A Symposium held by the
Eugenics Society in October 1964

Biological Aspects of Social Problems

J. E. Meade
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Springer

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EDITORS' FOREWORD

THIS volume contains a report of the proceedings of a symposium on Biological Aspects of Social Problems, organized by the Eugenics Society and held in four sessions on 1st and 2nd October 1964, at the Botany Department of University College, London. In organizing this Symposium the Council of the Eugenics Society wished to promote an interchange of ideas between biologists and geneticists on the one hand, and sociologists, demographers, economists and others concerned with the social studies on the other. The rather naïve enthusiasms of some of the early social Darwinists, together with the fearful crimes committed allegedly for eugenic purposes, have undoubtedly made many students consciously or unconsciously reluctant to consider the genetic element in social situations. Yet in fact the great majority of human situations are affected by the interplay of genetic and environmental elements. It was the desire of the Council of the Eugenics Society, without *parti pris*, to set in motion a debate on these questions between biologists and those concerned with social problems. The Council believes that the experiment was a useful and successful one—a claim, the justification of which the reader of this volume can judge for himself—and it is the Council's intention to promote other similar symposia in the future.

The Council of the Eugenics Society would like to take this opportunity of thanking the contributors of papers, the participants in discussion, and the authorities of University College, London, for their help in making a success of this venture.

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A. S. PARKES

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GENERAL INTRODUCTION

SIR JAMES GRAY
Cambridge

A SYMPOSIUM provides a convenient means whereby people working in relatively specialized fields can learn what others in related fields are thinking about; new and stimulating ideas tend to arise at the border-line of two or more intellectual disciplines.

This, our first Symposium, is concerned with biological aspects of social problems and we are faced with the immediate difficulty that biologists and social scientists tend to speak somewhat different languages but the difference is more apparent that real. I suspect that to a Social Scientist ' industrial competition ' is not fundamentally different from a ' struggle for existence ' to a zoologist.

In introducing the Symposium, it is appropriate to recall that an essential step in the theory of animal evolution, as enunciated by Charles Darwin and Alfred Russel Wallace, was derived from the writings of the social economist, Thomas Malthus. It is now generally recognized that most of our social and international problems stem from an uneven distribution of limited environmental resources within increasingly large populations. It is just such a situation which brought about animal evolution. The role of the economist is to define the factors which control the distribution of resources between individual human beings and between different nations; but at the same time it is possible to regard economics as a specialized and very highly developed aspect of human biology.

If one views human society against the background of animal evolution, a number of common features are revealed, but one highly important difference emerges. In the animal world, limitation of environmental resources leads to a struggle for existence either between individuals or between species, or both; those best adapted to the environment survive whilst others are eliminated and, so long as the adaptive characteristics are inherited, evolution continues. More and more

highly adapted types arise. If there is no competition from an excess of population or from predators, natural selection ceases and no further evolution occurs. In other words, a struggle for existence is the price which Nature pays for the production of more and more highly adapted communities. The course of animal evolution is thus directed by the animal's external environment; there is little or nothing that the animal can do to improve his own chances of survival. In Man the situation is very different.

To a phenomenal extent man has not only learnt to modify and control his external environment but by speech and writing he can transmit the fruits of his own environmental experience to other individuals and to his offspring. He can therefore direct the course of his own evolution. But he cannot go on exploiting his environment and at the same time increasing his numbers indefinitely. He still lives under the shadow of a struggle for existence. Some escape must be found from this impasse. How far can the struggle be relaxed by reducing the population of the world? Or, in the absence of competition, will social evolution be slowed down?

The implications of an expanding world population seem so immense and so impersonal that many people find it difficult to face up to them; we prefer to put our heads in the sand and hope that there are experts who can deal with it. A recent leading article in *The Times* on the drift of world population asked the question, " Will the check be imposed humanely or inhumanly? " and ended with the words, " It is for the scientists, the politicians, the clergymen to decide. " This seems rather a curious attitude to recommend to a democratic society. The scientist's duty is to collect the facts and present them to the public in a form free from political and even national bias, thus allowing every individual to form his own judgement about what is at stake. Once the public grasp the urgency of the problem it will not be long before the politicians will find that something useful can and should be done.

POPULATION TRENDS

Chairman: Sir James Gray

DATA AVAILABLE FOR THE STUDY OF FERTILITY IN GREAT BRITAIN

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THE study of population statistics began with an interest in mortality data. The fundamental instrument for the study of mortality—the life table—was developed in the seventeenth and eighteenth centuries, and correct methods for constructing these tables were known in the nineteenth century. When civil registration began in this country in 1837, the principal questions essential to the study of mortality were asked at death registration: information was obtained about the sex, age and profession of the deceased person, as well as about the cause of death. In conjunction with census data, detailed studies of mortality became possible in 1841. Although some of the information collected has become more accurate than it was 125 years ago, particularly in respect of the cause of death, essentially the system has remained unchanged since the beginning of vital registration.

This earlier development in the study of mortality reflected the fact that the reduction of the death rate was one of the most pressing social problems of the nineteenth century. It is probably also true that at that time mortality was a more important determinant of population growth than was fertility. The comparative interest taken by official statisticians in the two problems is well shown, when one looks at the collection of William Farr's writings on *Vital Statistics*, edited by Noel Humphreys;[4] only twenty-one pages of the book were devoted to the study of fertility, as compared with over 300 dealing with mortality.

The Act of 1836, which set up the system of vital registration in England and Wales, required much less detailed information to be provided at the registration of a birth than was the case with deaths. No information was collected about the age of either parent at the birth of the child: all that was asked for was the child's sex, the date of birth and—in the case of

legitimate children—the father's rank or occupation. In Scotland births did not become registrable until 1855, and stillbirths were not registered until 1928.

The analysis of fertility in the nineteenth century had, therefore, to be conducted in terms of crude rates, where the total number of children born was related either to the population or to the number of women or of married women of reproductive age. The shortcomings of this method were evident to the statisticians of the time: William Farr in the *30th Annual Report of the Registrar General* commented on the 'grave defects' in the birth registers, and specifically on the absence of any information on the age of the mother at the birth of the child and on birth order. Oddly enough, the first schedule of the Scottish Registration Act remedied some of these deficiencies, but the relevant parts of the schedule were discontinued, after the Act had been in operation for a year. The data gathered in the first year were used, however, by Matthews Duncan in his book entitled *Fecundity, Fertility, Sterility and Allied Topics*, published in 1866, and containing the only information available in Britain in the nineteenth century on fertility by age of mother. In the absence of information on the marriage duration of women giving birth, Farr constructed indices of marital fertility by relating the total number of births to the marriages of six years earlier, six years being then the difference between the average age of women marrying, and the average age of mothers giving birth in Sweden. Towards the end of the century Edwin Cannan refined this index by relating births to a weighted average of marriages taking place in previous years, and he used this analysis to forecast the decline in the rate of population growth that was to take place in England in the first half of the twentieth century.

Interest in fertility grew during the second half of the nineteenth century, when the crude birth rate began to fall, and when the existence of fertility differences between different social groups became apparent. Studies of differential fertility were difficult in the absence of information on the numbers of children born to women in different social groups, and the early inquiries into differential fertility carried out in Karl

Pearson's laboratory made use of correlations existing between crude birth or fertility rates in different localities, and certain socio-economic indices.[3] Some twenty-five years earlier, Charles Ansell published in 1874 his analysis of the fertility of members of the upper and professional classes,[1] which yielded statistics on the distribution of families by size, but it is not clear from his work how his respondents were selected, and it is possible that his data were not representative, even of the limited group with which he was concerned. However, the concern with these problems led to the first significant advance in the collection of fertility statistics since the beginnings of vital registration—the introduction of fertility questions into the census of 1911. Four additional questions were asked of all married women enumerated:

(i) the total number of completed years of the present marriage
(ii) the total number of children born alive to the present marriage
(iii) the total number of children still living and
(iv) the total number who had died.

Widows and divorced women were not required to provide this information, and the final tabulations did not even relate to all married women, but only to those 91 per cent who were enumerated on the same census schedule as their husbands, so that differential fertility could be studied in relation to the husband's occupation. Clearly, this restriction imposed serious limitations on the inquiry. The fertility of women under forty-five years of age was still incomplete, and, in the case of women over forty-five, their chance of inclusion in the tabulation depended upon the ages of their husbands; the younger their husbands were, the greater it was. There may well have been a correlation between marital fertility and the age of the husband which could have biased the results. The publication of the results of the 1911 inquiry was held up by the First World War, but they enabled T. H. C. Stevenson, then the Superintendent of Statistics at the General Register Office to write his important paper on differential fertility,[6] in which he traced the fertility of the various social classes in England and

Wales from the middle of the nineteenth century to 1911. It was also possible for the first time to obtain estimates of childlessness both for the country as a whole, and for different social groups.

In the meantime certain advances had been made in the measurement of current fertility. In 1884 Böckh calculated a net reproduction rate for the first time, and in 1907 R. R. Kuczynski developed the gross reproduction rate. Both these indices made use of a synthetic approach: the age-specific fertility rates of women in a particular calendar year were combined with mortality data of the same year, or summed on their own to obtain the net and gross reproduction rates respectively. The age of the mother was considered to be the important variable: just as, in the case of mortality, the risk of dying was a function of age, so the age of a woman was considered to be the most important variable associated with fertility. The use of these age-specific rates and the restriction of the analysis to women implicitly assumed that the biological factor was most important in the study of fertility. Thus, comparatively little interest was taken in nuptiality and the duration of marriage as factors which might influence fertility. These rates were widely used in the discussion of population problems in the inter-war period and the general public (outside the ranks of demographers) became familiar with them: thus, Sir Dennis Robertson in an address to the Royal Economic Society began by reminding his audience that like every patriotic Englishman he carried two memoranda in his pocket: one urging him to increase exports by at least 50 per cent, the other to raise the net reproduction rate to at least unity.[5]

British official statistics did not provide the information necessary for the calculation of exact reproduction rates, and as these were increasingly used in demographic analysis there were demands for a revision of the system of registration which would enable their values to be computed. These demands led to the passing of the Population (Statistics) Act of 1938, the first major revision of the system of birth registration since its inception in 1836. The gaps deplored by Farr in the 1860s were at last filled, and questions were now asked relating to the

age of the mother, the duration of her marriage and the order of birth of the child. The Second World War led to a delay in the publication of these data: but when they were at last published, it became possible to analyse fertility by age of mother, duration of marriage and birth order or by a combination of these factors, for the period beginning 1st July 1938.

Unfortunately, this information became available at a time when the focus in the study of fertility had shifted. In 1944 concern with the population of the country led to the appointment of a Royal Commission, which was advised on technical statistical problems by an expert committee under the chairmanship of Sir Alexander Carr-Saunders. When the Committee came to survey the fertility statistics of the period, it became clear that age-specific fertility rates and combinations of these rates in the form of reproduction rates were not really very useful as indicators of long-term fertility trends. The Royal Commission was advised that a special *ad hoc* type of inquiry was necessary for a proper assessment of fertility and, with the agreement of the Treasury, the Family Census of 1946 was undertaken, covering a sample of 10 per cent of all ever-married women in the country. This census differed from its predecessor of 1911 in several important respects. In the first place widows and divorced women were included, and a question was asked about the date of the end of their first marriage, so that the period during which they were at risk of bearing legitimate children could be ascertained. Secondly, information was obtained not only about the dates of birth and marriage of the women themselves and the total number of children they had borne, but questions were asked about the date of birth of each individual liveborn child.

The inclusion of this type of information made a new kind of fertility analysis possible and provided statistics of fertility which were more detailed than those collected for any other country. It became possible to build up a picture of the process of family building and to trace the distribution of groups of women married at a particular period of time (so-called ' marriage-cohorts ') at any specific duration of their marriage. Thus, it was no longer necessary to confine the analysis to women whose fertility was complete, but it could be extended to those

who were still fertile. Under modern conditions information about women whose fertility is complete tends to be of purely historical interest by the time it becomes available, as the bulk of their childbearing will have taken place in the past. Current fertility rates on the other hand are liable to fluctuate from year to year, and to be unduly influenced by short-term factors. The use of data of the type collected in the Family Census makes it possible to compare the fertility of women belonging to different marriage cohorts who had been married for ten, fifteen or twenty years, say, and who will therefore have had the same length of exposure to the risk of childbearing within marriage. As some 80 per cent of marital fertility falls within the first ten years of marriage, these women will have passed the period of maximum fertility, but will have had their children at a time sufficiently near the census date to yield information relevant to current fertility.

Since the Family Census the data collected under the Population (Statistics) Act have been ' spliced ' on to Family Census data so that statistics of average family size at various specified durations of marriage may be computed for married women as a whole, but not for separate social groups, and this is now done as a matter of routine in the Registrar General's *Statistical Review.*

No new detailed inquiry, such as the Family Census, has taken place since 1946, though the national censuses of 1951 and 1961 both included questions on marital fertility. In the former the inquiry was limited to married women under the age of fifty, but in 1961 all women who were then, or had in the past been, married were asked to state the date of their present and first marriage, and the total number of children that they had borne alive. In 1961 a question was asked relating to the date of termination of the first marriage, where this was applicable; and on both occasions women were asked to state whether or not they had borne a live child in the year immediately preceding the census enumeration. The fertility volume of the 1951 Census was published in 1959: no indication has as yet been given as to when the results of the 1961 Census on fertility will appear.

The above brief sketch of the development of fertility

statistics will have made it apparent that—since 1938—extremely detailed information exists on the relation of marital fertility to such variables as age of mother, duration of marriage and birth order. Indeed, the only major gap that remains is the absence of information on the spacing of successive births, a gap which could be filled very easily by including a question on the date of birth of the previous child at birth registration, wherever this question was applicable. A question of this type would produce valuable additional information which would help in the understanding of movements in fertility.

However, the really interesting—and from the point of view of forecasting, a crucial—aspect of the study of fertility lies in the understanding and documentation of group differences in fertility. Recent work in America by Freedman[2] suggests that such differences are becoming less important than they used to be. In Britain, we can study fertility differences between different socio-economic groups, as defined by the Registrar General on the basis of the occupation of the father, in the triennia with the population census as a centre, because it is only in the census that information sufficient to compute the basic population at risk is available. In 1951, too, a brief classification of fertility by the terminal education age of the husband was attempted in the fertility report of the census, but this information was of limited value as the questions on education in 1951 were restricted and badly phrased. But it is not possible from official data to relate fertility data to such important social variables as the previous occupational history of the mother, her own educational status and the social mobility of the family. For information of this type, it is necessary to rely on private inquiries, such as those conducted by the London School of Economics, or the Population Investigation Committee. Nor has an inquiry of the Family Census type been repeated, even though it is now nearly twenty years since 1946, and married women of a whole new post-war generation have almost completed their fertility.

Indeed, it almost seems as if fertility studies have been relatively neglected by the General Register Office. The fertility report of the 1951 Census took eight years to publish,

and at the date of writing there is no indication that the 1961 Report will appear more quickly. It is clear that in the census programme the analysis of the geographical distribution of the population and of its industrial and occupational characteristics is given precedence over the study of fertility. This is probably inevitable in view of the small professional staff in the General Register Office. There appears, for instance, to be no person in that office who is *exclusively* concerned with the study of fertility. This comparative lack of interest seems strange, as under modern conditions fertility, rather than mortality, is the most important determinant of population growth.

The high standard of our vital statistics systems should not blind us to the fact that improvements have been slow. Perhaps this feature is not unconnected with the organization of the General Register Office. The Registrar General is an administrative official, presiding over a department which has a dual task: the maintenance of the system of civil registration and of records, and the analysis of the data collected by the system. Ever since William Farr in 1880 failed to achieve promotion to the headship of the office, which he coveted, and for whose reputation and achievements in its early years he was so largely responsible, successive Registrars General have been drawn from the administrative class of the civil service, and have been men whose main interest did not lie in the statistical side of the work. They were fortunate to be assisted on the technical side by professionals of great ability, but ultimate control over policy remained with the administrators. This system was criticized by the Statistics Committee of the Royal Commission on Population, who wrote that " the Registrar General should normally be himself a person qualified to deal with statistical issues " and that " for future appointments to the position of Registrar General some experience of and competence in statistical work should be a necessary condition. " The Royal Commission itself endorsed these recommendations in general terms and added that " at present in Great Britain the arrangements for the collection and analysis of fertility statistics are not adequate to modern needs " (Para. 682).

However, the position has not changed since the Royal

Commission reported fifteen years ago. The number of professional statisticians in the General Register Office has increased and the Statistics Division no longer has an administrator in charge; but control remains in the hands of the administrative officials. In the type of work that lies within the responsibility of the General Register Office, the distinction between professional, technical and administrative duties is not always easy to draw: administrative decisions taken about the system of census taking and vital statistics will determine the types of analysis that are possible. It is the statistical work of the General Register Office that is increasingly becoming the most important part of its duties, and it is no disrespect to the distinguished holders of the Registrar General's office to suggest that a general administrative training is no longer an adequate qualification for the headship of an office engaged on increasingly complex and technical work. It is difficult to understand why the recommendations of the Royal Commission on Population in this field have been completely ignored by politicians and civil servants alike. Our system of fertility statistics has reached a high standard of accuracy and completeness of collection, but it has been slow to change. Improvements would be made much easier if a change in administrative structure could be made.

DISCUSSION

PROFESSOR GREBENIK, referring to a suggestion about the need for an extension of fertility surveys, agreed that it was desirable that the General Registrar's Office should undertake small-scale sample surveys into aspects of fertility. He thought it extremely noticeable that in the series on medical and population subjects, the GRO had been concerned very much more with problems of mortality and morbidity than with fertility. Indeed, he was not aware of any study that the GRO had made on fertility. He thought this might not be unconnected with the fact that we were all agreed in what we thought about mortality: we were agin it; but we were not all agreed on what we thought about fertility. Our attitude was ambivalent and politicians and civil servants were loth

to touch it. However, Professor Grebenik said, he would not go all the way with the questioner in saying that we should not try to extend the large scale collection of statistics of factors which might influence fertility. He did, for instance, think that an inquiry in a population census, even (as in 1956) on a sample basis, into such matters as the occupation or the work-history of married women before marriage in relation to their fertility, could be undertaken with great profit. He believed that more information about birth spacing could be obtained quite easily by a very simple amendment to the Schedule of the Population (Statistics) Act, and that this would give a great deal of information about the process of family formation.

REFERENCES

1. ANSELL, C. jr. 1874. *On the Rate of Mortality at early Periods of Life, the Age at Marriage, the Number of Children to a Marriage, the Length of a Generation and other Statistics of Families in the Upper and Professional Classes.* London. National Life Assurance Society.
2. FREEDMAN, R. 1961–62. The Sociology of Human Fertility. A Trend Report and Bibliography. *Curr. Sociol.* **10–11**, 2.
3. HERON, D. 1906. On the Relation of Fertility in Man to Social Status and on the Changes in the Relation that have taken place during the last Fifty Years. *Drapers Company Research Memoirs. Studies in Natural Deterioration* **1**.
4. HUMPHREYS, N. A. (Ed.). 1885. *Vital Statistics.* Memorial Volume of Selections from the Reports and Writings of William Farr. London. The Sanitary Institute.
5. ROBERTSON, D. H. 1945. The Problem of Exports. *Econ. J.* **55**, 321.
6. STEVENSON, T. H. C. The Fertility of the Various Social Classes of England and Wales from the Middle of the Nineteenth Century to 1911. *J. R. statist. Soc.* **83**, 401.

PATTERNS AND TRENDS IN RECENT BRITISH POPULATION DEVELOPMENTS

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THE title of this talk suggests a range of subjects more sweeping than can adequately be surveyed within the space of half an hour. My remarks must therefore be limited to a selection of topics: and in choosing these the first consideration must be the object of this Symposium, which is to highlight those problems that are of mutual interest to biologists and social scientists of all kinds.

Perhaps the most obvious way to satisfy this need would be to concentrate on demographic data that bear directly on human biology. This form of approach is illustrated by M. Louis Henry's paper to the Royal Society two years ago, for the Graunt Centenary,[2] when he dealt with the natural intervals between births, uncontrolled family size, sterility and the like. Such subjects, however, require recourse to data from many countries and it is not really practicable to speak of them in relation to Great Britain alone.

Moreover, to believe that biologists are interested only in purely biological population data is to do an injustice to the many among them who show a lively interest in, and appreciation of, social problems. Indeed, scientists of all types can readily occupy themselves with such problems if they have a mind to; demography at least is a relatively uncomplicated form of study, relying as it does mainly on observation, classification and exposition. A good knowledge of, and careful adherence to, the facts are particularly important.

Bearing this in mind, some interesting inquiries may be pursued by searching carefully through the mass of published population data in an attempt to track down any stable patterns, or uniform trends, which on a careful appraisal seem to be genuine, to be capable of rational explanation, and to bear a sufficiently recognizable relationship to other happenings to give some hope of predicting, rather than just guessing,

the course of future events. It is of three small pieces of research of this kind that I now propose to speak. Each is related to a principal demographic feature of the present day. My remarks are necessarily personal and in no way in an official capacity, and the data used will relate to England and Wales exclusively.

To start with marriage; the recent experience in this country may be briefly characterized by the proportions married at specimen ages for each sex. Table I shows that the proportions varied relatively little between 1911 and 1931—it is surprising that the war of 1914–18 had so little effect,

TABLE I

PROPORTIONS MARRIED 1911–61

England and Wales

AGE		1911	1921	1931	1951	1961
25	Men	·37	·41	·37	·53	·60
	Women	·46	·48	·48	·71	·79
35	Men	·77	·80	·83	·84	·84
	Women	·74	·73	·75	·83	·88
45	Men	·82	·84	·86	·88	·88
	Women	·73	·74	·73	·79	·84

particularly for women—but by 1951, and again by 1961, they had increased materially.

The figures show that women marry younger than men but also suggest that fewer of them ultimately have done so. The post-war increase is most marked at age twenty-five and least evident at age forty-five; a point of particular interest is that it is greater for women than for men.

External influences, such as economic developments, have almost certainly operated upon the marriage experience, but would be expected to affect men and women similarly. An interesting question, therefore, is whether the differing tendencies for the two sexes can be explained in terms of the changing balance of their numbers (it happens that for a variety of reasons this balance is altering rapidly and a preponderance of women is changing over to a preponderance of

men). In this connection it is significant to look at the comparative ages of men and women at marriage, and Table II shows some specimen figures for 1931 and 1961.

