | 1.90 | 1.72 | 2.60 | 2.07 | 1.53 | 2.03 |

TWINS

There is a higher incidence of multiple births in the Ghanaian population than has been reported elsewhere (G E Bonney, Washington 1977). Twin births occurred in 3·21% (and triplets in 0·05%) of 12 814 consecutive deliveries. Thus while one in every 30 consecutive deliveries in Ghana produces twins some figures given for other countries are one twin for every 80 single births in Britain, one for every 86 in the United States, and one in 145 for Japan.⁴

ABNORMAL HAEMOGLOBINS

The incidence of abnormal haemoglobin traits in West Africa is not less than 33% of the healthy population. ⁵⁻⁷ In a Ugandan population 45% of those examined had the sickle-cell trait. The usual explanation for such high trait incidence is through a balanced polymorphic phenomenon whereby the traits in Africa enjoyed a preferential survival advantage over both the abnormal homozygote (which was eliminated early in life anyway) and the normal homozygote which was generally decimated by falciparum malaria before passing many normal genes on to the offspring generation. The trait, it is argued, had a greater biological fitness over the centuries than either homozygote, and hence the present extraordinarily high rates when the very converse should have been the case for a deleterious gene.

Discussion

The following question needs asking: "Is the polymorphism hypothesis alone adequate to explain such high trait frequencies (30-45%) in some regions of Africa?" I think Roberts and Lehmann¹⁰ were right when they said that apart from polymorphic considerations gene frequency in any population sample will also depend "on a combination of social and ecological factors and the mating system of the people." Western geneticists must attempt to quantify accurately each of the factors that go to influence gene frequencies in real life in the rest of the world. Bonney and Konotey-Ahulu¹¹ produced a mathematical model clarifying the role of polygamy in the maintenance of genetic equilibrium. They showed that because the West African husband with sickle-cell trait (who has a one in three chance of marrying another trait, AC or AS) would tend to marry more wives in an attempt to find one whose children do not have chwechweechwe (sickle-cell disease), he has a greater fitness than the non-trait AA husband. As the latter could never have a child with sickle-cell disease regardless of whom he married one important reason why Africans acquire

non-malarious region of Lebanon and therefore had a lower sickling rate; the Moslems inhabited low-lying malarious plains and valleys and thus propogated the S gene. But could not the difference be partly due to the different procreative habits of the two religious groups?¹⁵ If Dabbous and Firzli could brave the present fighting in Lebanon and go back to measure the MPSI of the Christians and Moslems might they not conclude differently? True, Konotey-Ahulu¹⁶ described a woman with sickle trait who had 12 deliveries, half of them twin deliveries making 18 children in all, but this is about the limit a woman with trait can go in procreation to disseminate nine sickle-cell genes. Our fetish-priest farmer with 94 children with a one in three chance of himself having a sickle-cell trait or haemoglobin C trait would donate 47 abnormal genes. He had a 1% chance of being an adult SC phenotype in Ghana—he would then have passed on 94 abnormal haemoglobin genes to posterity. In that population eight of the wives would either have sickle-cell trait or haemoglobin C trait, with a one in four chance of their children having the disease (or one in two chance if the man had haemoglobin SC disease). The oldest haemoglobinopathy patient in Accra until recently was an 87-year-old homozygous haemoglobin C patient—he had 24 children, each with his haemoglobin gene.

What has been discussed above is no mere theoretical exercise. It is an everyday African reality that has been sadly ignored in international schemes of population control. The man in Africa has been completely ignored while he quietly moves out of his own generation into the generation of his children and grand-children to acquire female vehicles through whom he disseminates his genes. As has been pointed out before, if in countries where polygamy is legalised "if sickle cell trait men could be selectively persuaded to abstain from polygamy, the sickling rate in the population would be gradually reduced," and the man in a developing country who refuses to take a younger wife when his first wife arrives at the menopause is doing the country a great deal of service "not only by reducing the population explosion, but, if he is a sickler, by deliberately limiting his contribution of genes to the community." 17

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Requests for reprints should be addressed to: F I D Konotey-

Ahulu, Post Graduate Medical Centre, Royal Northern Hospital, Holloway Road, London N7 6LD.

References

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- ¹ Konotey-Ahulu FID. Male procreative superiority in African populations: the fact established and quantified. In: Szabo G, Papp Z, eds. Medical genetics. Amsterdam-Oxford: Excerpta Medica, 1977:599-607.
- Li CC. Genetic equilibrium under selection. Biometrics 1967;23:397-494.
- ³ Konotey-Ahulu FID. Ghana Institute of Clinical Genetics—Report on May 1974-May 1979. Accra: Managing trustees of the VALCO Fund, 1979:10.
- ⁴ Morison JE. Foetal and neonatal pathology. London: Butterworths, 1970.
- ⁵ Edington GM, Lehmann H. A case of sickle-cell haemoglobin C disease and survey of haemoglobin C incidence in West Africa. Trans R Soc Trop Med Hyg 1954;48:332-6.
- ⁶ Konotey-Ahulu FID, Ringelhann B. Sickle-cell anaemia, sickle-cell thalassaemia, sickle-cell haemoglobin C disease, and asymptomatic haemoglobin C thalassaemia in one Ghanaian family. Br Med J 1969;i:
- ⁷ Konotey-Ahulu FID. The sickle-cell diseases; clinical manifestations including the sickle crisis. Arch Intern Med 1974;133:611-9.