It might reasonably be supposed that the distribution of preferences of marrying men for brides of given ages, and that of marrying women for bridegrooms of given ages, were constant

TABLE II

PERCENTAGE DISTRIBUTIONS OF AGES OF SPOUSES AT MARRIAGE, 1931 AND 1961

England and Wales

Sex of one Spouse and age of that Spouse at Marriage			Distribution of other Spouses according to their age at Marriage						
			16–19	20–24	25–29	30–34	35–39	40 and over	Total
MEN	20–24	1931	18	68	13	1	...	...	100
		1961	36	58	5	1	...	...	100
	25–29	1931	4	48	41	6	1	...	100
		1961	14	59	21	5	1	...	100
	30–34	1931	2	24	42	24	6	2	100
		1961	6	35	31	18	7	3	100
WOMEN	16–19	1931	12	67	18	2	1	...	100
		1961	19	65	13	2	1	...	100
	20–24	1931	1	51	40	7	1	...	100
		1961	2	60	30	6	1	1	100
	25–29	1931	...	16	57	19	5	3	100
		1961	...	21	43	22	9	5	100

elements: yet in fact during a period of thirty years considerable changes occurred. Men, whatever their age at marriage, were choosing partners in marriage about one-and-a-half years younger (on the average) in 1961 than in 1931; women, too, have come to choose younger partners, although for them the change is smaller—only about half a year. Is this a biological development, associated perhaps with earlier maturation, or has some other cause been at work? In order to examine further the possible influence of the balance of the

sexes available for marriage, it is desirable to define the concepts of 'supply' and 'demand' in relation to marriage partners. Let the demand at a given age be represented by the numbers of either sex at that age who are unmarried. Then the supply should have regard to a distribution of ages of partners of the kind shown in Table II; let it be represented by the numbers unmarried among the other sex weighted with the proportions in which the other sex are chosen. Thus for men aged twenty to twenty-four, for instance, the supply in 1931 was 18 per cent of the numbers of women aged sixteen to nineteen, plus 68 per cent of women aged twenty to twenty-four. and so on. Here are the total figures of supply and

TABLE III

SUPPLY AND DEMAND IN RELATION TO MARRIAGE PARTNERS, 1931–61

England and Wales

	MEN			WOMEN		
YEAR	Demand	Supply	Ratio	Demand	Supply	Ratio
1931	4636	5781	1·25	4907	4190	0·85
1941	4386	4873	1·11	4179	4057	0·97
1951	3864	3964	1·03	3346	3382	1·01
1961	3720	3729	1·00	2820	3069	1·08

demand on this basis. They include widowed and divorced persons, and relate to demand arising at ages fifteen to forty-four.

For men, demand and supply have come reasonably in balance, as a result of the changes that have taken place in the preference proportions. If, however, the 1931 preference proportions had remained unchanged, the ratio of supply to demand for men would have fallen to 1·06 by 1941, 0·95 by 1951 and to 0·80 by 1961. So, in order to find enough partners, men have *had* to marry younger women. The changes in the relative ages of marriage partners could thus be interpreted not as a biological development but as the effect of a simple balance of numbers.

For women, the smaller changes in the age-distribution of partners that have occurred have not been important in

the same way, as even on the 1931 distribution the supply in 1961 would have exceeded the demand by 4 per cent. The product of the ratios for the two sexes is nearly constant in time. The changes that have occurred for women might be a reflection of the developments for men, which would be in accordance with the generally accepted notion that it is the man who takes the lead in such matters.

This analysis offers little direct help in the estimation of future marriage trends generally, but at least it illustrates how the assumptions on which population projections are founded need to be tested for consistency between the sexes, having regard to the numbers likely to be available for marriage from time to time.

My second subject is that of fertility. It is one of special importance, and the shifting patterns and prospects in Britain are of perennial interest. Figure 1 is in the form of a graph and shows the course of the numbers of births over the last twenty years. Their low level before the Second World War, the wartime rise and the post-war peak have all been discussed fully in recent years. But now there is a new feature—the steady and substantial rise in births since 1955, not preceded by any corresponding increase in the numbers of marriages.

The implications of this most recent change for population projections, and for the future possibilities they depict, are far-reaching, as Figure 2 shows. Up to 1955 there was little or nothing in the data, in the published literature, or in anyone's experience to suggest otherwise than that a peacetime plateau had been reached. The official projection published in 1954 was accordingly based on the assumption that births would stay at their current level, subject to a gentle fall reflecting a slight diminution in the number of married couples. When the upward movement in births began, no one could be sure whether or not it suggested a minor fluctuation, and at the start at least the most reasonable assumption was that it did. Thus, the projection made from the year 1958, for instance, assumed that the numbers of births would, in general, remain at the level they had reached.

With the passage of time, however, it has become evident that this is no small variation but a major development;

only as such can the sharp increase of 20 per cent or more in births be interpreted. Indeed, there seems now to be no special reason for supposing that the trend will speedily be reversed, although of course it is unlikely to continue unchecked for an indefinite period. Hence, the most recent published projection now assumes that the number of births will continue to increase, though less rapidly than of late. A rise of one-third

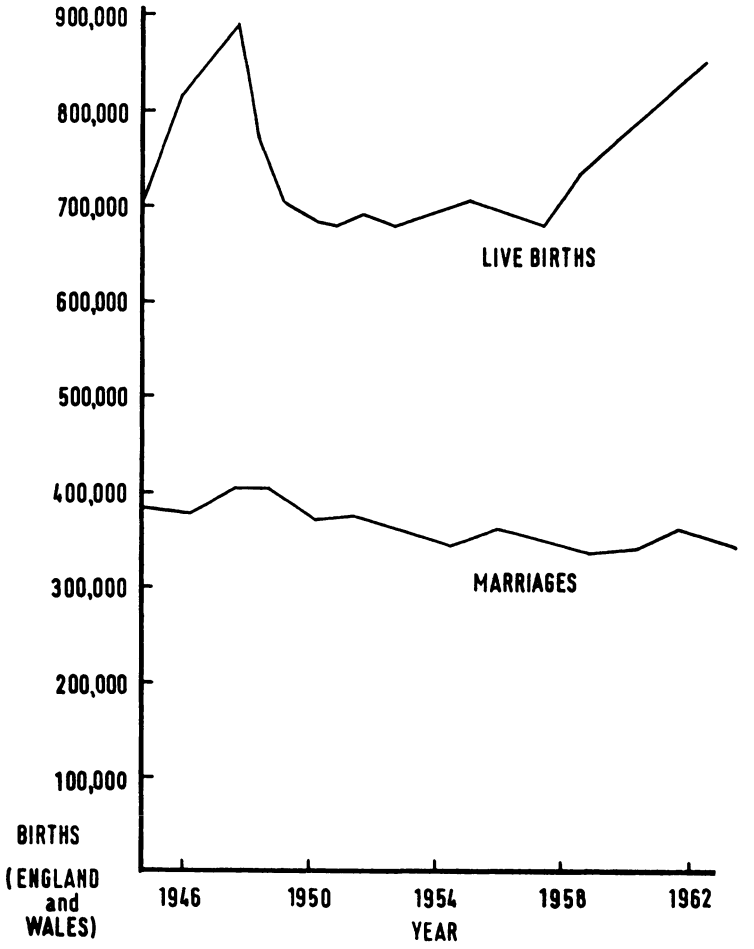

FIGURE 1

is expected by the end of the century, but much of this is attributable to the growth in the number of parents, which is itself a consequence of the recent developments and their expected continuation during the next few years. (The immigration picture has also changed the outlook somewhat.)

The implications of these changes of outlook are striking, as Table IV shows. The effects grow with the number of years one looks ahead. Even by 1963 the population had

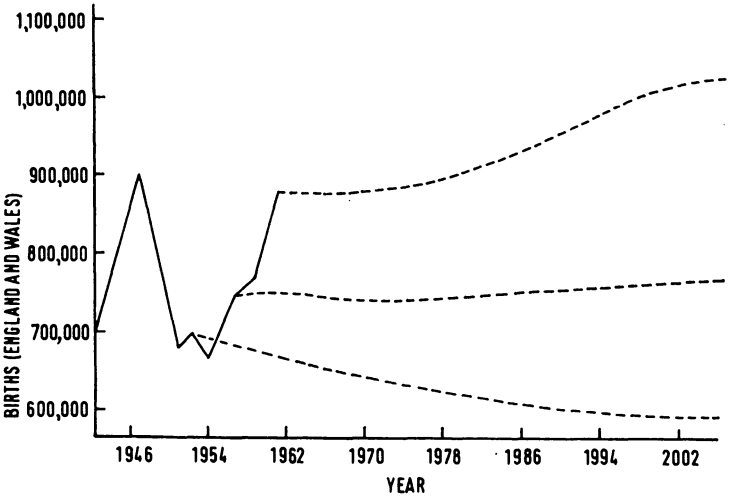

FIGURE 2

increased by two million over the expectation. By the 1970s there is a rise of five million (10 per cent) and at the end of the century the difference is no less than 18 million persons, or as much as 40 per cent. In view of these differences, and more particularly because it is desirable that everyone should understand what is happening to-day, analysis of the trend of births and fertility is specially vital.

Perhaps the most significant factors with which fertility is associated are age at marriage, length of time married and the date of marriage. Let us consider first how completed family size has been associated with age at marriage from time to

time. The number of children per couple has varied a good deal during the present century. For instance, women married under age twenty during 1900–09 had an average of five children in all whereas those married as young to-day have

TABLE IV

PROJECTED TOTAL POPULATION OF ENGLAND AND WALES

Year from which projection was made	Projected Population for Year—Millions				
	1963	1973	1983	1993	2003
1953	45	46	47	46	46
1958	46	48	50	51	53
1963	47 *	51	55	59	64

* Actual.

three. Those married at 25–29 in the early years of the century had nearly three children on average but this has fallen to two (Table V). It might reasonably be supposed that the proportionate differences in family size between younger and

TABLE V

AVERAGE COMPLETED FAMILY SIZE : ENGLAND AND WALES

AGE OF WIFE AT MARRIAGE	DATE OF MARRIAGE				
	1900–09	1910–14	1915–19	1929	1939
Under 20	5·2	4·6	4·0	3·4	3·1
20–24	3·8	3·4	3·0	2·4	2·2
25–29	2·7	2·4	2·1	1·7	1·7
30–34	2·1	1·8	1·7	1·2	1·2
35–39	1·3	1·1	1·0	0·7	0·6
40–44	0·7	0·5	0·4	0·2	0·2

older marriages have diminished over the years. In fact they have remained relatively constant, as Table VI shows.

The data exhibited in Tables V and VI derive from two separate sources of information, with slightly different

characteristics, and their results are not perfectly consistent at the point of contact—the year 1929. Nevertheless, the picture is broadly representative.

In theory a shift of a certain proportion of first marriages from one age to another in Britain need not make any difference to the size of the family the couple may have, in view of the fact that family planning is very widely and successfully employed in this country. In practice, the effect of the planning

TABLE VI

RELATIVE COMPLETED FAMILY SIZE ACCORDING TO AGE OF WOMAN AT MARRIAGE AND CALENDAR YEAR OF MARRIAGE

England and Wales

YEAR WHEN MARRIED	AGE OF WOMAN AT MARRIAGE					
	Under 20	20–24	25–29	30–34	35–39	40–44
1900–09	100	74	52	40	24	13
1910–14	100	74	51	40	24	10
1915–19	100	74	52	42	25	10
1920–24	100	74	52	41	24	11
1929	100	69	48	34	19	7
1934	100	68	49	34	21	11
1939	100	72	57	40	20	5
1944	100	78	64	47	24	8
1949	100	70	56	40	22	7

has been to reduce family size by a constant proportion all round rather than to produce a ' target ' number of (say) two children for couples of all ages. One result of this is that as the age-distribution of marriages becomes younger, the average family size for all married women grows, and the effect is appreciable: perhaps up to 10 per cent in all.

There are some interesting minor variations in Table VI, such as the ' closing up ' in the age differences around 1944 and the ' fanning out ' that has occurred since then, but it is to the comparative steadiness of the ratios rather than their divergence that attention seems most worth directing for present purposes.

The next table illustrates the effect on fertility of the length of time married:

TABLE VII

PROPORTION OF TOTAL CHILDREN IN FAMILY BORNE BY SPECIFIED DURATIONS OF MARRIAGE

England and Wales

NUMBER OF YEARS MARRIED	ALL AGES AT MARRIAGE			AGE OF MOTHER AT MARRIAGE								
				Under 20			20–24			25–29		
	Married in			Married in			Married in			Married in		
	1929	1939	1949	1929	1939	1949	1929	1939	1949	1929	1939	1949
1	·18	·12	·15	·19	·14	·15	·17	·11	·14	·16	·12	·16
2	·30	·23	·29	·28	·22	·27	·30	·21	·28	·30	·23	·31
3	·39	·32	·39	·35	·31	·36	·38	·30	·38	·41	·33	·42
4	·47	·40	·48	·41	·37	·44	·46	·38	·47	·50	·43	·52
5	·54	·48	·56	·47	·43	·51	·53	·47	·55	·58	·52	·62
6	·61	·56	·64	·52	·50	·58	·59	·54	·63	·66	·60	·69
11	·83	·85	·88	·73	·74	·81	·81	·85	·87	·88	·91	·93
16	·94	·95	·96	·86	·91	·90	·94	·96	·98	·98	·99	·99
21	·99	1·00	1·00	·97	·98	·99	1·00	1·00	1·00	1·00	1·00	1·00
26	1·00	1·00	1·00	1·00	1·00	1·00	1·00	1·00	1·00	1·00	1·00	1·00

The influence of the Second World War in slowing down the family building of couples married in 1939 is evident, as also is the fact that younger couples necessarily take longer to complete their (larger) families than do those who marry at older ages. These points apart, there is a considerable stability of pattern in the figures but, even so, to predict the completed family size from the progress of couples in the first few years of marriage must remain a somewhat hazardous process.

If these are some of the principal features of fertility trends in this country in the longer term, what can be said of the most recent short-term movements? Do they reflect the same pattern, or is a new configuration being set up? A few specimen fertility rates are shown in Table VIII. A detailed analysis of the full series of figures discloses that the rates have grown year by year since 1955. They have also increased for all ages

TABLE VIII

RECENT FERTILITY RATES CLASSIFIED BY AGE OF MOTHER AT MARRIAGE AND DURATION OF MARRIAGE

England and Wales

DURATION OF MARRIAGE IN YEARS	AGE AT MARRIAGE											
	Under 20			20–24			25–29			30–34		
	1955	1958	1961	1955	1958	1961	1955	1958	1961	1955	1958	1961
0	·418	·433	·480	·260	·275	·293	·251	·275	·304	·234	·247	·275
3	·260	·265	·283	·212	·229	·257	·193	·222	·249	·163	·162	·191
6	·178	·195	·191	·141	·159	·166	·120	·135	·145	·077	·085	·088
9	·119	·128	·136	·083	·091	·100	·066	·070	·076	·027	·029	·033
12	·074	·085	·091	·051	·056	·060	·030	·035	·034	·006	·007	·004
15	·058	·055	·062	·031	·034	·036	·012	·012	·014	·001	·001	...

at marriage, for all sizes of family, and for all durations of marriage. Some summary figures are given in Table IX:

TABLE IX

PERCENTAGE INCREASES IN FERTILITY RATES, 1955–61, CLASSIFIED ACCORDING TO VARIOUS CHARACTERISTICS

England and Wales

1. AGE OF MOTHER AT MARRIAGE	Under 20	13
	20–24	18
	25–29	20
	30–34	14
2. PARITY OF MOTHER	No children	8
	One child	17
	Two children	22
	Three children	20
3. DURATION OF MARRIAGE	0–2 years	15
	3–5 years	16
	6–8 years	17
	9–11 years	19
	12–14 years	17

Of these developments, by far the most uniform is that related to duration of marriage; and this suggests that the change is essentially a secular one. In other words, the recent rise in fertility is essentially a function of the calendar year of experience. A corresponding analysis arranged according to

the experience of couples married in successive years shows fluctuations between these cohorts which suggest that their fertility over this period is more directly associated with the calendar years during which they have been married women than with the date of their marriage as such.

The recent fertility increases are rather smaller for the youngest marriages than for the older ones. They are also less marked for first children than for second, third and fourth children, a feature that suggests a definite (if small) trend towards a larger family size. In this, and some other respects, the current developments do seem to be exhibiting some novel features as compared with the long-term patterns.

A short-term change that appears to be related mainly to the calendar years passed through would appear likely to be the direct result of an ' external ' influence such as the state of the economy, the housing situation or a change in fashion. From this one might suspect it to be in the nature of a temporary fluctuation. Nevertheless, the possibility of a beginning of a new long-term development certainly cannot be wholly excluded at this stage. What then are the implications for the future? It does not seem very likely that the recent upward movement in the numbers of births will be speedily reversed into a decline. Indeed, the increases might well ease off gradually, and a new plateau be reached in a comparatively short time. These conclusions are neither unexpected nor revealing and could well have been drawn without making any detailed study of the figures. Indeed, while it is possible to illustrate the fertility developments by means of demographic data, it is hardly practicable to explain them in this way. Possibly a careful economic analysis would be capable of throwing more light on the matter, but this also could hardly provide an accurate method of forecasting the future.

I now turn to mortality, and Table X illustrates the principal features of recent developments in the death rate from all causes in England and Wales. The comparative steadiness in male mortality at age fifty-five and over is evident.

In the latest of their series of impressively thorough investigations of the statistical associations between smoking

and mortality, Sir Austin Bradford Hill and Professor Doll came to the conclusion that " one of the striking characteristics of British mortality in the last half century has been the lack of improvement in the death rate of men in middle life. In cigarette smoking may lie one prominent cause."[3] This statement was based mainly on a direct association between the number of cigarettes smoked and the chance of death from (1) carcinoma of the bronchus, (2) chronic bronchitis, and (3) coronary thrombosis.

TABLE X

DEATH RATES BY SEX AND AGE (PER 1000)

England and Wales

	MEN				WOMEN			
	34–44	45–54	55–64	65–74	35–44	45–54	55–64	65–74
1926–30	6·2	11·6	24·4	58·3	4·7	8·4	18·0	44·4
1936–40	5·0	11·0	25·0	56·8	3·8	7·5	16·5	41·8
1946–50	3·2	8·6	22·4	51·6	2·6	5·5	12·8	34·4
1956–60	2·4	7·4	21·9	53·7	1·8	4·5	10·9	30·7

Mr R. E. Beard, in an ingenious demonstration delivered to the Royal Society in 1962,[1] analyzed the course of lung cancer death rates by age and sex over the last fifty years and showed that they could be regarded as being composed of an age component, a component varying with the calendar year, and a generation element dependent on the year in which the person was born. These components, when multiplied together, reproduced the death rates. He suggested that the generation element could well represent the proportion of persons who smoke cigarettes and the secular element the amount smoked per head; and indeed he was able to correlate his factors with measures of the volume of smoking. Both these elements have increased over the years, and thus their effect on the death rate is two-fold.

It seems a worthwhile exercise to attempt to apply Beard's analysis over the wider range of causes of death now shown by Bradford Hill and Doll to be relevant. This can be done in a variety of ways. One possibility is to use his method in relation to the death rates from chronic bronchitis or coronary thrombosis, or to the two causes combined. These rates are

considerably higher than those for lung cancer, and their distribution by sex, age and time is different from both that of lung cancer and from that of each other. The statistics of deaths from these two causes have, however, been considerably affected by changes in classification over the years, and it is not possible to identify coronary disease as such in British data before about 1930. Moreover, some of the deaths from these causes are probably attributable to factors other than smoking; for instance, overweight, or large consumption of alcohol or sugar have been adduced as correlated conditions. For these and other reasons I doubt whether it is really of value to apply Beard's analysis to the recorded data of deaths from coronary thrombosis or chronic bronchitis.

There is, however, another group of ways in which the question may be tackled. The absolute, or proportionate, improvements in women's mortality from all causes can be calculated, age by age, over the last fifty years, and compared with the corresponding figures for men. A study can then be made of the characteristics of the extent to which any improvement in the men's experience has failed to keep pace with that in the women's. This course has the advantage that the results are unaffected by changes in the rules for classification by cause, or in practice in so classifying deaths, or in the method of dealing with multiple causes. It does, of course, suffer from the disadvantage that the relative lack of improvement for men, as compared with women, may well be associated in some degree not with all men's smoking but rather with the excess of men's smoking over women's smoking. It seems, however, from the evidence quoted by Beard, that even to-day the ratio of tobacco consumption of women to that of men is only one-third or less: and that in the past it was much lower; women's deaths from lung cancer even now amount only to about one-sixth of men's; the disadvantage is not, therefore, a serious one.

The analysis may be applied to either of the following functions for all ages and years of experience:

(*a*) The actual fall in women's mortality rate since a given base year *minus* the actual fall in the men's rate, or

(*b*) The percentage diminution in women's mortality since a given year *minus* the corresponding percentage for men: the result to be multiplied by the men's rate in the base year.

Let us suppose that in 1911 the rates for men and women at a given age were respectively ·0100 and ·0080, and that by 1961 the corresponding rates were ·0060 and ·0032. Then the first method produces a result of ·0048 (the fall for women) minus ·0040 (the fall for men) or ·0008, while the second method produces ·0100 × (60 per cent minus 40 per cent) or ·0020. For a variety of reasons it seems better to work to method (*b*) —the percentages—and the data are as follows taking 1910–12 as the base period:

TABLE XI

PERCENTAGE FALL IN MORTALITY SINCE 1910–12 FOR WOMEN MINUS THE PERCENTAGE FALL FOR MEN

England and Wales: All Causes

	AGE GROUPS									
YEARS	35–39	40–44	45–49	50–54	55–59	60–64	65–69	70–74	75–79	80–84
1921–25	3	4	3	...	2	...	3	2	4	2
1926–30	3	7	6	2	2	2	5	6	7	2
1931–35	−1	3	5	5	6	2	6	5	7	5
1936–40	3	4	6	11	13	10	8	8	11	12
1941–45	3	4	7	11	15	16	12	10	9	9
1946–50	−1	2	8	12	17	22	16	11	9	9
1951–55	1	1	6	16	21	24	24	19	16	12
1956–60	2	2	5	14	25	26	23	23	16	16

At ages below thirty-five many of the figures are negative.

A study of these figures, in comparison with the lung cancer death rates operated upon by Beard, discloses that they are less smooth in their progression from age to age or from year to year. Moreover, they advance more rapidly with increasing age than the lung cancer rates do, and in the Table the secular trend is much flatter at the younger ages than it is at the old—a more intractable complication than confronted Beard.

The technical problem of finding age, secular and generation functions whose products will approximate to this series

of numbers is of relatively minor interest, especially as effective tests of significance of fit are hardly applicable. What is of much more significance is to discover whether the underlying influences revealed by the analysis are similar to those disclosed by Beard. If these influences proved to be exactly the same, then both his thesis and that of Hill and Doll would be strikingly confirmed. In fact, the dissimilarity of the two sets of data rules out very striking confirmation, owing no doubt, *inter alia*, to other causes of difference between the sexes in the progression of their mortality rates. Nevertheless, the results are quite interesting. It is possible to arrive at an approximate presentation of the figures as the product of age, secular and generation factors as the following specimen results show:

TABLE XII

ACTUAL AND EXPECTED PERCENTAGE MORTALITY DIFFERENCES
England and Wales

YEARS	AGE GROUPS							
	40–44		50–54		60–64		70–74	
	Actual	Expected	Actual	Expected	Actual	Expected	Actual	Expected
1921–25	4	2	...	2	...	3	2	2
1926–30	7	3	2	4	2	4	6	4
1931–35	3	4	5	7	2	4	5	5
1936–40	4	4	11	10	10	8	8	5
1941–45	4	4	11	10	16	15	10	6
1946–50	2	4	12	12	22	21	11	12
1951–55	1	3	16	13	24	21	19	21
1956–60	2	3	14	11	26	25	23	29

The degree of correspondence between the expectation and the reality is less close than that achieved by Beard, but when all the figures are studied it can be seen that, broadly, the pattern of the actual rates is reproduced by the expectation. What, then, is the make-up of this pattern? The basic elements are shown in Table XIII. First, it is possible to obtain a better representation by omitting a secular trend than by including one. Secular influences seem to have operated only in special periods such as 1916–20, with its influenza and war deaths.

Secondly, the changes from each successive generation to the next are less steep than Beard's; thirdly, the age-gradient is more marked than his.

The flatter generation development now disclosed, and the absence of a long-term secular factor, are not inconsistent with an association with smoking, bearing in mind that the *difference* between the smoking of men and women has probably increased less rapidly than the smoking of either. But the experience

TABLE XIII

COMPARISON OF PATTERNS BY FACTORS BY AGE AND YEAR OF BIRTH WITH THOSE OBTAINED BY BEARD

CENTRAL AGE	Beard	Cox	YEAR OF BIRTH	Beard	Cox
37	2	1	1846	1	1
42	5	5	1856	2	3
47	12	9	1866	4	4
52	27	22	1876	**10**	**10**
57	56	50	1886	28	25
62	**100**	**100**	1896	43	30
67	155	211	1906	63	27
72	202	329	1916	69	27
77	260	725			
82	287	1438			

cannot be dissected so neatly and convincingly as that in respect of lung cancer by Beard. In one respect the new analysis presents an interesting and perhaps satisfying feature: the age factors follow an exponential function at least up to age 80—in other words they obey the ' law ' of Gompertz—whereby the ratio of increase in mortality from age to age is constant. This suggests that the data could be a heterogeneous continuation of specific factors, such as the association between smoking and carcinoma of the bronchus.

It is now time to draw the threads together. Marriages are taking place at younger ages than before, and the relative ages of the brides and bridegrooms have adapted themselves to the needs of men to find mates for themselves in a changing demographic situation. A study of fertility shows that some of the patterns established during the first half of the present century could be undergoing some change in the recent uprising in births, although it is not possible to be certain what

will happen in future. An investigation into the severe check to the improvement in man's mortality rates in middle life, in comparison with the continued advance in the longevity of women, reveals that it follows a pattern that could be associated with the excess smoking of men.

While these analyses touch upon important demographic questions of the day, they by no means exhaust the possibilities of research. In particular, we look forward to the publication of the results of the 1961 Census, especially in relation to fertility, when a new picture of the recent rise in the birth rates will be revealed, including an account of the way in which class-differentials have developed.

REFERENCES

1. BEARD, R. E. 1963. Discussion of Actuarial Methods of Mortality Analysis; adaptation to changes in the age and cause pattern. *Proc. Roy. Soc. Bull.* **159**, 56.
2. HENRY, L. 1963. Aspects biologiques de la fécondité. *Proc. Roy. Soc. Bull.* **159**, 81.
3. HILL, Sir AUSTIN BRADFORD and DOLL, R. 1964. Mortality in relation to Smoking: Ten Years' Observations of British Doctors. *Brit. med. J.* i, 1399.

RECENT TRENDS AND PERSPECTIVES IN WORLD POPULATION GROWTH

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NEVER before has so much attention been given, nationally as well as internationally, to population trends and their economic and social implications. The accelerating population growth, particularly in the low income pre-industrial countries, is causing increasing concern, in view of the large populations involved (more than two-thirds of the total world population), the prevailing acute economic and social problems and the widening gap between the levels of living in these areas, and those in the economically advanced countries. Embedded in the present demographic situation are even higher rates of population growth which are bound to be of crucial importance for the economic, social and political conditions under which mankind will live.

The present world population of about 3300 million people is increasing by 70 million a year. At this rate the world population will double in barely thirty-five years. Never before has mankind experienced such rapid population growth. In the Middle Ages the population increase was erratic but as a whole very small—equivalent merely to a doubling of the population over a time span of a thousand years. In the nineteenth century the rate of growth increased slowly, amounting on the average to one-half of one per cent per annum. In the period between the two world wars world population increase was around 1·1 per cent. Soon after the end of the last war it began to climb rapidly and to-day it has almost doubled at 2·1 per cent.

The rates of population growth are, of course, far from being the same in various parts of the world. If we take as a whole the less developed countries mainly in Africa, Asia and Latin America, we find that they have a population

* The views presented in this paper are the author's, and not necessarily those of the United Nations.

increase of more than 50 per cent over that of the economically advanced countries. In Central America and the Caribbean region, population growth is almost 3 per cent a year and the population of Latin America and South East Asia is growing at a rate nearly as high (2·7 per cent). In these regions a number of countries experience at present a rate of increase around 3½ per cent or more, and have to face the fact that their population at the present rate tends to double in twenty years or sooner. In contrast, the European countries have a very modest rate of growth—on the average less than 1 per cent a year.

The rapid increase in world population growth in recent years has been caused mainly by the spectacular decline in mortality in the less developed countries. Important improvements in health conditions and the development and application of efficient methods of prevention and cure of diseases which can be easily applied at a low cost in these countries have, since the Second World War, brought about a revolution in mortality and morbidity. The gains are greatest in infant mortality. In some countries of Asia, Latin America and Africa, the expectation of life at birth has increased on an average by one year or more per annum. Such substantial gains have been made, for example, in Mexico, Mauritius, Ceylon and Japan. As a comparison, it might be mentioned that the mortality decline in the now advanced countries during the demographic transition was far more modest—in Sweden, for example, the life expectancy in the nineteenth century only increased by about two years per decade.

In spite of the recent great gains in mortality in the developing countries, there are many areas where the level is still fairly high. In a number of African countries the death rate is over 30 per thousand, more than three times the rate for many of the advanced countries. In all areas in West and Central Africa, life expectancy at birth is estimated to be as low as between thirty and forty years; in many of these countries one-fourth of all live-born children die before they are one year old. (In an economically advanced country like Sweden, the life expectancy is nearly seventy-five years and only one out of seventy live-born children do not reach the age of one.)

Besides the unprecedented mortality decline, the important feature of the present demographic situation in the world is the general stability in the level of fertility or the absence generally of any decline in the birth rate, particularly in the less developed countries. On the average, the birth rate in these countries is about twice as high as that in the economically advanced regions on the world. Birth rates of above 35 per thousand of the population are, with very few exceptions, found in the regions of Africa, Asia and Latin America. There are, however, no uniform levels of fertility, as there are of mortality; this is particularly true in the less developed countries. In several African areas, birth rates of 55 to 60 or more are found, such as in Guinea, Mali and Niger. For the whole of West Africa, the estimated birth rate is 54 per thousand. At the same time, in some areas of Africa, such as Zanzibar, Mauritania, Gabon and Basutoland, the birth rate is only 40 or slightly less. In all countries in North America and Europe, as well as in the Soviet Union, the birth rate is below 25—in some countries as low as 13 or 14 per thousand.

While there have been important declines and increases in the birth rate in the economically advanced countries in recent decades, the data available for the less developed regions give little indication of any major changes in the high levels of fertility, merely showing fluctuations in the birth rate from year to year. Apart from Japan, where a spectacular fertility decline has taken place since the last war, there are few examples of any major decline in birth rate, although a declining trend seems to be on the way in isolated cases such as Puerto Rico, Taiwan and Malaya. On the other hand, some cases might be cited which indicate a rise in birth rates in recent years, particularly in the Caribbean and Central American regions, although in some of these countries it may be due rather to an improvement in the recorded statistics than to a real increase.

In the absence of any significant declines in fertility in the major regions of the world, the spectacular declines in mortality have resulted in the rapid world population growth we are faced with to-day. The effect upon population growth

has been very pronounced as the improvement in mortality has to a major extent taken place at the younger ages. An increase in life expectancy from the low level of thirty to forty years will tend to raise the rate of population growth by nearly 1 per cent but a similar improvement in life expectancy from fifty to sixty years will tend to increase population growth by only one-half of 1 per cent and from sixty to seventy years by even less.

The potentiality of the upward trend in population growth in recent years has only slowly been recognized—even by demographers. The results of the population censuses held almost everywhere in 1960 or 1961 came as a surprise in many developing countries; in more than sixty of them it was found that the census results were above the estimated population; this was mainly due to an under evaluation of the decline in the death rate in the pre-census period. The total under estimation exceeded 40 million people, the biggest gap being found in India and Pakistan. Another example is the United Nations population projections published in 1958. Three sets of projections (high, medium and low) were prepared. In the few years which have passed, even the high series for the world as a whole has been shown to be too low—falling more than 4 per cent below the estimated actual population at present.

The regions with a high rate of population increase are also those with a large proportion of the population in the younger age groups due to the high level of fertility. In most low-income countries, around 40–45 per cent of the population is at present under fifteen years of age. There are even countries where half of the population is below this age. The large number of children and the heavy dependency burden is one of the most important handicaps to economic and social development of these countries. While the productive age groups in the Scandinavian countries have to support, on an average, only one person for every three of productive age, the ratio between these two population groups is two to three in the low-income countries.

Countries with a rapid population growth will have to spend an increasing amount on food, clothing, housing and other necessities merely to maintain the rising population,

leaving less for development purposes and improvement in the miserably low levels of living. In Asia, the rise in food production since the last war has barely kept pace with population growth, and the output *per caput* does not as yet exceed the pre-war level.

The major importance of the present high rate of population growth is the prospect of continued and even higher rates in the near future. In spite of the recent spectacular gains in mortality in many countries, there are still great improvements to come in the near future. Recent achievements in public health have clearly shown that a vigorous government policy, with assistance from abroad, can have an effective impact. No government in a developing country can avoid, in the long run, the implementation of a vigorous policy in this regard, if it does not succeed in making major improvements in economic conditions. A low level of mortality may therefore be expected to become general before too long.

However, in spite of further declines in mortality in the near future, the rate of world population growth will not continue to accelerate. Mortality decline in most of the developing countries in Asia and Latin America will, to an increasing extent, be in the higher age groups and therefore have a limited impact upon population growth. Furthermore, the mortality decline will tend to slow down as low levels are being approached; it is no doubt more difficult to raise the life expectancy from fifty to sixty years and over than to increase it from thirty to forty years or from forty to fifty years, as long as the living conditions in these countries are low and improvements therein are made at a slow pace.

It is not likely that the high rates of population growth will be checked in any country by a reversal of the mortality trend. Fears that this may happen are sometimes expressed in connection with a general concern about the unfavourable effects of population pressure; but, given the existing means of preventing and curing diseases, such a development would hardly be acceptable in any community for any length of time. In many countries, for various reasons, emigration cannot be expected to play a major role as an outlet for an expanding population as it did in Europe. That leaves us with fertility

as the only factor which can bring about a check in population growth. By reducing fertility (in terms of the gross reproduction rate) to about one-half of the present levels in low-income countries, the rate of population growth will be about 2½ per cent per annum lower, by and large irrespective of the actual levels of mortality and fertility.

A most important problem is how to bring about a change in the age structure in these countries and reduce the dependency burden in order to facilitate the so-called 'take-off' for economic development. The fact is that a decline in mortality will have only a minor influence upon the age structure of the population; if anything, it will tend to increase the proportion of the population in the younger age groups, particularly in countries where the level of mortality is still fairly low. Therefore, future declines in mortality will only help to increase the economic handicaps in the developing countries.

A favourable change in age structure can only be brought about by a decline in fertility. In a population with high fertility (gross reproduction rate at 4) about half of the population will be under fifteen years of age in stable demographic conditions, while only about one-fourth of the population will be in that age group in a country where fertility is low, as in many economically advanced countries to-day (gross reproduction rate at 1·5).

It is worth noting, however, that these effects of a fertility decline upon the rate of population growth and the age structure are only obtained in the long run. In a period of transition the picture is quite different. As an illustration, we may take the Asian countries of Ceylon, Malaya and the Philippines. A decline in fertility beginning now would only have a small impact upon the rate of population growth over many years to come. For ten to fifteen years the decline in fertility would, to a great extent, be counteracted by the expected further decline in mortality. A fertility decline of around 2 per cent a year would only mean that the rate of population growth after a decade would be around one-half of 1 per cent below the level which would have prevailed if fertility had remained unchanged. However, in about fifteen years or more the cumulative effect would be increasingly felt,

as the reproductive age groups would be affected numerically and a rapid decline in population growth would begin.

A similar effect is to be noted with regard to the age structure. A decline in fertility will only provide a major shift between the numerical importance of the active and inactive population groups after fifteen to twenty years. In the three countries mentioned the dependency burden will, in about ten years' time, still be about twice as large as in Japan, where the decline in fertility has been effective over a number of years.

In discussing the prospective trends in world population growth the most crucial and difficult question remains: How soon and how rapidly can a decline in fertility be brought about in the developing countries? Time does not allow me to go into this question, and I shall merely mention a few considerations.

High fertility patterns are not easily changed. They involve basic elements of human behaviour such as cultural traditions, family organization, economic needs, marriage practices, religious convictions and sexual behaviour. Resistance to change is considerable and progress is slow, particularly among predominantly rural people, poorly educated as they are and generally living a life in isolation, often with little hope of improvement. A major obstacle is the difficulty of finding appropriate ways of communicating new ideas and knowledge to the rural masses. Yet another problem is to find suitable means and devices which can be applied in these societies on a large scale, and which are acceptable to the majority of the people. A great deal of research has still to be carried out in these fields.

Although the difficulties standing in the way of a rapid fertility decline are great, there are on the other hand some favourable factors to be mentioned. Voluntary fertility control in various forms is not unknown in the developing countries. Fertility may be high as compared with European standards but it is generally far below what is biologically possible. The very high rates of fertility found in a few exceptional cases, such as in the Cocos Islands and among the American Hutterites, bring this out, as well as the fact that in various developing countries there are major differences in the levels of fertility not explained merely by differences in health conditions.

Studies carried out in many different societies in recent years have clearly shown that there is a widespread and increasing desire for limiting the family size although the norms in most of the developing countries are still very high (about half of the families and in many cases two-thirds or more want at least four children, thus limiting the scope, of course, for any drastic reduction in population growth).

The rising expectations and aspirations in these countries and the social change under way towards modernization and development will also promote the idea of limiting fertility. For one thing, the revolutionary improvement in mortality rates may bring out hopes for a better life, and the people may realize that fewer births are now needed to fulfil the old norms of family size in terms of the number of surviving children. The growing recognition of the problem of population growth among governments and other institutions and agencies at the national and international levels is another favourable factor to be mentioned.

There is no need to paint an exaggerated picture of the implications of the current high rates of population growth and to claim that we are heading for a disaster. Actually some improvements in the economic and social conditions at a moderate rate may be possible in the developing countries even if population growth is not checked soon. However, the important fact to bear in mind is that only a lower rate of population growth through a reduction in fertility will make it possible for these countries to attain some of their economic and social goals. Economic models for developing countries indicate that in a population growing at a rate of 3 per cent where only 10 per cent of the national income is invested there will be no appreciable increase in the income per head unless substantial assistance from abroad is obtained. Only a few Asian countries are able to spend more than 10 per cent of their national income on investment and a rate of population growth around 3 per cent is not uncommon. If the population was only growing at 1 per cent per annum the level of living would rise by 2 per cent annually even at the prevailing low rate of savings.

I have in this paper refrained from giving any concrete

estimates of the future world population. To make population projections is a dangerous business—in the past they have, as Mr Cox illustrated this morning, more often than not proved to be wrong. Furthermore, our knowledge about the actual population size and composition is rather sketchy in some areas of the world—in particular in mainland China. The main outline of future world population trends is, however, fairly clear in general terms. If no fertility decline should take place on a large scale in developing countries, the acceleration in population growth would continue and reach a level around 3 per cent a year before the end of the century. We should have a world population of well over 7000 million and it would tend to double in only twenty-three years. More than four-fifths of the population would be in the low-income countries of Asia, Africa and Latin America. This, however, is an unlikely development. Some fertility decline can no doubt be expected—but even if the decline sets in soon and with considerable force in the majority of the present low-income countries, the world population will at the turn of the century still be around 6000 million people.

THE TREND TOWARDS EARLIER PHYSICAL MATURATION

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DURING the last fifty to a hundred years children have been maturing progressively earlier in Europe, North America, some parts of China, and Japan. The evidence for this statement comes from three sources:

1. Heights and weights of children at each year of age, taken usually in baby clinics or schools. These data are not decisive as evidence, since a tendency for children to become larger at each age could merely mean that adults were becoming proportionately larger also. There is additional evidence, however, that though adults have been getting larger, this is only to a much lesser degree.

2. The curves of rate of growth in height during adolescence of boys and girls followed individually or, failing this, of cross-sectionally studied groups of children, as in (1). At adolescence a spurt in growth occurs, giving a characteristic peak in longitudinal data. The age at which this peak occurs indicates a particular maturity point.

3. The age at menarche, or first menstrual period, in girls. The age of first appearance of pubic hair in boys is a less reliable index of maturity, and one that has been less studied.

I shall deal with each of these three sources of evidence in turn, endeavouring to give some idea of the comparative situation in different countries as to when the trend started and how large it is. I will then discuss the possible reasons for this trend. I shall leave the educational and social implications for you to draw, though I have discussed them elsewhere.[66]

HEIGHTS AND WEIGHTS OF CHILDREN AT EACH YEAR OF AGE 1880—1960

Figure 1 shows the greater height and weight of Swedish schoolchildren in 1938 compared with 1883.[9] I have chosen these data for illustration since they are very extensive (8500

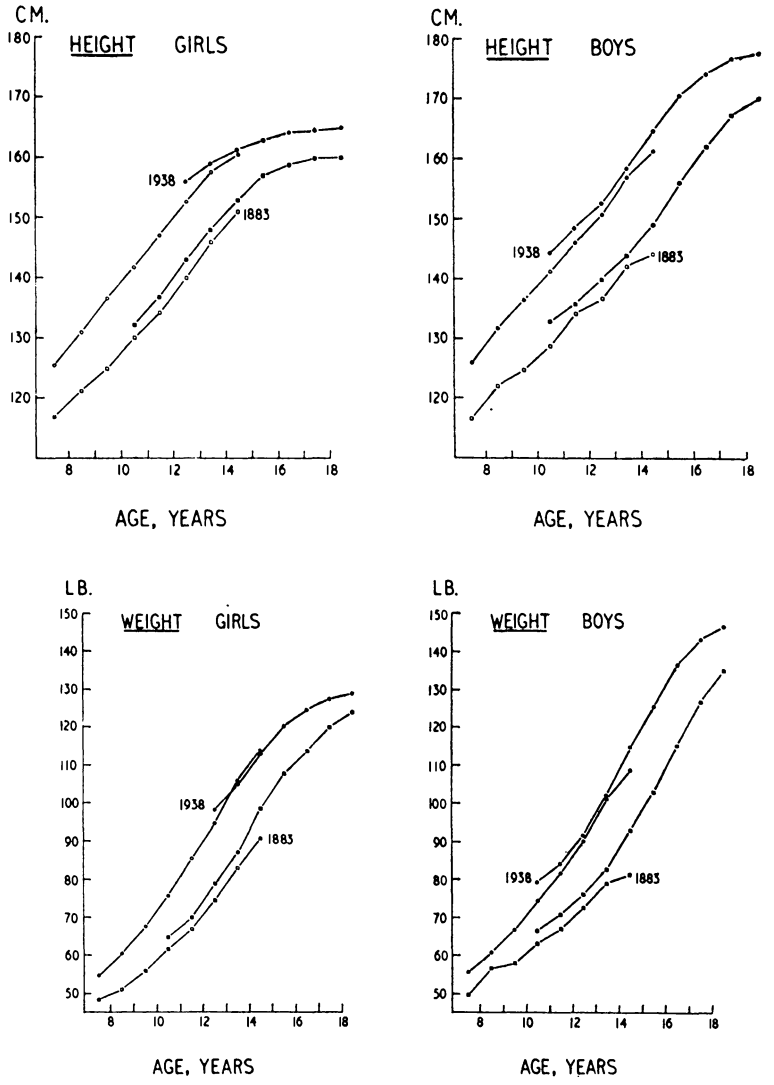

FIGURE 1

SECULAR TREND IN GROWTH OF HEIGHT AND WEIGHT

Height (above) and weight (below) of Swedish girls and boys measured in 1883 and in 1938–39. Elementary schools age 7–14, secondary schools 10–18. Distance curves; cross-sectional. (Data from Broman, Dahlberg and Lichtenstein, 1942, Tables 11–14) (from Tanner [67]).

children measured in 1938, by only two measurers). As in most data, the secondary schoolchildren are slightly larger than the children receiving only elementary education, but these differences are completely dwarfed by the secular trend. The difference 1883–1938 is well established by age seven; the 1938 children are of a size corresponding to about 1½ years' advancement in growth. When height growth ceases it is evident that the secular difference is less than during the growing period; but nevertheless it exists, as can be seen from the height graph for girls, since by eighteen the girls even in 1883 have stopped growing. (They are, however, rather specially selected girls, since they are still in school at eighteen, and represent the best-off part of the population; we shall return to the question of the trend in adult height later.)

Extensive data are available on school age children for Sweden 1883–1938; 1938–50,[1] Norway 1920–55,[64] Finland 1916–56,[65] Germany 1911–58,[34, 35, 20] Poland 1880–1958,[75, 31, 47] Austria 1935–52,[60] and Czechoslovakia 1895–1951.[57] In the United Kingdom we have figures for Glasgow 1906–55 at age nine [15] and 1906–50 at ages five, nine and thirteen [73]; London 1938–59 [63]; and the much earlier, but more scrappy data illustrated in Figure 2. There are data for New Zealand 1943–54,[53] New South Wales 1915–54,[45] Canada 1890–1945,[41, 22] the United States 1880–1960.[39, 40, 42, 43, 30] There are also figures for Japan 1900–52 [29, 20] and Hong Kong 1920–60.[12]

The European and North American data are all in good agreement; from about 1900, or a little earlier, to the present time children in average economic circumstances have increased in height at ages five to seven by 1 to 2 cm each decade and at ages ten to fourteen by 2 to 3 cm each decade. Thus in Glasgow present-day five-year-olds are about 5 cm (or 2 inches) taller than five-year-olds in 1906; nine-year-olds some 3 inches (or 8 cm) taller than in 1906, and eleven-year-olds nearly 4 inches taller.[15] In Iowa nine-year-old girls are also about 8 cm taller now than in 1900.[43] There is an approximately proportional gain in weight, and also in other bodily dimensions; thus shape changes have been non-existent or very small [35, 30]; the change is in size and not proportions.