- 8 Lehmann H, Raper AB. Distribution of the sickle-cell trait in Uganda and its ethnological significance. Nature 1949;164:494-5.
- ⁹ Allison AC. Protection afforded by sickle-cell trait against subtertian malarial infection. Br Med J 1954;i:290-4.
- 10 Roberts DF, Lehmann H. A search for abnormal haemoglobins in some Southern Sudanese peoples. Br Med J 1955;i:519-21.
- ¹¹ Bonney GE, Konotey-Ahulu FID. Polygamy and genetic equilibrium. Nature 1977;265:46-7.
- ¹² Ringelhann B, Dodu SRA, Konotey-Ahulu FID, Lehmann H. A survey for haemoglobin variants, thalassaemia and glucose-6-phosphate dehydrogenase deficiency in Northern Ghana. Ghana Med J 1968;7:120-4.

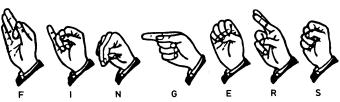
 13 Roberts DF, Boyo AE. On the stability of haemoglobin gene frequencies
- in West Africa. Ann Hum Genet 1960;24:375-87.
- 14 Dabbous IA, Firzli SS. Sickle-cell anaemia in Lebanon-—its predominance in the Mohammedans. Z Morphol Anthropol 1968;59:225-31.
- 15 Konotey-Ahulu FID. Maintenance of high sickling rate in Africapolygamy. J Trop Med Hyg 1970;73:19-21.
- ¹⁶ Konotey-Ahulu FID. Sickle-cell disease: the case for family planning. Accra: Astab Books Ltd, 1973:28.
- 17 Konotey-Ahulu FID. Taking health to Ghanaians. Tropical Doctor 1971; 1:134-7.

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Fingers and digits

B J FREEDMAN

My readers would doubtless agree that fingers are part of the human anatomy. Yet there is a certain evasiveness on the part of modern textbooks of anatomy in explicitly stating this fact. Fingers are mentioned, as it were, by implication. Gray's Anatomy (33rd edition) states, in the section on osteology, "The phalanges are fourteen in number, three for each finger and two for the thumb." Cunningham's Textbook of Anatomy and Grant's Method of Anatomy exhibit a similar obliquity of approach to the point that we normally have five fingers. I was distressed to find words of advice, in Grant's, that are scarcely appropriate to members of a dignified profession: "When striking, use the second and third knuckles." Possibly the writer had inter-hospitals rugby in mind. Lorenz Heister's Compendium Anatomicum (1721) is explicit and clearly states, "We come now to an examination of the hand, which is divided into carpus, metacarpus, fingers, and the little bones called sesamoides, because of their resemblance to grains of sesamum." Fingers and toes, however, get their due in the Nomina Anatomicanamely, the Basel N A, 1895; the Paris N A, 1955 (PNA); and the Birmingham Revision, 1933 (BR). Fingers are used primarily for grasping and feeling. The word is derived from the same root as fang (the tooth that grasps) and G fangen, to grasp. They have secondary uses in counting, measurement, and gesture. The various names that have been given to the individual fingers in the course of historical times have stemmed in part from these uses. It is of some interest to review these names.



-Deaf and dumb alphabetical hand signs spelling the word "fingers."

London N19 3TR

B J FREEDMAN, MB, FRCP, retired consultant physician

- (1) PNA: pollex, digitus I. BR: thumb; ultimately from L tumor, swelling; the stout or thick finger; compare Sw tumme, G Daumen. The implication of clumsiness in the phrase "all thumbs" is scarcely merited, since it is the most useful single digit for grasping and the one whose loss causes most disability. The thumb's power is implied in the phrase "under the thumb of. . . ." Indeed, L pollex derives from L pollens, powerful. Though its use in gesture has not contributed to nomenclature, British custom is the reverse of that in ancient Rome. In Britain thumbs-up signifies "all well" and thumbs-down "disaster." Contrariwise, at the gladiatorial games, thumbs-up from the spectators meant death to the vanquished, while thumbs-down meant "spare him." Thumbs-up in Britain was formerly a rude gesture (see middle finger below) and it still is so in much of Europe. Hitch-hikers abroad should beware. Thumbing a lift could lead to delays, second only to those of air travel.
- (2) PNA: index, digitus II. BR: index finger. The forefinger, pointing, indic/ator finger, from its use in the gesture of showing topographical direction. L digitus index, (gen indicis). L indicere, to point out, reveal, inform against—therefore the pointing finger, especially in exposing guilt. Also L digitus salutarius, the greeting or saluting finger.
- (3) PNA: digitus medius, digitus III. BR: middle finger. L digitus summus, the tallest finger. L. digitus infamis, disgraceful finger; digitus impudicus, shameless, lewd finger, so called because of its use in unbecoming, insulting, or lewd gestures. The "middle-finger jerk," with the middle finger extended upwards and the remaining fingers flexed, is still widely used in many European countries and in America as an insulting, obscene, or phallic gesture, though in Britain the extended index keeps it company (the Harvey Smith salute). In Arabic countries the middle finger is extended downwards, and one can only speculate on the reason for this. That the epithet infamis was used by Romans without any implication of impropriety is evidenced by the writings of the satirist Persius Flaccus, where he describes how to avoid the influence of the Evil Eye. "See how granny or aunt, in fear of the god, takes the boy from the cradle and, skilled at averting the evil eye, with her middle finger (infami digito) applies the charm with shining spittle on his forehead and little wet lips."