The data on pre-school-age children are very scanty; such as they are they indicate that the trend in Europe and America starts directly after birth and, relative to absolute size, may even be greater from two to five than subsequently.[34] The last large series of London data however show most of the change

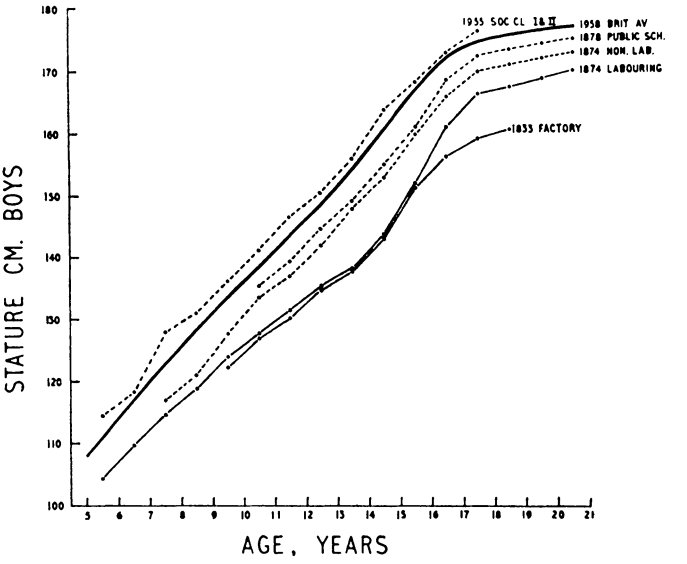

FIGURE 2

HEIGHT OF ENGLISH BOYS 1833–1958 TO SHOW SECULAR TREND

1833 factory boys, from Cowell, quoted in Bowditch (1877); 1874 labouring and non-labouring classes, 1878 'Public' school (upper classes) from Roberts (1874, 1876, 1878), Fergus and Rodwell (1874), Galton (1874); 1955 social class I and II from Birmingham Survey (Clements, unpublished); 1958 British average from Tanner.[67]

between 1954 and 1959 concentrated between ages eight and fourteen with little difference at five, six or seven.[63] Nearly all data agree in indicating that the trend is still continuing, and has in most areas been more marked in the last twenty years than in the preceding forty. In Japan it seems that the gain before age six has been less than in Europe, though the school-age gain is nearly up to European values.

The trend has been a steady one, though slowed from time to time by the famines of war and, to a less extent, of economic crisis. Evidently it started, at least in Britain, some considerable time before 1880, because Roberts, writing in 1876, said that " a factory child of the present day at the age of nine years weighs as much as one of 10 years did in 1833 . . . each age has gained one year in forty years." [67]

The trend in adult height

If adult height had remained constant all the time, Roberts's implied interpretation of the childhood increase would be correct; all the gain in children's height would be due to earlier maturing. The Glasgow five-year-old of 1950 would be (not look like, but *be*) the Glasgow six-year-old of 1900, the eleven-year-old of 1950 the 12½-year-old of 1900. In fact, however, there has been also an increase in adult height, though this is much less than the increase in height of children. Thus the secular trend in children's size is due chiefly to acceleration of the maturing process, and in a smaller degree to the ultimately reached adult size being greater.

There has been some dispute about this adult height increase. This was because maximal height in men, some fifty years ago was reached only at the age of around twenty-six[28, 49] whereas now in Europe and America it is reached at eighteen to nineteen. Consequently we cannot estimate the adult secular trend by comparing the heights of twenty-year-olds in 1900 and 1960; one must either compare twenty-six-year-olds in 1900 with twenty-year-olds in 1960, or at least make an allowance for the 20–26 year gain fifty years ago. Fortunately there are a number of sets of data which permit us to do this.

The classical series is that from Norway, reported at length by Kiil in 1939.[28] Not only have there been height measurements extending back to 1741 of men aged seventeen to twenty-six, but even longitudinal records from the eighteenth and nineteenth century of individuals' heights from fifteen to thirty years old. This superb archive makes it clear that final adult height increased little (i.e. less than 1 cm) in Norway from 1760

to about 1830; from 1830 to 1875 a gain of around 1½ cm, or 0·3 cm/decade, took place and from 1875 to 1935 a gain of 4 cm, or 0·6 cm/decade. In Holland also records were kept of the height of men called up at nineteen or twenty for the civil militia and remeasured at age twenty-five. This series extends from 1819 to 1902, and has been analyzed by Oppers.[55] The fully adult or twenty-five-year-old height actually dropped slightly from 1820 to 1860, but in the hundred years since 1860 is estimated to have risen approximately 9 cm, or 0·9 cm/decade. Unfortunately the British data are much less satisfactory and led Morant,[49] in a paper quoted much and uncritically, to suppose that no adult secular trend had occurred. As far as Britain itself is concerned, the safest conclusion would be that the data permit of no final statement; but for most Western European countries the data are in excellent agreement and point to an adult secular trend of between 0·6 and 0·8 cm/decade from about 1870 to the present day.[23, 5, 14, 71, 17, 21, 8, 32, 27] In the United States the trend has been about 0·3 cm/decade for whites and 0·7 cm/decade for negroes during the years 1940 to 1960, but was probably nearer 0·7 cm/decade for whites from 1917 to 1940.[25] It is of much interest that Dutch, Norwegian and Danish data (the last is quoted in Lenz, 1959) all seem to show little gain until about 1860, and an accelerated gain from around 1880 till approximately the present day. We will discuss the possible reasons for the trend below: suffice it here to say that Sauvy [62] estimates that the real wages of a labourer in France began to rise about 1850, and that historians, I believe, say that in England the labourer's diet began to improve about 1815 but progressed only very slowly till about 1850, after which it got rapidly better.[37] Mortality in England began declining around 1840 and the decline (except in neo-natal mortality) became marked after 1860.[37] The stature of persons living in medieval village communities may well have been higher than that of labourers in the sixteenth and seventeenth centuries. When the population expansion began (around 1750 in England) it seems likely that the social conditions of most manual workers grew worse for at least a hundred years.

The secular trend evidently still continues. In Figure 3

the average heights at each age from seventeen to twenty-two are shown for all male university students in France, divided according to year of birth. The trend in mature height is 0·7 cm/decade from 1941 to 1951.

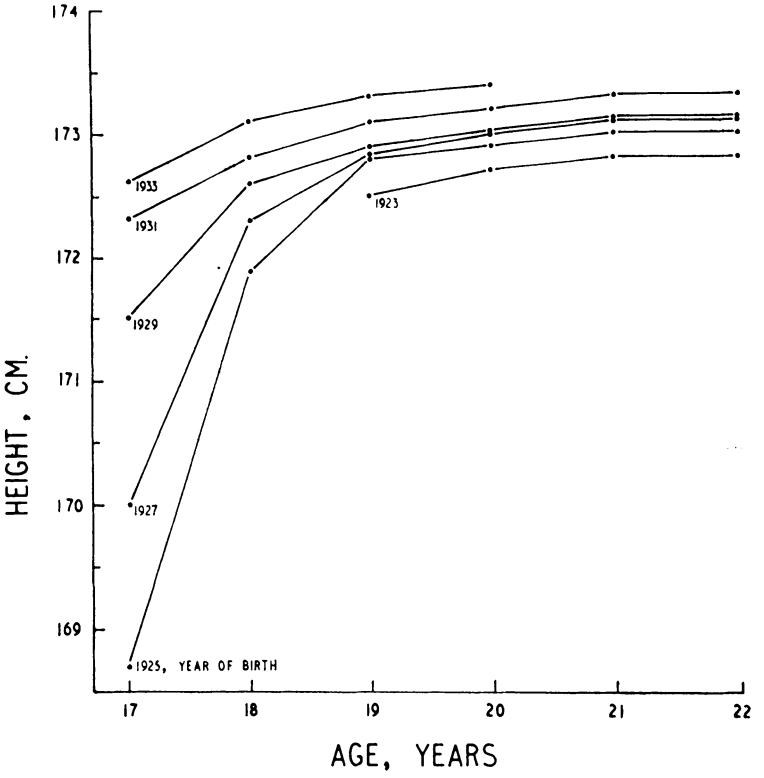

FIGURE 3

SECULAR TREND IN HEIGHT OF FRENCH UNIVERSITY STUDENTS DURING 1940s

Average of all men students, mixed longitudinal data, according to year of birth. Note greater maximal height in more recently born and earlier approach to maximum. Redrawn from Aubenque (1957) (from Tanner [67]).

THE AGE OF OCCURRENCE OF PEAK HEIGHT VELOCITY AT ADOLESCENCE

The curve of rate of growth in height in man has a very characteristic shape. The velocity is high at birth and falls

progressively until adolescence. At this time a marked increase occurs, known as the ' adolescent growth spurt '. The velocity reaches a well-defined peak, then falls progressively to zero. In England at present peak height velocity is reached on average at 12·0 years in girls and at 14·0 years in boys. The first menstrual period occurs on average one year after peak height velocity has been reached. The peak is shown

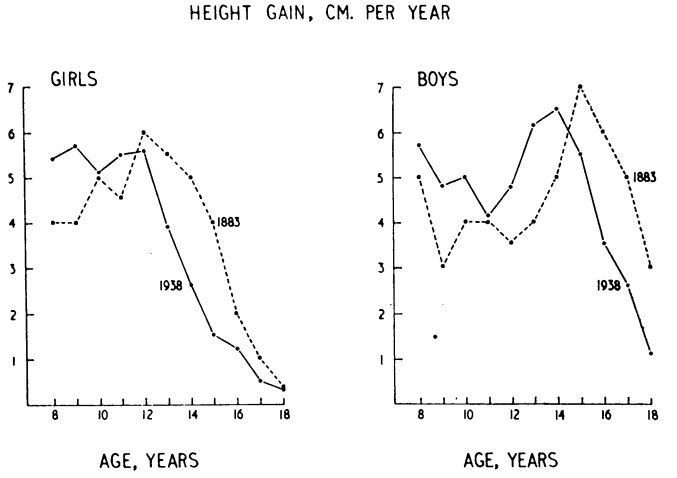

FIGURE 4

SECULAR TREND IN TIME OF ADOLESCENT SPURT

Velocity curves of height for Swedish girls and boys measured in 1883 and in 1938–39. Cross-sectional data. (Data from Broman, Dahlberg and Lichtenstein, 1942, Tables 11–12) (from Tanner [67]).

considerably better in individual longitudinal data than in mean velocity curves derived from cross-sectional data by subtracting the mean at age twelve from the mean at age thirteen and so on. The reason for this is the damping effect on the mean introduced by individuals having their peaks at very different ages.[67] However, some sets of cross-sectional data do show up the peak well enough for our purpose. In Figure 4 the boys' velocity curves observed from Swedish data of Figure 1 are shown. Clearly the peak velocity was reached at least a year earlier in 1938 than in 1883.

Additional data comes from Kiil's longitudinal series. Instead of using peak velocity he considers the figures for the standard deviation of height in the population at each age; because of the variation between individuals in age of peaking the variance of height increases during the adolescent spurt, and reaches a maximum at the age at which the peak velocity occurs. Kiil found that the standard deviation was maximal at seventeen years in young men measured in Norway in 1825–37, compared with fourteen years in schoolboys of the 1930s. His figures are not entirely conclusive here since his longitudinal data only commenced at the age of sixteen. However,

FIGURE 5 (*Opposite*)

SECULAR TREND IN AGE AT MENARCHE 1830–1960

Sources of data as follows:

Finland: 1862–1915, hospital patients Helsinki, from Malmio (1919) and age at interrogation 17 to 27 only, from Simell (1952).

Sweden: 1886–1915 hospital patients, Lund and Stockholm, from Essen-Moller (quoted in Lenner, 1944), Lundh (1925), Samuelson (1942) and Lenner (1944) (hospital data of last two pooled for value at 1915); 1950 schoolchildren, estimated from data of Romanus (1952).

Norway: 1844–81, from Backman (1948); 1907 Oslo hospital patients, from Skerlj (1939); 1928–52 Oslo schoolchildren, data of Schiotz (1930), and Kiil (1953) fitted by probits.

Germany: 1860–1928 hospital patients various towns, successively from Schlichting (1880), Heyn (1920) and Schaeffer (1908) pooled, Risopoulos (1936), Scheibner (1938); 1937, schoolchildren S.W. Germany, probits fitted to data of Ley (1938). See also values reported in Backman (1948) and Wallau (1952).

Great Britain: 1948–60 schoolchildren, probits, successively S. England from Wilson and Sutherland (1950), Edinburgh from Provis and Ellis (1955), Bristol from Wofinden and Smallwood (1958), London from Scott (1961).

U.S.A.: 1905–40 University of South Carolina entrants, from Mills (1950), 1960 estimated, see text.

Denmark: 1950, Copenhagen schoolchildren, probits, from Bojlen, Rasch and Weiss-Bentzon (1954).

Values are plotted at year in which the average menarche took place, i.e. in ' recollected age ' data if average menarche of 40-year-olds interrogated in 1900 was 15 years, this is plotted at 1875. This places old data on same age scale as modern probit data. Where age of interrogation is not recorded an estimated amount has been subtracted according to nature of population studied (primiparae, etc.). Grouping errors have been corrected where necessary (i.e. ' 13-year-olds ' centred at 13·5 years, not 13, as in some of older literature) (from Tanner [67]).

his conclusion that in Norway puberty was brought forward by about three years between 1830 and 1930 is entirely in accord with the data on menarche.

AGE AT MENARCHE

The trend towards earlier maturing during the last hundred years is perhaps best shown by statistics on the age of menarche. A selection of the best available data is illustrated in Figure 5. The sources are listed in the legend of the Figure.

Naturally these data are not all equally reliable. Age at menarche is known, from longitudinal studies, to be distributed in Gaussian fashion. As a consequence, the statistical technique of probits or logits can be used to estimate the median age of menarche from cross-sectional data. This is a very fortunate circumstance, since all one has to do is to select a proper sample—of the schools in a certain area, or of all girls belonging to a certain occupational group for example—and then simply ask every girl whether or not she has experienced her first period. Ideally all girls aged nine to seventeen should be interrogated, but very little information is lost by restricting

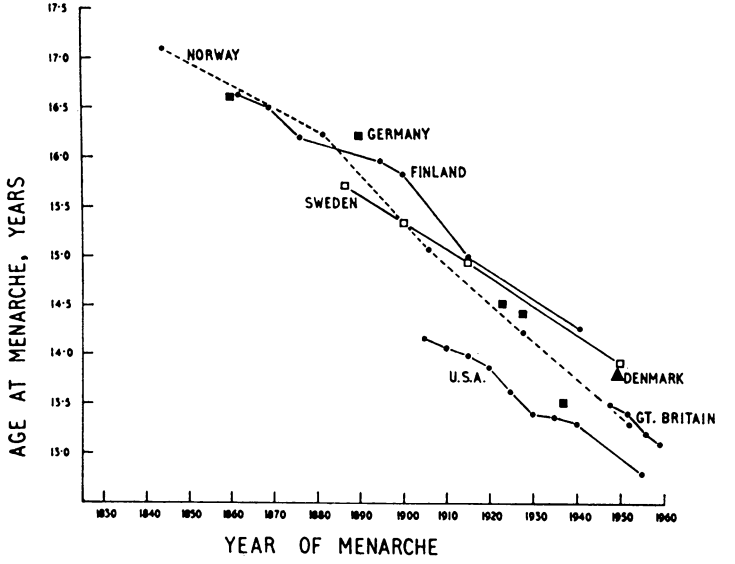

FIGURE 5

the ages to 10·0 to 15·9. A plot of per cent menstruating against age (ranging from say 10 per cent at 10·0 to 90 per cent at 15·5) gives a sigmoid curve, which repeated experience has shown to be very well fitted by either probits or logits. All modern studies are carried out in this way, and some older data can be subjected to this method also. A procedure that may *not* give a valid estimate of the mean in cross-sectional data, but has been much used in the past, is that of inquiring of all children in a school or college what age they were when they first menstruated. Apart from the difficulty of exact recollection on the part of those who had their menarche several years before, a more important bias is introduced if there remain any girls who have not yet menstruated. Such girls will have high values for menarcheal age, and if these values are omitted, the mean age obtained is spuriously low. Either only girls above 17·0 must be interrogated or else the rather complex statistics of the truncated Normal distribution must be used to allow for the absent tail (an example is given in Bojlen, Rasch and Weis-Bentzon [6]).

The older data suffer from disadvantages both of sampling and technique. Most of the pre-1920 data concern hospital patients, who may be a biased sample of the population at large. Worse, these data concern entirely the recollected age of menarche, that is the age at which menarche was remembered to have occurred by women interrogated five, ten or even twenty years after the event. Other rather obvious sources of error that need to be avoided are mentioned in the legend to Figure 5.

Despite these defects the main conclusion implicit in Figure 5 is perfectly clear. The data are impressively consistent: and additional lines for girls in Budapest from 1860 to 1960,[18] in Czechoslovakia (Prokopec, unpublished) and Poland (Wolanski, unpublished) are almost exactly parallel to the ones in the graph. Evidently menarche in Europe has been getting earlier during the last hundred years by between three and four months per decade. Other data, besides those illustrated, are given in Backman [5] for France, Denmark and Holland, and in Mills [48] and Michelson [46] for America; they agree well with this conclusion. The trend in height and weight at the

age of puberty is in good agreement also, children of twelve thirty or forty years ago having the size of children of eleven at present.

The best figures for menarche in Great Britain, for example, are: Manchester working women, about 1820, 15·7 years; 'educated ladies' of the same time and place, 14·6 years [73]; University College Hospital, London, obstetric patients about 1830 and 1855, 15·4 and 15·5 respectively; [52, 59] London 'middle classes' about 1880, 15·0[19] and Edinburgh Royal Infirmary gynaecologic patients about 1905, 15·0.[26] The fall is of the same order as in Scandinavia, but in the nineteenth century the menarche was not quite so delayed as it was in Norway and Finland. The 1820 figure for 'educated ladies' is particularly interesting, as these were upper class women corresponding to those with an average age of menarche of about 12·9 nowadays. This corresponds to a rate of fall of about half the general figure given above.

The present-day ages of menarche in various groups are listed in Table I. These data are selected as all being modern and highly reliable, and obtained, with a few exceptions, by probit or logit analysis. It appears that Chinese girls in Hong Kong, if belonging to well-off families, have the earliest recorded menarche; even the very poor Chinese girls still have menarche as early as most much better-off Europeans. East Europeans seem to mature somewhat earlier than West Europeans, especially when economic circumstances are matched. Comfortably-off Americans are slightly ahead of West Europeans. Well-off Africans are not much later than Europeans, though badly-off Africans (the South African Bantu) certainly are. No group is currently known in which menarche occurs anything like as late as in Europe a century ago.

There is at present little sign that the trend shown in Figure 5 has stopped, or that girls are now maturing at something like the earliest possible age. It seems that these lines may be extrapolated onwards for at least another decade or two. Extrapolation backwards, however, is clearly an impossibility: if the trend we see between 1840 and 1960 had been going on equally before 1840, menarche in medieval times would have occurred, absurdly, after the age of thirty. There is little real

information available for dates before 1800, but Quarinonius,[58] writing of Austria in 1610 says:

The peasant girls in this landschaft in general menstruate much later than the daughters of the townsfolk or the aristocracy, and seldom before their seventeenth, eighteenth or even twentieth year. For this reason they

TABLE I

AGE OF MENARCHE IN RECENT YEARS

All estimates by probits or logits unless indicated

COUNTRY	YEAR	MEAN AGE AND S.E.	AUTHOR
Norway, Oslo	1952	13·4	Kiil, 1953
Sweden, all	1951	13·9 * approx.	Romanus, 1952
Denmark, Copenhagen	1950	13·8±0·3	Bojlén *et al*, 1954
Copenhagen	1963	13·1±0·12	Andersen, unpubl.
Holland, S.	1956	13·7±0·06	Rusbach *et al.*, 1961
Hungary, Budapest	1959	12·8±0·08	Thoma, 1960
Szeged	1961	13·0	Farkas, 1962
all	1959	13·2±0·02	Bottyán *et al.*, 1963
England, London	1959	13·1±0·02	Scott, 1961
Bristol	1956	13·2±0·02	Wofinden *et al.*, 1958
Scotland, Edinburgh	1952	13·4	Provis *et al.*, 1955
U.S.A., Denver (well-off)	1955	12·8 †	Deming, 1958
California	1950	12·8±0·12	Nicholson *et al.*, 1953
Chinese, Hong Kong	1962		Lee *et al.*, 1963
rich		12·5±0·18	
average		12·8±0·20	
poor		13·3±0·19	
Burma and Assam town, well-nourished	1957	13·2±0·08	Foll, 1961
Ceylon, Colombo	1950	12·8±0·07	Wilson *et al.*, 1950, 1953
rural		14·4±0·16	
Nigeria, Ibo (well-off)	1960	14·1±0·16	Tanner *et al.*, 1962
East Africa, Buganda, Kampala (well-off)	1960	13·4±0·16	Burgess *et al.*, 1964
South Africa Bantu Transkai reserve	1958		Burrell *et al.*, 1961
not poor		15·0±0·03	
poor		15·4±0·04	

* Estimate only.

† Longitudinal data.

also live much longer than the townsfolk and aristocratic children and do not become old so early. The townsfolk have usually borne several children before the peasant girls have yet menstruated. The cause seems to be that the inhabitants of the town consume more fat food and drink and so their bodies become soft, weak and fat and come early to menstruation in the same way as a tree which one waters too early produces earlier but less well-formed fruit than another.

Passing by Quarinonius's ruro-humoral prejudices, we may conclude that in the towns menarche was expected around fourteen or fifteen. The earlier of these figures agrees with the inference we may draw for Shakespeare's England:

Romeo and Juliet, I, ii.

Capulet (of Juliet): She hath not seen the change of fourteen years . . .
Paris: Younger than she are happy mothers made.

Ibid., I, iii.

Lady Capulet (to Juliet):

> . . . younger than you,
> Here in Verona, ladies of esteem
> Are made already mothers: by my count
> I was your mother much upon these years
> That you are now a maid.

The Winter's Tale, II, i.

Antigonus:

> I have three daughters; the eldest is eleven . . .
> If this prove true, they'll pay for't; by mine honour,
> I'll geld 'em all; fourteen they shall not see,
> To bring false generations.

Buffon writing in 1748 says the age of puberty depends on climate and nutrition, and in southern Europe and in the towns (presumably of France and the neighbouring countries) is twelve in girls and fourteen in boys; in the northern countries of Europe and in the villages it is fourteen in girls and sixteen in boys. Very likely Buffon is referring to the growth of the pubic hair, and if so, then menarche might be placed as $1\frac{1}{2}$–2 years later. If so we would again have a figure of

about fourteen for girls in north European towns such as London.

It seems likely then, that in the towns of Europe the rate of maturation was slowed down during the late eighteenth and early nineteenth centuries [5]; in the villages of the less agriculturally rich countries the rate had always been substantially slower than in England. From the Shakespearean figure of fourteen we arrive, about 1820, at a menarcheal age of 14½ for the educated upper classes and 15½ for the less well-off townspeople. By 1960 the figure had decreased to 13·0 for both groups.

CAUSES OF THE TREND

In discussing possible causes of the trend towards earlier maturity, we must clearly distinguish between the trend towards greater height and weight in children and the lesser trend seen in adults. It is probable that various environmental changes are chiefly responsible for the change in menarcheal age and for that portion of the greater size in children which reflects earlier maturation; the trend in adult height, on the other hand, seems to be due at least as much to genetical as to environmental factors.

Of the environmental factors better nutrition is of course the most obvious. In acute periods of starvation growth is certainly delayed and puberty temporarily postponed. When such an acute starvation ends the child accelerates to above his normal growth-rate, in a ' catch-up ' towards his normal growth curve.[56] He may or may not reach it again, depending on the severity and duration of the malnutrition. In chronic malnutrition it is fairly certain that not only a great delay in maturation can be caused but also a stunting of final adult height (see below).

Trend of menarche

If better nutrition is the major cause, then one might have expected that the trend both towards earlier puberty and greater size in childhood would have been less in the better-off children than in the poor, on the grounds that in

most industrialized countries the circumstances of the poor have altered more than those of the rich during the last hundred years. The trend of menarcheal age in England has indeed been somewhat less for the well-off than the poor, but the differential is not so much for height or weight, and the menarche differential may simply be because both rich and poor are now beginning to reach the lower, genetically controlled, threshold. The latest English [16] and Scottish [54] data show no significant differences in age of menarche between girls whose fathers are in different occupational groups which reflect, at least approximately, differences in income and the comfort of the home. In Copenhagen in the 1950s there was a difference of two months in menarche between the daughters of members of the managerial and professional classes and those of unskilled workmen.[6] This is only a very modest difference. Menarche occurred in the 1940s some eight months earlier in well-off negroes in New York than in negroes in the Southern States [46] and some six months earlier in well-off Indians in Durban than in poorly-off ones.[24] The Hong Kong data already quoted show a difference of nine months between rich and poor, presumably because the poor are clearly much worse off than the poor in England or Denmark. Studies of identical twins, non-identical girl twins and sisters clearly show that age at menarche is controlled by genetical factors when the environment is good (see Tanner,[67] p. 113). Hence we must suppose that the genetical threshold for Chinese is below that for English, and perhaps that the threshold for East Europeans is below that for West Europeans, unless some unidentified climatic difference between the groups is responsible.

In Hungary in 1960 an extensive study showed that girls in the towns had menarche a few months earlier than those in the country [7]; and in Poland also Warsaw boys reached puberty a little earlier than rural boys.[10] In Szeged industrial workers' daughters matured two months earlier than the daughters of intellectual or white-collar workers; these latter, however, included only few professional people and were estimated to spend less on food than the manual workers. The latest menarche (13·4 to 13·5) occurred in small towns

and villages near the Austrian border; the authors argue against nutritional or climatic differences as an explanation of this and suppose rather that it represents a genetical cline in the population, linked with dark pigmentation. Girls educated in co-educational schools showed no difference from girls educated in single sex schools, a result also obtained in Sweden by Romanus (1952)[67]. In Czechoslovakia [72] a study of the daughters of farmers living at different altitudes in North West Slovakia showed that those at higher altitudes had a significantly later menarche; it was 14·9 years at the highest altitude and 14·2 at the lowest. The authors point out that, in general, the higher the altitude the worse the economic and nutritional situation and the greater the caloric output by the girls in travelling to and from school. They incline to think that these factors may be of more importance than altitude *per se*, though this cannot be ruled out as a contributory cause. In similar mountainous country in the Carinthian province of Austria, Wurst [76] found an exactly similar effect on height and weight of children and he also attributes this more to nutritional and caloric deficit than to altitude itself. Perhaps one of the most convincing arguments for nutritional causes is the example of the Lapps, who had practically the same average age of menarche, 16½ years, from 1870 to 1930, while maintaining intact their pastoral nomadic way of life. During the same period the neighbouring Norwegians, being settled farmers, lowered their age of menarche by nearly two years.[28]

Hot climates used to be cited in the older literature (e.g. Skerlj, 1939) as a potent cause of early menarche. But the evidence for this seems chiefly anecdotal, and nobody nowadays supposes climate exerts more than a very minor influence, if any at all. Little more can be said until equally well-nourished groups are available living under greatly different climatic conditions. At present Eskimos and Nigerians have the same menarcheal age, but their diets are perhaps too dissimilar for a climatic difference to be distinguished. The mean world temperature has been rising since the 1850s (until, it seems, about 1940 when the trend began to reverse); the polar ice caps have been melting and the glaciers of Norway

and the Alps retreating. But on present evidence it seems unlikely that this general warming-up process has contributed significantly towards the earlier menarche of girls.

The one thing that all authors find significantly related to age at menarche is the number of children in the family. The larger the number, the later the menarche and the less the height and weight at all ages, both of the earlier- and later-born children. In the Czechoslovakian data [72] the menarche for children with 0 or 1 sib was 14·3; for those with 6 or more sibs 14·6. In England the same relationship holds (Douglas, unpublished, Scott [63]).

The most obvious explanation of this sibling number effect would be nutritional; the more mouths to feed and children to bother about, the less well the feeding, and perhaps the general care, may be done. An alternative or supplementary explanation might be that children with more sibs get more diseases.

The effect of childhood disease on growth is, however, uncertain. A severe disease certainly retards growth, and retards also the maturation process. But when the disease ends, a catch-up occurs just as it does at the end of a period of malnutrition.[56] Some authors (e.g. Acheson [2]; McCance [36]) hold that disease always or usually causes a greater slowing down in growth than in skeletal maturation. If this were so, then the final height reached would be lessened, since the epiphyses would unite at a lessened height. Acheson puts forward a lessening of disease, therefore, as an explanation for the trend in *adult* height. However the evidence for a differential effect of disease on growth and maturation is very poor indeed at present (see full discussion in Tanner,[67] p. 128): most data agree in showing both processes slowed down equally. Disease has also been invoked as an explanation for a slowing down of maturation and a diminution of *childhood* height, but the evidence here is even scantier. Most childhood diseases are in fact totally without effect on the growth of well-nourished children (see evidence quoted by Tanner,[67] p. 130, also Meredith and Knott [44]). But persistent chronic disease operating upon malnourished children may be another matter; possibly the removal of this could have helped create the childhood secular trend. Even here, however, the evidence is

largely negative; suppression of malaria in heavily parasitised populations has not noticeably increased rate or amount of growth (Tanner,[67] p. 134). Until we have much more evidence we can hardly lay the secular trend, either of children or of adults, at the door of lessened infection.

Trend of children's size

Everything said about the trend of menarche applies equally to the trend of childhood height and weight. One would expect better-off children to have gained less than the poor, but most data show remarkably little difference between the gains of these groups.[39, 47, 15] Schoolboys at Marlborough College, consisting mostly of sons of parents in the professional class, averaged 166·2 cm at 16·5 years in 1873 (Galton, 1874) and 177·0 cm seventy years later (Boyne, 1960). This increase of 1·5 cm/decade represents at least two-thirds of the general increase in Britain. One is reminded of the trends in infant mortality and in social class differences in adult diseases. Though since 1911 infant mortality has greatly decreased in all social classes, the gap between the classes has not narrowed; and while the overall incidence of various adult diseases has risen or fallen, the social class affinities have changed scarcely at all.[51, 50] No convincing reason for the presence of these class differentials has yet been offered. But movements between one social class and another from generation to generation are very frequent in Britain; so that these differentials could perhaps be characteristic more of the sort of person who reaches a particular social class than of the circumstances in which, as a member of that class, he lives.

The family size effect is shown in height and weight just as it is in menarche. But whereas nowadays in Britain it seems that menarche is little if at all linked with social class, height and weight remain persistently associated with it.[16] This is curious, as for many years we have always considered advancement in height and weight to be very closely linked with advancement towards menarche. It seems that the link between social class and body size persists into adulthood.[13, 69] Perhaps it may not be explained entirely on nutritional grounds.

In short, then, we must admit that we are uncertain why the trend towards earlier maturation has occurred. Probably better nutrition is the factor chiefly responsible, but there are certain facts, mentioned above, which at least make us hesitate before accepting the explanation too readily. It seems likely to be more than a matter of simple calories; and it seems likely to be due to an influence which starts early in life. The best guess might be that better infant feeding, and particularly giving infants more protein early in life, is more responsible than any other aspect of nutrition.

The adult trend

The adult trend may well have another explanation in whole or part. Undoubtedly in circumstances where severe chronic malnutrition is frequent the final adult height is affected; Tobias [70] for example has shown that the South African Bushmen have been getting larger as they settle more in agricultural communities. But the European trend may have a genetical explanation, first offered by Dahlberg.[9]

Suppose that some degree of dominance occurs in the genes governing human stature; that is that on average the offspring of a tall and a short parent lies not exactly halfway between their heights but a little closer to the tall parent. The person with many heterozygotes amongst the genes influencing height would be slightly taller, therefore, than the average of persons with many homozygotes. If this is so then the increase of heterozygotes caused by the breaking down of genetical isolates, that is, of the tendency for marriages to be contracted between members of the same village community, would cause an increase in height. It has been shown in several West European communities that outbreeding has in fact increased at a fairly steady rate ever since the introduction of the bicycle.

There is also some direct, though not perhaps completely conclusive, evidence that outbreeding does increase stature in man. Hulse in 1957 found that grown-up sons of parents who came from different Swiss villages averaged 2 cm taller than sons of parents both of whom came from the same village. Similarly Mange [38] has shown that amongst the Hutterite

religious isolate in North America, persons whose parents represented the degree of inbreeding of first cousins averaged about 3·5 cm shorter than persons whose parents were unrelated. Kherumian and Schreider [27] showed that the adult height in different French regions was significantly and inversely correlated with the degree of inbreeding in the region. Thus there seems to be rather solid evidence that genetical causes may explain at least a part of the trend in adult height; whether it can explain it all is more doubtful, since the change towards outbreeding seems unlikely, on the figures given above, to be responsible for an increase of more than 2 cm per generation. Natural selection, on the assumption that tall people have more marriageable offspring than short ones, operates too slowly to account for the trend; and in any case, our scanty evidence on the subject contradicts that assumption.

IMPLICATION OF THE TREND

It is unnecessary to detail the implications of the trend toward earlier maturation. The chief ones lie in the field of education and social behaviour.[65] The trend may perhaps contribute to the earlier age of marriage and the increased fertility rate, in that so far as we know the menopause has become later rather than earlier, thus increasing the total reproductive span, or, from a demographer's point of view, the woman's time at risk.

DISCUSSION

DR J. M. TANNER, asked whether the reproductive phase of life was extended solely by the earlier age of menarche or was also affected by a change in the age of menopause said that there was much less data on trends in menopausal age. First of all it was not such a definite event. When you asked the relevant question, you got a number of ' don't knows ', as in the national opinion polls. This made the whole thing technically more difficult. Very few inquiries had been made in this country, but the indications were that the menopause had been pushed back and that the average menopausal age at the present time might be nearly fifty.

Asked about the truth of the popular beliefs that the small height of people in the north of England was due to poverty resulting from the Industrial Revolution and the small height of French people was due to the fact that Napoleon had destroyed the manhood of France; and that the intake in England for the Boer War indicated the slow downward growth of English people, Dr Tanner replied that the actual number of soldiers killed off in the Boer War, and perhaps to a lesser extent in the Napoleonic Wars, was not very great as a selective factor when considered in relation to the whole population. The statement that the small stature of people in the industrialized parts of this country was due to poverty had been denied by some anthropologists, who said that final adult height was purely genetically controlled and that eventually children under poor conditions, after a prolonged period, reached their genetically controlled height. Dr Tanner did not think the data bore this out; they only reached somewhat below their height potential. There might be a genetic background to the increase in adult height; since if heterosis occurred in human stature—and there was some evidence that it did—then the increased outbreeding which had universally followed the introduction of the bicycle would have given rise to increased stature.

REFERENCES

1. ABRAMSON, E. and ERNEST, E. 1954. Height and weight of schoolboys at a Stockholm secondary school, 1950, and a comparison with some earlier investigations. *Acta Paediat.* (*Uppsala*) **43**, 235.
2. ACHESON, R. M. 1954. A method of assessing skeletal maturity from radiographs: a report from the Oxford Child Health Survey. *J. Anat.* (*Lond.*) **88**, 498.
3. ACHESON, R. M. 1960. Effects of nutrition and disease on human growth. In *Human Growth*, ed. J. M. Tanner. *Symp. Soc. Study Hum. Biol.* **3**, 73.
4. ACHESON, R. M. and FOWLER, G. B. 1964. Sex, socio-economic status and secular increase in stature. *Brit. J. prev. soc. Med.* **18**, 25–34.
5. BACKMAN, G. 1938. Wachstumsdauer und Lebenslänge beim Menschen. *Kungl. Fysiograf. Sallskap. Lund Förhandl.* **8**, No. 10.

6. BOJLEN, K. W., RASCH, G. and WEIS-BENTZON, M. 1954. The age incidence of the menarche in Copenhagen. *Acta. obstet. gynec. scand.* **33**, 405.
7. BOTTYÁN, O., DEZSÖ, GY., EIBEN, O., FARKAS, GY., RAJKAI, T., THOMA, A. and VÉLI, GY. 1963. Age at menarche in Hungarian girls. *Ann. Hist.-Nat. Mus. Nat. Hung. Pars Anthropol.* **55**, 561.
8. BOYNE, A. W. and LEITCH, I. 1954. Secular change in the height of British Adults. *Nutr. Abstr. Rev.* **24**, 255.
9. BROMAN, B., DAHLBERG, G. and LICHTENSTEIN, A. 1942. Height and weight during growth. *Acta paediat. (Uppsala)* **30**, 1.
10. BRZEZIŃSKI, Z. 1959. Wysokosc i waga ciala chlopca w okresie dojrzewania. *Roczn. Pzh.* **10**, 447.
11. BURGESS, A. P. and BURGESS, H. J. L. 1964. The growth pattern of East African schoolgirls. *Hum. Biol.* **36**, 177.
12. CHANG, K. S. F., LEE, M. M. C., LOW, W. D. and KVAN, E. 1964. Height and weight of southern Chinese children. *Amer. J. phys. Anthropol.* N.S. **21**, 497
13. CLEMENTS, E. M. B. and PICKETT, K. G. 1957. Stature and weight of men from England and Wales in 1941. *Brit. J. prev. soc. Med.* **11**, 51.
14. COSTANZO, A. 1948. La statura degli italiani ventenni natti dal 1854 al 1920. *Annali di Statistica*, Series 8, **2**, 59.
15. CRAIG, J. O. 1963. The heights of Glasgow boys: secular and social influences. *Hum. Biol.* **35**, 524.
16. DOUGLAS, J. W. B. 1964. *The Home and the School.* London. MacGibbon and Kee.
17. DURNIN, J. V. G. A. and WEIR, J. B. de V. 1952. Statures of a group of University students and of their parents. *Brit. med. J.* i, 1006.
18. FARKAS, GY. 1962. Az elsö havi veízés (menarche) ideje csongrad megyei leányoknál. *Anthropol. Kozlemenyek* **6**, 83.
19. GILES, A. E. 1901. The factors which lead to variations in the age of puberty and the clinical characters of menstruation. *Med. Chron.* **34**. (1 of 4th series) 161; 254.
20. HAGEN, W., PASCHLAU, G. and PASCHLAU, R. 1961. *Wachstum und Gestalt.* Stuttgart. Thieme.
21. HOLMGREN, I. 1952. The increase in the height of Swedish men and women from the middle of the nineteenth century up to 1930, and the changes in the height of the individual from the ages of 26 to 70. Based on measurements of 46,000 persons. *Acta med. scand.* **142**, 367.
22. HOPKINS, J. W. 1947. Height and weight of Ottawa elementary schoolchildren of two socio-economic strata. *Hum. Biol.* **19**, 68.
23. HULTKRANTZ, —. 1927. Über die Zunahme der Körpergrösse in Schweden in den Jahren 1840–1926. *Nov. Acta soc. Scient., Uppsala*, Vol. extra-ord.
24. KARK, E. 1956. Puberty in South African girls: II, social class in relation to the menarche. *S. Afr. J. Lab. clin. Med.* **2**, 84.
25. KARPINOS, B. D. 1961. Current height and weight of youths of military age. *Hum. Biol.* **33**, 335-354.

26. KENNEDY, W. 1933. The menarche and menstrual type: notes on 10,000 case records. *J. Obstet. Gynec. Brit. Emp.* **40**, 792–804.
27. KHERUMIAN, R. and SCHREIDER, E. 1963. Répartition départmentale de la stature, du poids et de la circonférence thoracique en France métropolitaine. *Biotypol.* **24**, 1.
28. KIIL, V. 1939. Stature and growth of Norwegian men during the past 200 years. *Skr. norske VidenskAkad.* No. 6, 175.
29. KIMURA, K. and KITANO, S. 1959. Growth of the Japanese physiques in four successive decades before World War II. *Zinring. Zassi*, **67**, 37.
30. KNOTT, V. B. and MEREDITH, H. V. 1963. Body size of United States schoolboys at ages from 11 years to 15 years. *Hum. Biol.* **35**, 507.
31. KOPCZYŃSKA, J. and BRZEZIŃSKI, Z. 1961. Rozwoj somatyczny dzieci Warszawakich na podstawie pomiarów wykonanych w 1958 r. *Roczn. panst. szkl. higien.* **12**, 1.
32. KURTH, G. 1955. Zwei nebeneinander wirksame Faktoren in der schwedischen Körperhöhenzunahme seit 1840. *Acta med. scand.* **153**, 159.
33. LEE, M. M. C., CHANG, K. S. F. and CHAN, M. M. C. 1963. Sexual maturation of Chinese girls in Hong Kong. *Pediatrics* **32**, 389.
34. LENZ, W. 1959. Ursachen des gesteigerten Wachstums der heutigen Jugend. *Wissen. Veröff. deut. Gesellsch. Ernäh.* **4**, 1.
35. LENZ, W. and ORT, B. W. 1959. Das Wachstum von Hamburger Schülern in den Jahren 1877 und 1957. *Medizinische* **47**, 2265.
36. MCCANCE, R. A. 1962. Food, growth and time. *Lancet* ii, 621; 671.
37. MCKEOWN, T. and RECORD, R. G. 1962. Reasons for the decline of mortality in England and Wales during the nineteenth century. *Population Stud.* **16**, 94.
38. MANGE, A. P. 1964. Growth and inbreeding of a human isolate. *Hum. Biol.* **36**, 104.
39. MEREDITH, H. V. 1941*a*. Stature and weight of children of the United States. With reference to the influence of racial, regional socio-economic and secular factors. *Amer. J. Dis. Child.* **62**, 909.
40. MEREDITH, H. V. 1941*b*. Stature and weight of private school children in two successive decades. *Amer. J. phys. Anthrop.* **28**, 1.
41. MEREDITH, H. V. and MEREDITH, E. M. 1944. The stature of Toronto children half a century ago and to-day. *Hum. Biol.* **16**, 126.
42. MEREDITH, H. V. and MEREDITH, E. M. 1953. The body size and form of present-day white elementary schoolchildren residing in West-Central Oregon. *Child Developm.* **24**, 83.
43. MEREDITH, H. V. and KNOTT, V. B. 1962*a*. Descriptive and comparative study of body size on United States schoolgirls. *Growth* **26**,283.
44. MEREDITH, H. V. and KNOTT, V. B. 1962*b*. Illness history and physical growth III. Comparative anatomic status and rate of change for schoolchildren in different long-term health categories. *Amer. J. Dis. Child.* **103**, 146.

45. MEYERS, E. S. A. 1956. Height-weight survey of New South Wales schoolchildren. *Med. J. Aust.* **1**, 435.
46. MICHELSON, N. 1944. Studies in physical development of Negroes. IV. Onset of puberty. *Amer. J. phys. Anthrop.* N.S. **2**, 151.
47. MILICER, H. 1962. Investigations on the physical development of youth. In *Physical Education in School.* Ed. W. Missiuro and J. Sadowska. Warsaw. Inst. Physical Culture.
48. MILLS, C. A. 1937. Geographic and time variations in body growth and age at menarche. *Hum. Biol.* **9**, 43.
49. MORANT, G. M. 1950. Secular changes in the heights of British people. *Proc. Roy. Soc. B.* **137**, 443.
50. MORRIS, J. N. 1959. Health and social class. *Lancet* i, 303.
51. MORRIS, J. N. and HEADY, J. A. 1955. Social and biological factors in infant mortality. V. Mortality in relation to father's occupation. *Lancet* i, 554.
52. MURPHY, E. W. 1844. A report of the obstetric practice of University College Hospital, London. *Dublin J. med. Sci.* **26**, 177.
53. NEW ZEALAND GOVERNMENT. 1956. A preliminary report on the 1954 survey of heights and weights of New Zealand primary schoolchildren. *Parl. Paper H. 31*, 22.
54. NISBET, J. D. and ILLESLY, R. I. 1963. The influence of early puberty on test performance at age eleven. *Brit. J. educ. Psychol.* **33**, 169.
55. OPPERS, V. M. 1963. Analyse van de acceleratie van de menselijke lengtregroei door bepaling van het tijdstip van de groedifasen. Amsterdam: Thesis.
56. PRADER, A., TANNER, J. M. and VON HARNACK, G. A. 1963. Catch-up growth following illness or starvation. *J. Pediat.* **62**, 646.
57. PROKOPEC, M. and KAPALIN, V. 1958. Anthropometrie a školm hygiena. *Przeglad antropol.* **24**, 171.
58. QUARINONIUS, HIPPOLITUS. 1610. *Die Grewel der Verwürs lung menschlichen Geschleckts.* Innsbruck.
59. RIGDEN, W. 1869. On the age at which menstruation commences. *Trans. obstet. Soc. Lond.* **11**, 243.
60. ROUTIL, R. 1953. Über die körperliche Entwicklung von Schulkindern (An Karntner Landkindern erhobene Daten und deren Beurteilung). *Öst. Z. Kinderheilk* **8**, 377.
61. RUSBACH, H. W., VAN LAAR, F. and DE HAAS, J. H. 1961. Menarcheleeftijd. Verglijkung van status-quo- en navraag methode. *Tijdsche. soc. Geneesk.* **38**, 411.
62. SAUVY, A. 1961. *Fertility and Survival.* London. Chatto and Windus.
63. SCOTT, J. A. 1961. Report on the heights and weights (and other measurements) of school pupils in the county of London in 1959. London County Council.
64. SUNDAL, A. 1957. The norms for height (length) and weight in healthy Norwegian children from birth to 15 years of age. *Univ. Bergen årbok. Med. rekka.* No. 1.

65. TAKKUNEN, R. L. 1962. Anthropometric studies on Finnish children. *Ann. Paediat. Fenn.* **8**. Suppl. No. 19.
66. TANNER, J. M. 1961. *Education and Physical Growth.* London. University of London Press.
67. TANNER, J. M. 1962. *Growth at Adolescence* (2nd edition). Oxford. Blackwell.
68. TANNER, J. M. and O'KEEFE, B. 1962. Age at menarche in Nigerian schoolgirls, with a note on their heights and weights from age twelve to nineteen. *Hum. Biol.* **34**, 187–196.
69. THOMSON, A. M. 1959. Maternal stature and reproductive efficiency. *Eugen. Rev.* **51**, 157.
70. TOBIAS, P. 1962. On the increasing stature of the Bushmen. *Anthropos.* **57**, 801.
71. TREMOLIÈRES, J. and BOULANGER, J. J. 1950. Contribution à l'étude du phénomène de croissance et de stature en France de 1940 à 1948. *Rec. Trav. Inst. nat. Hyg.* **4**, 117.
72. VALŠIK, J. A., ŠTUKOVSKY, R. and BERNÁTOVA, L. 1963. Quelques facteurs géographiques et sociaux ayant une influence sur l'âge de la puberté. *Biotypol.* **24**, 109–123.
73. WEIR, J. B. DE V. 1952. The assessment of the growth of schoolchildren with special reference to secular changes. *Brit. J. Nutr.* **6**, 19.
74. WHITEHEAD, J. 1847. *On the causes and treatment of abortion and sterility.* London: Churchill. Manchester: Simms & Dinham.
75. WOLANSKI, N. 1958. Výška těla polských hochů v posledním půlstoletí. Zprav. *Anthropol. Společnosh*, **11**, 1.
76. WURST, F., WASSERTHEURER, H. and KINESWENGEN, K. 1961. *Entwicklung und Umwelt des Landkindes.* Wien. Österreichischer Bundesverlag.

SOCIAL MOBILITY AND EDUCATION

Chairman: Sir Sydney Caine

SOCIAL MOBILITY AND FERTILITY

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INTRODUCTION

STUDIES in a number of different countries, which have shown that rates of mobility between different occupational classes are more alike than they were expected to be, have raised questions about the mechanisms underlying social mobility. In preparation for a large-scale investigation, we have made two small pilot studies designed to clarify certain preliminary problems. In the second half of this paper we are going to concentrate on the second pilot study, whose concern was with fertility. Before proceeding to that discussion we shall in the first part give a brief outline of the main idea we hope to explore in the large-scale investigation. It has been rather more fully developed elsewhere.[29]

I. THE MODEL OF SOCIAL MOBILITY

The conclusion about social mobility to which we have just referred is supported by a number of sociological studies. For comparisons over time the Glass report [18] is still the best source. When ten-year age-cohorts born from before 1890 in Britain were compared, very little difference was found in rates of mobility. As for comparisons between countries at more or less the same time, Lipset and Bendix [21] have recently made a comprehensive review of the literature. They measure the rate of social mobility upwards by the proportion of the sons of manual workers who became non-manual, downwards by the proportions of the sons of non-manual workers who became manual. In urban areas rather similar proportions of sons move up in a number of countries—in

France 35 per cent, in Germany 26 to 30 per cent, in Switzerland 44 per cent, in Sweden 29 per cent, in Japan 33 per cent and in the United States 31 to 35 per cent. There is more discrepancy in the proportion moving down, varying from 13 per cent to 38 per cent. Studies in particular cities also show rather similar rates—in Poona and in Tokyo, in Sao Paulo and Kansas City, in Aarhus and Indianapolis. A recent study of a London suburb, Woodford, produced the same results.[27] If, instead of a two-class system, a division is made into several more classes, the rate of mobility—say, into and out of the professions—would probably be less strikingly similar.[22] But at any rate the general conclusion of the work done so far is that rates of mobility, over time and space, are much more alike than they were expected to be.

Cybernetic mechanism

How is this similarity to be explained? Our suggestion is, in brief, that social mobility is the variable element in what Cannon [7] in 1932 called a homeostatic and Wiener [26] in 1948 a cybernetic mechanism, whereby something like a 'steady state' is maintained in each occupational class by means of constant movement into and out of it. We shall discuss how it might work in relation to the distribution of intelligence before we move on to discuss fertility.

We are here defining 'intelligence' in our populations rather narrowly as the qualities measured by the admittedly highly fallible instrument of intelligence tests, orthodox ones, not of the type which tests 'creativity'.[17] This is not the only quality to take into account in any full investigation. The tenor of what Fisher said about what he called 'social promotion' rather than 'social mobility' is clearly right, even though the expression now sounds somewhat Victorian:

> What is perhaps more important is that a number of qualities of the moral character, such as the desire to do well, fortitude and persistence in overcoming difficulties, the manliness of a good leader, enterprise and imagination, qualities which seem essential for the progress, and even for the stable organization of society, must, at least equally with intelligence, have led to social promotion.[14]

Our argument stems from two main observations. The first is that the higher the occupational class, the higher the measured intelligence (I.Q. for short). The usual figures of this sort, for instance those collected by Floud, Halsey and Martin,[14] are for children only. It has been shown again and again in many different countries that the children of (say) professional class parents score better than those of manual workers. There is less published information for adults: most intelligence tests are designed for children, not adults; but what there is, for instance, on the testing of recruits for the

TABLE I

MEAN I.Q.s OF PARENT AND CHILD ACCORDING TO CLASS OF PARENTS

	PARENT	CHILD
Higher Professional	139·7	120·8
Lower Professional	130·6	114·7
Clerical	115·9	107·8
Skilled	108·2	104·6
Semi-skilled	97·8	98·9
Unskilled	84·9	92·6
AVERAGE	100·0	100·0

Armed Services shows that there are the same sorts of differences for adults, although always more marked for the adults in a particular class than for the children.[10] The figures in Table I illustrate this from a paper by Burt.[6]

The second observation is that the I.Q.s of children whose parents' I.Q.s fall in the extreme groups 'regress' from the extreme towards the mean of the general population.[3] Fathers (or mothers) with mean I.Q.s of about 84, or 140, have children with I.Q.s of about 92, or 120, respectively (Table I). These values of course refer to averages; some individuals will be more extreme than their parents but more will be less so. Since the children from any particular class have a greater spread of intelligence than their parents, the shape of the distribution for the population in general is not progressively pinched, but remains roughly the same from generation to generation. Another way of putting it is to say that there is a correlation of less than unity between the mean I.Q.s of parents

and of their children. From Galton [16] onwards many investigators have found this effect not only for I.Q. but also for other quantitative characteristics such as height and span of arms. The ' regression ' towards the mean of the population by the progeny of extreme parents is characteristic of all incompletely inherited quantitative traits, the amount of ' regression ' depending in part on the degree to which the variation is determined by environmental variation. There can be confusion between this phenomenon of ' regression ' and the statistical regression coefficient of offspring on parent. They are different things and the distinction should always be kept in mind.

Given that these two observations are ones that do not apply just to one point of time, it follows that there must have been mobility upwards of more intelligent children to take the place of the less intelligent who move down from the higher classes.[5] Had such mobility upwards and downwards not occurred, the mean I.Q.s for the parents in the upper classes would not be as high as appears in Table I, but would be at least as low as the means for the children; indeed, if the regression had continued without compensation for a series of generations, they would by now have reverted to the general average, and the distribution of intelligence in each class would have become much more like the distribution in the general population than it actually is. A rough stability can be maintained within each class only by a continuous interchange between the classes.[11]

In the absence of inquiry, we do not know what has been happening over time to the distribution of intelligence in the various classes. But in our first pilot study we made an attempt to discover who, in I.Q. terms, actually moved up and down. A sample of forty-seven sons in their twenties was interviewed in 1962 in the town of Cambridge (students being excluded), and the forty-seven fathers in whatever part of England they were living. The results show that there was the same regression from fathers to sons as is found by all investigators, although less neat than that shown by Burt's figures (from a larger sample) in Table I. But the effect of regression was offset by ' exchange ' of sons between the

classes in the way that our idea suggests should happen. Sons more intelligent than their fathers by and large moved up, and those less intelligent down; and, more important, if the distance of movement was measured on a six-point class scale and related to the extent of the difference between the intelligence of fathers and sons, the greater the difference in intelligence, the greater the distance of movement. The consequence of this reshuffling was that the correlation between intelligence and class for non-manual workers as a whole was very nearly restored in the sons' generation to the level it had been in the fathers'. The correlation which had been 0·70 for the fathers came back, as the result of mobility, to 0·68 for the sons. The first Cambridge study therefore supports the suggestion we are making in this section of the paper.

II. MOBILITY AND FERTILITY

One weakness of this first Cambridge study was that it did not readily allow the influence of fertility to be examined. One son was compared with one father. The same applies to all the sociological studies made so far. None has been able to gather data about the differential fertility of various occupational classes because questions have been put in one generation alone, to samples of sons, the sons being questioned about the occupations of fathers. Since the comparison has been between father-son pairs, each father in each class has been shown as having been replaced by just one son. In the Glass report [19] all that could be done was to estimate, not from the data gathered but from general demographic data for the pre-war period, how much mobility there would have to be to counter the effects of such differential fertility as was not fully offset by differential class mortality. Over the period with which he was concerned 300 non-manual fathers would have been replaced by only 252 adult sons, and 700 manual by 748 adult sons. To maintain stability, just in terms of the proportionate numbers in each class, 5 per cent of sons would therefore have had to move up on this score alone.

For our purpose, we hope, in any inquiry, quite specifically to collect data about fertility. In the second Cambridge

pilot study (which was aided by a grant from the Eugenics Society) we therefore adopted a different design, and started with a sample of fathers. The sample comprised 100 men over sixty-five years of age selected at random from the Electoral Register. Each person was interviewed and asked to submit to an intelligence test. The same was done with probands' wives and all their male children. Information was collected from everyone about education, occupation, marital status and number of children. We shall report on the results of this little inquiry later on. Before doing so we want to discuss in a more general way the bearing of fertility on our problem.

An old paradox

Its relevance is fairly clear. So far, in the first part of this paper, we have suggested that if the ' intelligence level ' in the higher classes (or the correlation between class and intelligence) is to be maintained, the effect of regression will have to be offset by mobility. The assumption was the one we have just referred to as being built into the sociological studies, that all fathers were replaced by a son apiece. This may obviously be unjustified; if it is, and in particular if the more intelligent people in the higher classes do not in fact reproduce themselves, there will have to be further mobility upwards on that score to maintain the intelligence level.

Up till recent times at any rate it has been taken that this was actually happening, that mobility was perhaps maintaining stability in the short run but only at the cost of disrupting it still further in the long run, because the more intelligent people who moved upwards would themselves be less fertile. Although not put in these terms, the idea was that the cybernetic mechanism was liable to a kind of increasing oscillation or ' hunting ', with every correction creating a need for a greater correction. It was argued by Fisher [14] that " the economic situation in all grades of modern societies is such as favours the social promotion of the less fertile ". For Fisher this was the reason why all civilizations have in the course of time been eroded. At the same time it was thought that another consequence of the assumed lower

fertility of more intelligent people and higher fertility of the less intelligent was that intelligence in the population as a whole was declining.[1, 2]

The firm finding was that there was a negative correlation between family size and intelligence. A decline in intelligence over time seemed to be implied. The surprise was that in so far as there was any evidence it did not in fact show any decline over time.[9, 24, 25] What could be the resolution of the apparent paradox?

As we have already mentioned, Fisher's suggestion was that genetic factors for intelligence and for infecundity have become associated in the upper social classes. But the fall in average size of families in all social classes seems to have been too rapid to be attributed to any physiological mechanisms affecting the capacity to have children. Penrose[23] produced a theoretical model in which he suggested that genetic equilibrium for the distribution of intelligence could be maintained by the greater fertility of those of near-average intelligence with lesser fertility of those at either extreme.

The best supported explanation of the negative correlation was that it was due to differences in the degree to which parents are successful in planning their families. Contraceptive techniques, natural and artificial, are known to have been more effectively used by the more educated parts of the population and then to have spread downwards through the community.[15, 20] Thus family size has depended more and more upon the willingness to have children. Darlington[12] has pointed out that, as the quality of willingness is to some extent genetic, parents who wanted children would be selectively favoured. Thus, after an initial decrease in the birth rate in any class when it first took to contraception on a large scale, the birth rate would recover as the proportion of 'willing' people in the population increased. Carter[8] has recently reviewed data that seem in agreement with this argument, for generally the birth rates in the upper classes appear to have been increasing during the last fifteen years.

However, the explanation now looks to be quite simple; for the original deduction was wrong, no account being taken of childless people in the generation of the parents or of

the children. This explanation was first suggested by Willoughby and Coogan,[28] who made a survey of the I.Q.s of a small sample of High School graduates in the United States. Their population was divided into two equal parts, a higher I.Q. group and a lower I.Q. group. The married graduates of the lower I.Q. group had a larger average family size than the married members of the higher I.Q. group. However, if the unmarried graduates of both groups were also included, the higher I.Q. group produced more children than the lower I.Q. group because fewer members of the lower I.Q. group married. This study was criticized and its implications ignored because of its small size and non-random sample, but later work has shown that the broad conclusions are applicable to a number of populations.

Minnesota study

In particular, large-scale surveys carried out in Minnesota have shown that past studies of the relationship between I.Q. and reproduction have almost always been faulty and open to bias because of the failure to include those siblings who never reproduced. Higgins, Reed and Reed [19] investigated the fertilities of 4071 persons from 1016 families in which I.Q. values for both parents and one or more of their children were available, together with the I.Q. values for the unmarried or non-producing siblings of the parents. The families were derived from a sub-sample taken fifty years ago of the families of over 500 patients at the Minnesota State School and Hospital for the mentally retarded.

Both the correlations of the parents' I.Q. and the children's I.Q. with the family size were negative and of the same order as in previous studies. The reproduction rate of the parents shows that the groups with I.Q. of 70 or less produced most children (3·81) but the high I.Q. group (above 131) also had a relatively high reproduction rate of 2·94. The high reproduction rate for the group with high I.Q.s is perhaps a recent development as suggested by Carter.[8]

However, Higgins *et al.*[20] also had information about the non-reproductive sibs of the parents in the sample. The highest proportion of unmarried siblings was in the group

with I.Q. of below 55 and the lower I.Q. married couples included a high proportion who never produced offspring. Thus the high reproduction of the lower I.Q. group who had children was offset by the larger proportion of their siblings who never married or who failed to reproduce when married. Higgins *et al.* argue that the I.Q. level of the whole population should remain relatively static from one generation to the next, or at least not drop rapidly.

Cambridge pilot

Our small pilot survey was specifically designed to see whether the Minnesota results on fertility are the same in this country and to investigate a model for social mobility in which differential fertility is considered. In our sample the correlation between probands' I.Q. and social class was 0·5 and the correlation between parents' I.Q. and family size was found to be negative, $r = -0{\cdot}25$, very similar to the values previously found for these relationships. However, when the unmarried probands and those who though married are childless are included in the sample, the correlation between I.Q. and family size becomes positive ($r = 0{\cdot}03$). The effect of this change in correlation on the fertilities of the various social classes can be readily seen in a comparison of manual and non-manual workers. Those couples in the manual classes in our sample who produce children have a reproduction rate of 2·8 and the non-manual classes have a reproduction rate of 2·2 children per couple. Inclusion of the non-reproductive sibs in the data reduces the reproduction rate for the manual class to 2·08 and for the non-manual class to 1·97. The socio-economic classes that are on the border line between manual and non-manual, i.e. Registrar General's classes III and III*a*, have the lowest rate of 1·7 children per couple in this sample. The proportion of unmarried and/or non-reproducing probands is highest in the low I.Q. groups. Further, the correlation between non-reproductive probands and the size of their sibships is 0·38. If the sibship size is larger than four, then on average less than a third of the sibship will marry and produce children. These data are slight, obtained in a pilot survey; yet the results suggest that the old paradox of the I.Q. level

of the population failing to fall despite a negative correlation between I.Q. and family size can be resolved by including in the data the non-reproductive sibs.

A bimodal relationship between I.Q. and family size is suggested by the Higgins data and again by ours, and the same thing has recently been emphasized by Bajema [4] in a larger study in Kalamazoo. Bajema suggests that in his population the positive relationship between intelligence and fertility is a recent development and indicates that a slight increase in the frequency of the genetic factors favouring high intelligence has taken place. It would be useful to know if the same effect is observed when the amount of education received is related to fertility.

THE UPSHOT

The results we have noted may reflect the changes in fertility patterns over time, and particularly the recovery in the birth rate in the higher classes which has occurred in the last quarter of a century in the United States and (to a less but still marked extent) in Britain and some countries of the Commonwealth and Western Europe. Whether this is so or not, it certainly seems clear from the evidence we have presented that differences in fertility between people of different intelligence are not so great as was once thought, and, therefore, that a less amount of mobility is required to offset the effects of differential fertility. But even though differential fertility is not the prompter it was once believed to be, it is obviously important enough to be taken into consideration in any future inquiry into mobility.

The discussion in the second half of this paper has been about one of the many quantitative factors affecting mobility. It has not, as we see it, in any way vitiated the main idea put forward in the first half of the paper. It still seems to us useful to consider mobility as part of a cybernetic mechanism.

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REFERENCES

1. ANASTASI, A. 1956. Intelligence and Family Size. *Psychol. Bull.* **53**, 187.
2. ANASTASI, A. 1959. Differentiating Effects of Intelligence and Social Status. *Eugen. Quart.* **6**, 84.
3. ANDERSON, C. A., BROWN, J. C. and BOWMAN, M. J. 1952. Intelligence and Occupational Mobility. *J. pol. Econ.* **40**, 218.
4. BAJEMA, C. J. 1963. Estimation of the Direction and Intensity of Natural Selection in Relation to Human Intelligence by Means of the Intrinsic Rate of Natural Increase. *Eugen. Quart.* **10**, 175.
5. BURT, C. 1959. Class Differentiation in General Intelligence. *Brit. J. statist. Psychol.* **12**, 15.
6. BURT, C. 1961. Intelligence and Social Mobility. *Brit. J. statist. Psychol.* **14**, 3.
7. CANNON, W. B. 1932. *The Wisdom of the Body.* New York. North.
8. CARTER, C. O. 1962. Changing Patterns of Differential Fertility in Northwest Europe and in North America. *Eugen. Quart.* **9**, 147.
9. CATTELL, R. B. 1950. *Personality: A Systematic Theoretical and Factual Study.* New York. McGraw-Hill.
10. CONRAD, H. S. and JONES, H. E. 1940. *Yearbook Nat. Soc. Stud. Educ.* **39**, 97.
11. CONWAY, J. 1958. The Inheritance of Intelligence and its Social Implications. *Brit. J. statist. Psychol.* **11**, 171.
12. DARLINGTON, C. D. 1960. The Future of Man. *Heredity* **15**, 441.
13. FISHER, R. A. 1930. *The Genetical Theory of Natural Selection.* Oxford. Oxford University Press.
14. FLOUD, J. E., HALSEY, A. H. and MARTIN, J. M. 1956. *Social Class and Educational Opportunity.* London. Heinemann.
15. FREEDMAN, R. F., WHELPTON, P. K. and CAMPBELL, A. A. 1959. *Family Planning, Sterility and Population Growth.* New York. McGraw-Hill.
16. GALTON, F. 1889. *Natural Inheritance.* London. Macmillan.
17. GETZELS, J. W. and JACKSON, P. W. 1962. *Creativity and Intelligence.* New York. Wiley.
18. GLASS, D. (Ed.). 1954. *Social Mobility in Britain.* London. Routledge.
19. HIGGINS, J. V., REED, E. W. and REED, S. C. 1962. Intelligence and Family Size: a paradox resolved. *Eugen. Quart.* **9**, 84.

20. LEWIS-FANING, E. 1949. Family Limitation and its Influence on Human Fertility during the past Fifty Years. *Papers of the Royal Commission on Population*, Vol. 1. London. H.M.S.O.
21. LIPSET, S. M. and BENDIX, R. 1960. *Social Mobility in Industrial Society*. London. Heinemann.
22. MILLER, S. M. 1960. *Comparative Social Mobility*. Oxford. Blackwell.
23. PENROSE, L. S. 1949. *The Biology of Mental Defect*. London. Sidgwick and Jackson.
24. SCOTTISH COUNCIL FOR RESEARCH IN EDUCATION. 1949. XXX. *The Trend of Scottish Intelligence*. London. U.L.P.
25. TUDDENHAM, R. D. 1948. Soldier Intelligence in World Wars I and II. *Amer. Psychol.* **3**, 54.
26. WIENER, N. 1948. *Cybernetics*. New York. Wiley.
27. WILLMOTT, P. and YOUNG, M. 1960. *Family and Class in a London Suburb*. London. Routledge.
28. WILLOUGHBY, R. R. and COOGAN, M. 1940. The Correlation between Intelligence and Fertility. *Hum. Biol.* **12**, 114.
29. YOUNG, M. and GIBSON, J. B. 1963. In Search of an Explanation of Social Mobility. *Brit. J. statist. Psychol.* **16**, 27.

EDUCATION AND SOCIAL MOVEMENT

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EDUCATION is seen by many as the most important influence determining social class change in Britain to-day. As the educational process becomes more effective in sifting the able from the less able, so the possibilities of social movement for an individual become concentrated in the school years and limited after that. Owing to this there has been a progressive transfer of social competition " from the economic to the educational world and from the office and workshop to the school and university."[6]

That men with superior education should improve their social status is not unexpected. The eugenically important question which I propose to discuss to-day is whether social movement is still restricted by unequal educational opportunities. The top rungs of the social ladder are more readily reached by those with high social origins than by those with lower ones, even when standards of education are equal.[5] Given suitable education they both ascend, but the former have less far to go. This may reflect real differences in opportunity; it could equally be explained by differences in drive, aspirations or many other factors—including the knowledge of where to find skilled advice when needed. It would be naïve to seek the reasons for social movement or the extent of unequal opportunity in measured intelligence or attainment alone; many other qualities are needed for success.

I propose here to illustrate the association between education and social movement with data from the National Survey of Health and Development and then to discuss the apparent limitation of educational opportunity which we find in certain types of family to-day.

The National Survey of Health and Development is a continuing study of some 5000 children born during the first week of March 1946. It has been fully described elsewhere[4]

and all I need to say here is that a great deal is known about the home circumstances of these children and the education, social origins and employment of their parents. When they were eight, eleven and fifteen years of age the children were given tests of mental ability and school performance, selected by the National Foundation for Educational Research in England and Wales, who also checked and scored them. At eight years the children were given a picture intelligence test, and tests of sentence completion, and reading and vocabulary; at eleven years the same reading and vocabulary tests were repeated, and new tests of verbal and non-verbal intelligence and mechanical arithmetic were added; at fifteen there were tests of verbal and non-verbal intelligence, reading and mathematics. For each test separately, the crude scores were converted into T scores with a mean of 50 and a standard deviation of 10. Averages of the summed scores at each age were also standardized in the same way as the individual test scores and are used to give a combined assessment of ability and performance.

The association between education and social movement across the generations is well illustrated by the husbands in the survey who had manual working class origins. Table I shows that the social status of these men, in middle age, is closely related to the education they received. These figures are similar to those given by Hall and Glass.[5]

The chances of reaching non-manual employment were small for those whose education ceased at fifteen, and the few who became non-manual workers were mainly employed as travellers, shop assistants, non-commissioned officers, policemen, etc.—jobs that have relatively few middle class characteristics.

Any effort at further education, even among those who did not proceed to secondary school, was associated with improved chances of upward social movement, and the extent of this movement was directly related to the standard of education achieved. Thus, 94 per cent of those with elementary and no further education were employed in manual work, as compared with 68 per cent of those who, on leaving elementary school, took some course of further education. Of those with

the higher standards of education—who on leaving secondary school have taken a degree or diploma—18 per cent remained in the manual occupations where their fathers had spent their working lives and 42 per cent were in the professions.

These men with manual working class origins are of widely different ages; some were at school before the First World War, whereas others were still at school in the Second. All, however, had left school before the passing of the 1944 Education Act.

TABLE I

OCCUPATION OF HUSBANDS RELATED TO EDUCATION
(1955 men with manual working class origins)

	Occupation (when survey child was 11 years old)					
	Profes-sional	Other middle class	Manual: Skilled	Manual: Other	Self-employed	Total per cent
Elementary only (per cent)	nil	4·7	52·4	41·5	1·5	100·1
Elementary and diploma or degree (per cent)	4·7	24·0	63·2	4·7	3·5	100·1
Secondary only (per cent)	0·6	23·7	40·7	31·1	3·9	100·0
Secondary and diploma or degree (per cent)	41·6	37·0	13·4	4·5	3·4	99·9

A total of 8 per cent had stayed on at school after fifteen and are grouped on this basis as receiving secondary education, and a further 17 per cent who had left school before this age took some type of further education, though only one-quarter of them had a diploma or degree to show for it; thus a total of 24 per cent may be said to have had a better than elementary education. How does this compare with the present generation of children from working class homes? It is impossible, yet, to give an exact account of educational achievement in the National Survey as we are still following these young people into employment and noting the technical or university courses they are following. Undoubtedly, however, they will have better educational opportunities than their fathers:

18 per cent of them went to selective schools (i.e. grammar, technical or independent) and a further 9 per cent of those who went to non-selective schools have enrolled in classes in technical colleges.

Education beyond the elementary school, as we have seen, improved the chances of men with working class origins entering middle class occupations. It also widened their social contacts as is evident when we look at the educational background and social origins of their wives. More than a third of those who gained degrees or diplomas married women with secondary education. In contrast, of those whose education ceased when they left elementary school, only 8 per cent married middle class wives and only 4 per cent married wives with a secondary education. The effects of education on social movement are thus reinforced by the pattern of marriage and this in turn influences the opportunities for social advancement at every level. Among the men with purely elementary school education, social advancement may be along unusual lines if they have wives who are above them in education or origins; they are then likely to move into self-employment and this is particularly so if their wives' parents were self-employed. Even among those with a better than secondary education, marriage to a woman from the middle classes or with a superior education confers stability of social status and an insurance against downward social movement.

The superior education and social origins of the wives of those who are moving up the social ladder increase the educational opportunities of their children, both boys and girls, since the aspirations parents have for selective or higher education for their children and the extent of interest they take in their school work depend as much as on the education and origins of the wife as of her husband. And, we have shown elsewhere that a high level of interest on the part of the parents is associated in the child with hard work in class and success in school work.

To come from a manual working class home and leave school at fifteen restricts opportunities for social advancement though, for the 94 per cent with this background who spent their lives doing manual work, there is considerable movement

between different levels of skilled employment; during the first eleven years of this study 19 per cent moved to more skilled employment, 11 per cent to less skilled and 10 per cent moved erratically from one level of skilled employment to another, not establishing themselves at any one level. As the fathers were sorted out by changing their employment, so their children were indirectly sorted by ability as Table II shows.

The children of men who moved from a less skilled to a more skilled job make higher scores than the children of those who remained in the same type of job throughout the survey and their advantage is greater in the 11-year tests than in the

TABLE II

MEASURED ABILITY AND ATTAINMENT

(Children whose fathers had manual working class origins and no more than elementary schooling)

	AVERAGE TEST SCORE		
Social movement	At 8 years	At 11 years	Difference (+ = improved 8–11 — = fell below 8–11)
Up	49·06	49·29	+0·23
None	48·16	48·15	—0·01
Down	47·42	46·61	—0·81
Erratic	47·15	46·71	—0·44

eight. The lowest scores at eight are made by the children of men who move erratically from one level of skill to another, but the greatest relative decline in score between eight and eleven years is shown by the children of men who moved to less skilled jobs so that, at eleven years, they made the lowest scores of all. We cannot say what part of these differences is due to innate endowment and what to the effects of environment, though the divergence of these groups between eight and eleven years suggests that the influence of the environment is important.

I now come to the question of whether educational opportunity is fairly distributed over the population. Even ten years ago there would have been general agreement that the number of children who could benefit from a grammar school, or even more from a university education, was limited and that higher

education could expand only if we were prepared to accept students of inferior quality. To-day we are beginning to realize that there are still large untapped sources of ability—untapped owing to economic, social and medical reasons and to lack of incentives which we may hope to understand and so overcome. Between 1954 and 1961 the proportion of English students with university entrance qualifications rose by 50 per cent, and, if this rate of increase continues, by 1980 some 14 per cent of pupils at school will qualify. So far, the increasing numbers with university entrance qualifications cannot be explained by lower standards of examination marking and there is no evidence that the students doing undergraduate studies to-day are any less able than their predecessors. For example, in Edinburgh the average scores of university students in a verbal intelligence test, which closely relates to the class of degree obtained, has remained constant over a period of twenty-five years in spite of a great increase in the total number of students.[3]

It is generally assumed that, as the facilities for education expand, so social inequalities in opportunity will diminish. There is, however, no evidence that this is true. In the National Survey, the social class differences in entry to selective secondary schools are as large as those reported thirty years ago. The Committee on Higher Education also reached the same conclusion, producing evidence from various sources to show that social class differences in educational opportunity and achievement have in relative or proportional terms changed little in recent years. Many more young people reach the universities but the excess of middle class over manual working class entrants is still, in proportional terms, as great as ever—similar conclusions are reached when General Certificate successes are examined. In other words, even in the most favourably placed social classes where one would have thought that very little talent would, in the past, have remained unused, there has recently been a marked increase in the number of successful G.C.E. candidates and an expansion of university entry.

The persisting social class differences in educational opportunity are comparable with the persisting social class differences in infant mortality, and a description of the latter

may help us to understand some of the difficulties we are likely to meet in trying to provide equal educational opportunities for all social classes and in all parts of the country. During the last fifty years the maternal and child welfare services have been greatly expanded and improved and the chances of survival of infants have improved dramatically in each social class. However, the relative levels of the infant death rates in the social classes have been maintained; indeed the unskilled manual workers are relatively rather worse off to-day than they were in the past. Where services have been expanded or improved it seems that those who least need them have benefited most. For example, the considerable number of additional maternity beds provided during the last twenty years has been taken up largely by women with relatively small numbers of previous pregnancies, whereas those who are having their fourth or later baby—who are in fact a more risk laden group—are still as likely as they were twenty years ago to be delivered at home.[1] The fact that services are improved or expanded does not necessarily mean that they will be available to those who need them most and, put in the educational context, one could imagine circumstances in which large sums of money might be spent on education without improving the opportunities of those sections of the community that are least well provided for to-day.

The very self-fertilizing nature of education, by which those who benefit in one generation see to it that their children benefit in the next, increases the danger of persisting inequalities of opportunity. There are wide regional differences, for example, in the provisions of grammar and technical school places which are historical and bear no relation to the ability of the children who sit the eleven-plus examinations. Since the pressure for educational reform is likely to be greatest in those areas which are already well supplied with grammar schools and least in those which are most deficient, there is a danger that priorities will shift from remedying the gross deficiencies of the worst areas to tinkering with the minor ones in the best.

The evidence of the National Survey is that there are still wide inequalities in educational opportunity in different parts

of the country and in different types of family. There are two main reasons for this: first, during the early years at school social class differences in measured ability increase; second, in the secondary selection examinations children from the poorer homes appear to be at a disadvantage, even when those of similar ability are compared.

The children who between the ages of eight and eleven fall behind the rest in tests of ability and school achievement are characterized as coming from the poorer families, living in the least satisfactory homes, having parents who take little interest in their school progress and attending primary schools with a poor past record of academic achievement. It has been suggested that I have exaggerated the extent of the increase in social class differences in test performance as these children get older.[2] The following table takes account of the statistical objections that have been raised and shows that there is a substantial moving apart of the social class scores between the ages of eight and eleven, when the same tests (of reading and vocabulary) were used on each occasion. The scores of the middle class children improve in relation to the scores of the manual working class in each of the two tests and in the whole group of four tests given at each age.

Between the ages of eight and eleven many teachers and parents put increasing pressure on children in the hope that they will gain a selective school place and it is likely that the middle class children, who are on the average of higher measured ability than the manual working class children and also have the more aspiring parents, are the more pressed. This may explain part or the whole of the increasing advantage of middle class children in the tests, though this would not alter the importance for selection of the improved performance of the middle class children. That this explanation may be correct is suggested by our data for Scotland where children are selected for secondary schools a year later than in England and Wales. Owing to this, the pressures on Scottish eleven-year-olds are likely to be less acute and so the social class divergence in measured ability should be smaller. This is so; at eight years the difference in the average reading scores made by middle and manual working class children in Scotland is 5·85 points

and, at eleven, 5·97 points and in vocabulary the relevant differences are 8·58 points at eight years and 8·47 points at eleven. Thus for these two tests, which were given in the same form at both eight and eleven years, the Scottish children show no social class divergence in contrast to the substantial moving apart of the social classes noted in Table III for the English and Welsh children.

TABLE III

DIVERGENCE OF MIDDLE CLASS AND MANUAL WORKING CLASS SCORES BETWEEN 8 AND 11 YEARS

(English and Welsh children only)

Type test at 8 and 11 years	Divergence in social class test scores between 8 and 11 years	Social class difference at 11 as a percentage of social class difference at 8
Reading	+1·03	118 per cent
Vocabulary	+0·71	111 per cent
Average of all tests *	+0·74	111 per cent

* AT 8 YEARS the average of picture intelligence test, tests of reading, vocabulary and sentence completion.

AT 11 YEARS tests of verbal and non-verbal intelligence, reading, vocabulary and arithmetic.

These average test scores have in each year a mean of 50 and a standard deviation of 10.

The second source of unequal opportunity lies at the point of secondary selection where our evidence is that, when children of the same measured ability are compared, those from the lower manual working class have only half the chance of getting selective school places of the upper middle class children.[4] * Part of this difference is explained by the ability and willingness of middle class parents to pay the fees of independent schools, but inequalities persist when grammar school allocation alone is considered and are most acute in those areas of the country when there is the greatest shortage of places.

The information we are now receiving on school leaving

* For a definition of upper middle class and lower manual working class see Douglas, J. W. B. : *The Home and the School*, pp. 44–45.

age, G.C.E. results and university entry is beginning to show a consistent picture of unequal opportunity and wasted talent. At the moment, we are comparing the educational and employment opportunities of children who have been brought up in areas which show wide differences in the availability of selective secondary school places. I cannot yet give our final conclusions but preliminary tables suggest that, even among children of relatively high ability, the chances of staying on at school after fifteen, of success in the G.C.E. O-levels, and of reaching the sixth form differ considerably from one part of the country to another. This is particularly so for children from manual working class families; they are most at a disadvantage in those areas where selective school places are in the shortest supply.

It is sometimes assumed that the better educated move up the social ladder because they are the most able and that the schools and universities sift out the best endowed and give them the opportunity to rise. This is how we would wish it to be. The vigour and quality of present day societies depend on the efficiency with which their educational systems sift out the able children from all levels and allow them to qualify for posts of responsibility. The evidence from many sources is that this process in Britain to-day is still far from perfect.

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This study was possible only because of the generous help given by medical officers of health, school medical officers, health visitors, directors of education, school nurses and teachers in all part of Great Britain.

REFERENCES

1. BUTLER, N. R. and BONHAM, D. G. 1963. *Perinatal Mortality.* Edinburgh. Livingstone.
2. CARTER, C. O. 1964. The Home and the School: A Review. *Eugen. Rev.* **56**, 93.
3. COMMITTEE ON HIGHER EDUCATION. 1963. *Report: Appendix* 1, 85, H.M.S.O. Cmnd 2154–I.
4. DOUGLAS, J. W. B. 1964. *The Home and the School.* London. MacGibbon and Kee.
5. HALL, J. R. and GLASS, D. V. 1954. Education and Social Mobility in *Social Mobility in Britain.* (Ed. D. V. Glass.) London. Routledge and Kegan Paul.
6. MARSHALL, T. H. 1950. *Citizenship and Social Class.* Cambridge University Press.

GENETICISM AND ENVIRONMENTALISM

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IN 1894, in one of the classics of genetics, Bateson wrote the following:

> It is especially strange that while few take much heed of the modes of Variation or of the visible facts of Descent, everyone is interested in the *causes* of variation and the nature of ' Heredity ', a subject of extreme and peculiar difficulty. In the absence of special knowledge these things are discussed with enthusiasm even by the public at large.[1]

Bateson was then arguing for less talk, opinion and prejudice and some study, experimental study, of variation. He practised what he preached and was rewarded by becoming one of the most distinguished of the early geneticists: it was in fact he who gave the science its name.

Since then our knowledge of genetics has advanced considerably. Nevertheless in many respects and in many fields, what Bateson complained of is still true, most notably in respect to psychological characters in man, where the advance of genetic knowledge has been slight.

In this field more than in others experts and laymen alike too often know more than is known, and, further, know about some particular characteristic things that contradict all knowledge acquired from study of other characteristics of other organisms. Resistance to accepting that others differ from ourselves, resistance to believing that what is good for us may be bad for others, relics of Cartesian dualism, emotional concern about race and class, indignation because so-and-so failed the eleven plus, exaggerated egalitarianism, our habit of trying to find a *single* cause for each phenomenon, and many other factors tend to limit our capacities for dispassionate or critical thought about such matters.

Added to this, we are in a situation where many, most biologists and many geneticists included, have extraordinarily crude ideas of the nature and meaning even of Mendelian genetics, still more so of the relevant genetics of characteristics

showing continuous variation. Many biologists are in this situation, not so much through lack of teaching, but because biology is still pervaded by the type concept, where the structure, form and other characteristics of imaginary, ideal adults is the almost exclusive interest. The concepts are Victorian, and they should have gone out with the belief that children should be seen but not heard! The adult is merely an abstraction from a process of continuous development in which genes and environment and interactions between them play determining parts. Genes affect processes. Adult 'characteristics' are consequences and aspects of those processes.

Bateson himself, like all the early geneticists, was of course reared in this traditional attitude that gave exaggerated importance to adult form, and fell himself to some extent into the trap it creates. He and many others thought in terms of genes for particular adult colours or forms, and, as Stebbins [5] has pointed out,

> started the kind of thinking in which some scientists who are not geneticists indulge, when they speak of genes for an eye, a nose, or for musical genius or the ability to remember mathematical formulae.

It is this kind of thinking that leads to discussion in terms of heredity *or* environment, and to the extreme environmentalism of many physiologists, psychologists and educationalists, and, partly by reaction, to the antithetic extreme geneticism of others.

An illustration of this deficient understanding may be taken from the Newsom report.[4] I would first say that this is one of the most imaginative of the reports on educational problems I have seen. It contains, in Chapter 2, an unexceptionable statement:

> There were well over two and three-quarter million boys and girls in maintained secondary schools in 1962, all of them individuals, all *different* (italics mine).

This statement sets the tone of the report, which frankly recognizes that people differ, and that different people may *need* different treatments.

This is excellent. And it is in no sense of criticism of the

Newsom Committee that I draw attention to my next quotation, which illustrates how little the role of genetic variation is really understood. I quote:

> The results of such investigations increasingly indicate that the kind of intelligence that is measured by the tests so far applied is largely an acquired characteristic. This is not to deny the existence of a basic genetic endowment; but whereas that endowment, so far, has proved impossible to isolate, other factors can be identified. Particularly significant among them are the influences of social and physical environment; and since these are susceptible to modification, they may well prove educationally more important.

Now there is no statement of fact to disagree with there. And no one would disagree that the modifiable factors are more important in the practical sense. What I want to draw attention to, however, are two statements whose meaning will not pass analysis. The first statement is that the intelligence measured by these tests is largely an acquired characteristic and the second is that the basic genetic endowment has proved impossible to isolate.

About the first let us be clear right away. No characteristic is largely acquired. Every characteristic (apart from the breeding potential of an individual *) is entirely acquired. Every character of an individual is acquired during the development of that individual. Likewise every character is genetic, for to acquire a character during development in any particular environment the individual must have the necessary genetic endowment; and this is as true of learned characters as of those that are not apparently learned. But knowledge of the genetic endowment is of little use without knowledge of the environmental circumstances also. Every character is both genetic and environmental in origin. Let us be quite clear about this. Genotype determines the potentialities of an organism. Environment determines which or how much of those potentialities shall be realized during development. The doctrine of fixed abilities is nonsense.

The second statement is more perplexing. The assertion that " the basic genetic endowment has proved impossible to isolate " immediately gives rise to the idea that it may in

* I.e. the potentialities that an individual can hand on to his offspring.

principle be possible to isolate genetic endowments, as if we could pick out the gene or a group of genes for an I.Q. of 126·5. The statement can only have meaning in the context of genes for I.Q. or genes for musical genius, and given such a concept of genetics we are straight in the situation Stebbins complained of. Furthermore, it provides a context in which proof that environment can affect a character is easily taken as proof that genotype does not.

Let us then consider what it is that genetics has to say about such characters as height, or I.Q. in man, yield in corn or hair number on flies, in order to approach some feeling for the complementary roles of genotype and environment in determination of the developmental processes concerned.

First we must remember that all such characters are relative. When we say a man is 5 ft. 6 in. high we are saying that he is 6 in. shorter than two standard yards, tucked away in the National Physical Laboratory or the British Museum or wherever the standard yard is kept. Another man is 6 ft. 3 in., and these measures have only two kinds of use. One is to determine which sized door, trousers or what you will will be relatively more suitable for one or other man. The other is to enable us to say one man is taller than the other. The taller man may be said to have the character tallness or to be tall, but this is purely a shorthand statement meaning he is taller than some or taller than average. Gulliver was tall in Lilliput but dwarf in Brobdingnag. His tallness or dwarfness had no more to do with him than with the other members of the society in which he found himself. The character is an abstraction and we give it meaning more than as a mere relationship at our peril. Talk of genes for tallness, or of isolating a basic genetic endowment for tallness is dangerously misleading.

It is the more so with intelligence, musical ability, mathematical ability. When we try to measure these we are measuring relations, and we say nothing about the individual as such except to place him on a scale relative to other individuals. We can say nothing useful about intelligence as measured, except in relation to a population.

When considering any such character therefore, we are

considering a population. When asking about the genetic factors or the environmental factors that influence intelligence, or any other character, we are asking about the causes of variety in a population. And whatever we say of an individual can only be a statement concerning the differences between that individual and others in the population, whether what we say concerns his intelligence as measured, or the causes of his relatively high or low I.Q.

Let us then consider a character about which much is known, hair number in flies, which can provide a model to show how characters of this kind may be influenced. Let us remember we can only talk about differences of hair number. Let us ask what factors may affect hair number.

First, of course, any population we may consider has a mean or average hair number.

Second, it shows variety of hair number, it has variance.

Third, the variance has causes, many causes. (The concept of *a* cause of *a* character has no place in genetics and should have none in biology.)

Fourth, different populations may differ in mean, variance or both.

Fifth, if two populations differ in mean, there are causes of the difference.

Sixth, if two populations differ in variance, then there are more, or more effective, causes of variety in that which has the greater variance.

Seventh, unless the populations have been artificially produced by special breeding programmes designed to eliminate genetic variety, the causes of variance are *always* both genetic and environmental, and the variance can be partitioned into three components, genetic variance, environmental variance and variance arising from genotype-environment interaction. In other words we may classify the causes of variance into these three groups and assign them relative importance. This is true of every continuously varying character that has been adequately studied of any species of outbreeding organism. It is undoubtedly true of I.Q. variation.

What may differ from population to population, from character to character, or from time to time, is the relative

importance of these three classes of cause, or, more precisely, the proportion of the variance that is assignable to each of these three causes. The proportion of the variance assignable to genetic causes, which we call the heritability of the character, may vary for either of two reasons. First, it will increase with increase of, and decrease with decrease of the amount of genetic variety in the population. Second, it will decrease with increase of and increase with decrease of the variety of environments in which the individual members of the population develop.

This is of the utmost importance. The more uniform the environment, the greater will be the importance of genetic variety. In other words the greater the equality of opportunity the more important will differences of genetic endowment become.

This is all rather simple. But relations become more complex when we consider the third factor—genotype-environment interaction, a factor whose importance will increase with the complexity and variety of the genetic and the environmental causes of variation, and which must therefore be of maximum importance in characteristics that vary with social background, home background, etc.

Here I want to give an example from experiment, and also to illustrate the concepts with two simple models.

The experiment concerns hair number of flies. A population of flies was classified for hair number, and two samples, exactly comparable, were taken from it. Each sample was used to generate a new generation, by mating together the least hairy female and male, the next least hairy pair and so on to the most hairy pair. The average hair number was obtained from the progeny of each pair. The two samples' progenies were grown in different environments, one at 20° C, the other 25° C, the parents having all been grown at 25° C. The results are summed up in Figure 1, in which the statistical relation between parent hair number and progeny mean hair number is shown for each environment.

Two things are immediately obvious. First, the lower temperature produces higher hair numbers. The environmental difference alters relevant developmental processes

effectively. Second, the higher hair number parents produce higher hair number progenies. Relevant developmental processes are effectively altered by genetic variety in the samples. Two less obvious things are important. In neither environment is hair number completely inherited, that is to say that the extreme hair number parents produce offspring with less extreme average hair numbers. Second, the relative

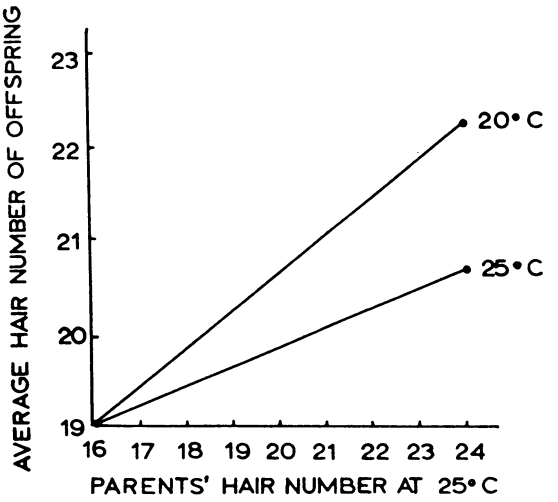

FIGURE 1. THE RELATION OF OFFSPRING HAIR NUMBER AT 20° and 25° C to PARENT HAIR NUMBER AT 25° C

The differences of regression coefficient and mean both differed significantly between environments.

effect of genetic difference varies with environment, and the relative effect of the environmental difference varies with genotype. In fact the temperature of development made no difference at all to the progeny of the low hair number parents, but a good deal of difference to the progeny of high hair number parents. This is an example of genotype-environment interaction. It leads to different heritabilities in the differing environments. When the flies developed at 20° C, 40 per cent of the variance was genetic, the remaining 60 per cent being largely assignable to unknown environmental variables other

than temperature. When the flies developed at 25° C, only 20 per cent of the variance was genetic, 80 per cent being assignable to residual environmental factors.

I give this example not only to show what genotype-environment interaction means, but also to underline the point that heritability, that is the proportion of variance that is genetic, is not a quantity that belongs to a character in a species, but to a population in its environment and will vary according to the population and the environment. Controversies concerning the heritability of I.Q. in man, arising from different estimates obtained by different workers from different samples are rather silly. For example it is to be expected that heritabilities estimated from study of twins reared together should differ from those derived from study of twins reared apart.

To return to genotype-environment interaction, the situation can be more striking than that illustrated above, for a change of environment can actually change the order of genotypes.

For a simple example I turn to yield of wheat, two varieties being compared over two years, that is in two environments. In one year Turkey Red yielded significantly less than Big Frame. In the next year significantly more. Neither environment is best, neither variety is best. One variety is better in one year, the other in another year.

Two imaginary model situations will illustrate the significance of these considerations. Consider I.Q., and imagine a population genetically simple enough to contain only five kinds of genotype each equally frequent. Set it in a situation so simple that there are only five kinds of environment, all likewise equally frequent.

Without genotype-environment interaction we might get phenotypic I.Q. classes as shown in Table II.

In this simple situation in which there is no genotype-environment interaction it is obvious that, if we wished to maximize I.Q., we should give everyone environment E. If on the other hand our motives were egalitarian, we should give the A genotypes environment E, and the E genotypes environment A. Or if for economic or other reasons we were only able to give environment E (the best environment from this

point of view) to a few, and wished to have as many high I.Q.s as possible in the population, we should have to devise selection techniques so that we could choose E genotypes for the E environment.

TABLE I

RELATIVE YIELDS OF TWO VARIETIES OF WHEAT IN TWO YEARS

		Variety	
		Turkey Red	Big Frame
Year	1913	100	107
	1914	100	85

(Data in Hayes and Immer,[3] Table 38, p. 299.)

TABLE II

		Genotypes				
		A	B	C	D	E
Environment	A	80	85	90	95	100
	B	85	90	95	100	105
	C	90	95	100	105	110
	D	95	100	105	110	115
	E	100	105	110	115	120

By contrast, with genotype-environment interaction we might have something like this:

TABLE III

		Genotypes				
		A	B	C	D	E
Environment	A	80	90	100	110	120
	B	120	110	80	90	100
	C	90	80	120	100	110
	D	110	100	90	120	80
	E	100	120	110	80	90

Now, because I have chosen an extreme situation in which all the variance is interaction variance, we have no environment better than any other, and no genotype better than any

other. Each genotype requires a different environment if it is to develop the highest I.Q. it is capable of. A requires environment B, B requires E, C C, D D and E A.

Now no natural situation is as extreme as these, and no natural population as simple. In natural situations there will be some interacting and some non-interacting variations of both genotype and environment, so that we must think of a model somewhere between the two I have given. But in addition, we must remember that natural populations are of fantastic genetic complexity, and natural environments are also complex, especially the natural environment of civilized man, with all the variation in social, economic, educational, home and school background, etc., etc.

It is becoming more and more clear, the more experiments we do on the genetics of natural populations, that the old idea, itself relating to the biological type concept, that populations are genetically rather uniform, there being by and large one normal or wild-type, along with many abnormalities, each rare, is totally misleading. Normal flies, or normal men, comprise an extensive array of differing genotypes, genotypes that differ in their effects on any characteristic we like to study closely enough. So extensive is this variety that we may say without exaggeration that, apart from identical twins, no two individuals are, or ever have been, genetically exactly alike. Each human being is genetically unique. We are gradually coming more and more to understand why this should be so; but the fact and its immense implications, still less the value of such variety to society, are barely recognized.[6, 7] Superimposed on this genetic uniqueness, we have the uniqueness of environment of each individual during development, some of the environmental variation itself having genetic variation as part cause. Unique genotype and unique environment are interacting in the development of each individual in unique ways, and, though we must classify individuals into groups for scientific, administrative or educational purposes, we ignore this uniqueness to our great loss and at our peril, and it makes nonsense of segregation of races justified solely on the basis of differences of average even if the average differences may be real.

Consider a simple example of this variety, using a clear case of a genetic difference in the development of which environmental variance plays little part: red-green colour-blindness. First this is a genetic difference leading to a difference in perception of the outside world. A colour-blind individual meets a different environment, experiences a different world, from a colour-perceptive individual. No doubt there is loss in this for him, though it must be loss he cannot appreciate any more than we can. But there is also gain. One factor contributing to the success of Drosophila experiments in my department is the colour-blindness of one of our senior workers. He can classify some phenotypes important to our breeding programmes which the rest of us cannot. We can classify some that he cannot. He tells me also that colour-blindness has advantages in another field, the dyeing industry. For certain jobs which involve careful matching of tints of the same colour, colour-blind individuals are in great demand. Both genotypes have value, and society is the better for the existence of this variety.

We may even question whether a complex, integrated society such as ours is possible without genetic variety, for the integration of a society clearly requires differences as well as similarities between the individuals that have to be integrated. The rigid social systems of ants and bees depend largely on age structure and rearing differences and rather little on genetic differentiation apart from that distinguishing the sexes of bees. But it is striking that among primates even so relatively simple a social organization as that of baboons seems to depend on uniqueness of the individual. All members of the group are individually recognizable by each other. It seems doubtful whether the complexity of social organization upon which human success depends could have developed without the extensive genetic variety human populations contain.

Another example I wish to consider illustrates an even greater complexity of genotype-environment interaction: this time from work on mice. De Fries[2] has recently reported the following facts. Pregnant female mice of various inbred strains were subjected to stress conditions in swim tanks, sound chambers, etc. Studies were made of behaviour of

their offspring and those of controls. Significant effects of treatment of mother on behaviour of offspring were observed, but these effects of treatment varied both with genotype of mother *and* with genotype of offspring. Here we see a beautiful example of mother's genotype interacting with mother's environment to affect offspring's environment, and offspring's environment interacting with offspring's genotype to determine the mode of offspring's development and hence the observed characteristics of offspring. Such situations, made more complex by considering interaction between parent and offspring in the house, parent, offspring and teacher in the school and so on provide the sort of model we must think about when considering biological variation in relation to educational policy.

An old cardinal principle of education takes these complexities into account. It is the principle that each pupil requires individual attention, in other words a unique environment. Yet at the present time we see more and more stress on a different principle, that it is unfair if some get better opportunities than others. This would be fine if it were not coupled with an implicit assumption that the best opportunity for one would be the best for all. Thus arises much of the disturbance about selection for education, and the unfairness of giving some ' better ' opportunities in grammar schools and universities, controversy about the eleven plus, and so on.

All these attitudes are tenable only if we assume that there is such a thing as human nature. But there is not. There are as many human natures as men. To do his best for society, or to get the most from life, or to have the best compromise between the two, each individual would require a unique, carefully chosen environment. This we cannot provide, if only because we do not know enough. But in one sense at least selection techniques for educational streams are an attempt to do the best we can. Some injustice arises from selection because our selection techniques are of limited efficiency, but we know, both on the theoretical grounds, and from direct experience, that injustice can also arise through lack of selection, or from giving individuals a so-called better environment which is unsuitable for them.

For an example of this experience I quote Valentine: [8]

Again, one is constantly brought up sharply by ignorance among parents of elementary facts as to innate differences in mental ability. Some parents simply cannot believe that one of their own children may be innately dull and unable to emulate the achievements of father or elder brother; and it is unfair to expect the teacher or the psychologist called in to treat the child, to be the first to inform the parents of the main facts as to individual differences.

Such ignorance may be found even in well-educated parents. For example, one of such recently brought his eighteen-year-old daughter to me for advice about difficulties in reading. She was a pleasant and sociable girl, and in a large private school she had been pushed up finally into one of the Senior classes. Yet her I.Q. I found to be only about 90, and her reading quotient even lower; she made mistakes in reading words usually assigned to the age of seven. Her arithmetic was even worse; though she was studying stocks and shares, she was uncertain on multiplication tables, and simple fractions. It was another example of the familiar fact that a child's progress may be made worse than it need be by work beyond his capacity. The father was told that the girl must be started in reading and arithmetic from the beginning. Expert coaching brought her along very well for a time, but it was eventually spoiled by the father's strong conviction that more advanced work would increase the rate of progress. The father attempted this at home, and the girl at once showed confusion and retrogression. Our educational psychologists could give many such examples in which parents' ignorance as to elementary psychological facts, or worse, their strong belief in erroneous ideas, have made proper treatment much harder.

In our local press recently parents have protested against a child being placed in a C class on the grounds that it did not give him the same chance as those who were in the A or B stream; they were completely unaware of the fact that a child is placed in a C class precisely in order to give him the best chance of progress.

Even some teachers still allow too little for individual differences. Children are pushed up to higher classes on the ground of age; lack of progress is sometimes attributed too readily to the child's not doing his best. Even a child's poor performance in an intelligence test is sometimes taken by a teacher as a reflection on her own teaching.

Let me end by pleading that geneticism and environmentalism both may die a rapid death; that we begin to realize that the question " Is this character inherited or is it acquired? " can never have an answer other than 'No', because the question has no meaning; and that we take into account, in social and educational theory, policy and polemic, the biological fact of the uniqueness of the individual, implying as it does that the

needs of each individual are unique and that it is *just* to treat different people differently so long as each is treated as well as possible.

Nobody has the equipment necessary for discussion of these topics unless he thoroughly understands the concept of genotype-environment interaction in development, and appreciates that all men are different, different from conception.

DISCUSSION

It was pointed out during the discussion following this paper that many business men used to regard it as better to let their children gain experience in offices from fourteen rather than go on at school since they were not scholastically-minded, and the questioner asked whether the Butler Act was wrong in raising school leaving age rather than reducing the size of classes. He quoted Dean Alington, Headmaster of Eton, as holding the view that there were two kinds of boys, the scholastic and the non-scholastic, and that the latter were better put to practical work without delay. PROFESSOR THODAY replied that he would not feel able to judge the cultural, social and economic merits of smaller classes versus longer schooling, but he did not feel the concepts of two distinct classes of boys likely to be anywhere near the truth, the variation being continuous not discontinuous.

Asked whether he had not been unrealistical in dividing characters into three classes, genetically determined, environmentally determined and interacting, and in suggesting little action of environment, Professor Thoday said that he had not classified characters at all, and in fact did not think it proper to do so. It is the causes of variation of any character that must be so classified, all characters varying for all three causes.

In answer to another questioner Professor Thoday said that he would thoroughly agree that we should have equal opportunity to enter the maximum variety of educational systems.

REFERENCES

1. BATESON, W. 1894. *Materials for the Study of Variation.* London. Macmillan.

2. De Fries, J. C. 1964. Effects of Prenatal Maternal Stress on Behaviour in Mice: A genotype-environment interaction. (Abstr.) *Genetics* **50**, 244.
3. Hayes, H. K. and Immer, F. R. 1942. *Methods of Plant Breeding.* New York and London. McGraw-Hill.
4. Newsom, J. (Chairman). 1963. *Half Our Future.* London. H.M.S.O.
5. Stebbins, G. L. 1963. ' Perspectives. 1.' *Amer. Scientist* **51**, 362.
6. Thoday, J. M. 1962. Molecular Individuality and Population Structure. *Advanc. Sci. Lond.* **19**, 45–49.
7. Thoday, J. M. 1963. Causes and Functions of Genetic Variety. *Eugen. Rev.* **54**, 195.
8. Valentine, C. W. 1948. Some Present Day Trends, Dangers and Possibilities in the Field of Psychology. *Brit. J. educ. Psychol.* **18**, 1.

SOCIAL MOBILITY AND EDUCATION

Conclusion

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MUCH has been written in the past about the relationship between social class and fertility, and more particularly about differential fertility and its possible effects on movement between one class and another. The three papers given at this session, while each throwing light on individual aspects of the problem, together emphasize the difficulty of formulating any general conclusions.

The paper prepared by Dr Young and Dr Gibson, and presented by the latter, discusses various theories advanced in the past, on the one side connecting mobility with the need to maintain the numbers of the higher social classes in conditions of differential fertility and on the other relating fertility inversely to I.Q. levels. The authors of the paper raise doubts concerning these theories, examining in more detail the statistical basis of previous conclusions regarding the comparative fertility of different I.Q. classes. They suggest that differential fertility, though still to be reckoned as one of the factors involved, is not as large an influence as some writers have supposed; and they themselves rely for the explanation of the mobility between classes, the rates of which they find to be unexpectedly uniform in varied societies, on a 'cybernetic' mechanism based directly on intrinsic differences in intelligence rather than on differences in fertility.

Dr Douglas's paper was more concerned with mobility as affected by differences in the education actually received, and in particular with the varying extent to which children of different class origins take advantage in practice of the opportunities in principle open to them. He was not to any large extent concerned with relationships between mobility and fertility and it would not, I think, be unfair to summarize his conclusion as being that class differences still have a very

strong tendency, through psychological attitudes and economic pressures, to perpetuate themselves via the educational system.

Finally, Professor Thoday, speaking as a biologist rather than a sociologist, strongly emphasized the individual character of reactions to any situations and gave a warning against too much generalizing on the basis of classes and categories of a rigid kind. His paper rubbed in the lesson to be learned from the others that much more detailed investigation is needed into the complex of what Professor Thoday called " genotype-environment interaction in development " if we are to understand effectively the interactions of social mobility and fertility.

GENETIC ASPECTS OF MEDICINE

Chairman: PROFESSOR L. S. PENROSE

INTRODUCTION

L. S. PENROSE

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THE clash between the aims of medicine and eugenics has for a long time raised a basic ethical principle. It is supposed that the advance of therapeutic techniques enables weaklings to survive and thereby endangers the genetical structure which the human race has achieved by natural selection. Potentialities for physique and intelligence are consequently expected to decline. Must we, therefore, for the good of the race, abandon the physician's primary duty to the individual?

In the early days of Eugenics Societies, the expected dysgenic evils were actively combated by propaganda directed against proliferation of the supposedly unfit. Much of this energy was expended on the basis of incorrect assumptions about human genetics and the psychological atmosphere, so created, sometimes tended to hinder unbiased fact-finding researches. I am not a member of this Society, so I shall not be accused of boosting it if I say that its members have always practised considerable restraint in these matters as compared with other groups.

When Galton first expressed his opinion that it should be quite practicable to produce a highly gifted race of men by judicious marriages and by checking the birth rate of the unfit, very little was known about genetics. As more was learned about the mechanisms of genes, chromosomes, population dynamics and the interaction of genes with environment, many of the fears of the early eugenists were seen to be unfounded; but most of the hopes of dramatic success have also been shattered.

The central question of eugenic policy remains still unanswered: namely, how to specify which genes should be encouraged. Naturally, everyone thinks that his own family, tribe, nation or race must be the best and the healthiest for the

future; but this is an arbitrary decision. The control, prevention or cure of hereditary disease is a legitimate aim of ordinary medicine and grandiose eugenical ideas about race improvement are unnecessary. Eugenics introduces values not recognized in medical philosophy and, on occasion, in the form of race hygiene they have even been used to justify inhuman and disastrous policies.

At the back of some of this thinking there are two types of fallacy which have been gradually exposed. First, and most obvious, is the attribution to heredity of a disease or social disturbance which subsequently is shown to be mainly caused by environment. This happened with leprosy, tuberculosis, syphilis, nutritional deficiencies, poverty and criminality. People are becoming increasingly inclined now to accept the significance of environmental influences on physique and intelligence.

Then, secondly, there is the false assumption that genes are naturally and consistently either good or bad so that extermination of all bad genes could be a reasonable aim. This dogma is contradicted by the subtle phenomena of gene interaction. The same gene can be favourable in one circumstance and unfavourable in another like anti-malarial genes, which will be discussed later in this symposium. The problem of maternal-foetal incompatibility also belongs here. The genes responsible for it are not bad in themselves, but in specific situations they cause serious damage to the foetus. Chromosomal aberrations, especially inversions, are also known to have surprisingly unpredictable effects on fitness.

Furthermore, as environment alters, there can be neutralization or even capitalization of formerly deleterious genic effects. A typical example, which I have quoted before, is the hereditary loss of body hair which obliges the human animal to wear clothes in cold climates. Correction of myopia by glasses or of diabetes by insulin is no more extraordinary than correction by clothes of the effect of our gene for hairlessness, which we take for granted. Continuous evolution in a civilized society has involved changes of gene constitution in countless ways. These changes are deleterious only if the ideal constitution is considered to be that required for life in uncivilized,

wild conditions. Formerly, we had to acquire particular genes to defend ourselves against parasitic diseases, for example, through many centuries of natural selection. Now we do the same thing more efficiently in a few decades by pharmacological research and the introduction of public health measures. Civilization and its medical appurtenances have, in fact, increased human fitness to such an extent that a fundamental problem now confronting the human race is how to diminish its fertility.

The founder of the Eugenics Education Society, Francis Galton, said that its business was to publicize and popularize results of relevant scientific inquiries; perhaps also it could learn about the needs of investigators and encourage them. The existence of the present symposium indicates emphasis on this tradition.

MEDICINE AND NATURAL SELECTION IN MAN

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WE take it for granted in our ordinary daily life that no two of us are alike. We just assume that we can distinguish among our fellow men at sight and indeed the organization of our society could not continue as we know it were this not the case. Nor are the differences among us merely superficial: they extend to every aspect of our being, anatomical, physiological and mental. Some of them reflect the differential action of external agencies, and others the effects of society itself. But much of our diversity is inborn, springing from differences in the genetical materials with which we are endowed by our parents. Indeed it is not only true that phenotypically each of us is unique: identical twins apart, no two of us are alike genetically either.

Now, genetical differences are expressed in every character or trait that we can display and their effects may be of any size from those that involve the grossest of upsets to others so small as to be detectable only by the most refined observation. Furthermore, these genetical differences affect our fitness, using this term in its Darwinian sense, that is our prospective contribution to posterity. In other words, we, like other species, are subject to the action of natural selection. In fact, some of the clearest and most elegant examples of the action of natural selection come from man.

The action of natural selection is, of course, by no means always or even generally towards continuing change in the genetical constitution of a population. Change will be brought about when circumstances alter, but once an adequate genetical adjustment has been achieved selection will act towards stabilizing that adjustment. We thus have a picture of human populations as mixtures of genetically diverse individuals, but mixtures which tend towards equilibria under constant

or near constant conditions. In so far as equilibrium is approached or attained it is because a balance is achieved among the agencies of variation—especially mutation and recombination—and the forces of selection. If these stay unaltered, the equilibrium will persist once it is achieved; but if the agencies of variation or the forces of selection are disturbed the genetical constitution of the population will alter accordingly. Equilibrium is not rigid but labile, springing from an interplay of pressures and subject to adjustment by any alteration of that interplay. And one of man's most active pursuits is the alteration of these pressures and their interplay, not only on other species wild as well as domesticated, but also on man himself. Sometimes our action is deliberate and sometimes unwitting. Sometimes it is directed and sometimes incidental. But conscious or not, directed or not, it must be expected ultimately to have its genetical consequences in the population.

MUTATION AND SELECTION

Many genetically determined upsets have been recognized and though each is relatively rare in its occurrence, taken together they have been estimated to account for at least 1 per cent of all births in a population such as our own.[4, 11] Where it has been established, the mode of inheritance is in some cases through a dominant gene and in others through a recessive, the genes sometimes being autosomal and sometimes sex-linked. But, whatever the mode of inheritance, these genes must be expected to reduce in greater or lesser degree the Darwinian fitness of the individuals displaying their effects. In some cases we have reliable estimates of the size of this reduction in fitness, though in others we can still do little more than guess.

Now any such reduction of fitness must be tending to reduce the frequency in the population of the causal gene, for by its very action the gene is reducing the numbers of offspring to whom it can be transmitted by the normal process of heredity. Yet these genes, and with them the disabilities they determine, have not vanished. They must be held in the population by some process which balances this action of natural selection

and this process must commonly be replenishment of the pool of these genes by mutation from their normal alleles. So any agency which raises the rate at which this mutation occurs, or which reduces the loss of fitness by increasing the affected individuals' prospects of leaving offspring, must tend to raise the frequency of the gene and hence the frequency of the disability in the population.

We can explore the consequences of raising the mutation rate or reducing the loss of fitness with greater precision if we assume that the effects of mutation and selection balance one another so that the population is in equilibrium in respect of the gene and the disability it causes. Then if f is the proportion of the population displaying the effect of the gene, μ is the rate of mutation to the gene from its normal allele and s is the reduction in fitness suffered by the affected individuals (i.e. their fitness is $1-s$, where that of normal individuals is 1) it can be shown that at equilibrium $f = k\frac{\mu}{s}$ where k is a constant whose value depends on the mode of inheritance, being 1 for recessive genes and 2 for dominants.

Clearly increasing the mutation rate, μ, will raise the incidence of the disability, f, as also will reducing the unfitness, s. Furthermore the effect of change in one can be matched exactly by an appropriate change in the other. For example, either doubling μ or halving s will result in doubling f, when equilibrium has been re-established.

The consequences of raising the mutation rate in increasing the incidence of disability have been the subject of wide discussion in relation to the hazards of ionizing radiations. They require no further emphasis now, though we may note in passing that if the present pattern of exposure continues, any incidence in mutation rate as a result of man-made radiation will arise in the main from their medical uses. The consequences of reducing the unfitting effect of the genes has received less attention, though this may be said to be one of the aims of medical science in that means are constantly being sought for remedying or at least alleviating the disabilities caused by these genes. The introduction of treatment by insulin has materially raised the fitness of diabetics with the

result that the genes which appear to underlie this condition must be increasing in frequency. To take another example, the treatment introduced some years ago for phenylketonuria offers promise of materially alleviating this condition with the result that phenylketonuriacs may be leaving progeny behind them. With such a reduction in *s*, the phenylketonuria gene will inevitably increase and with it *f*, the incidence of the condition in the population. True, an increase from this cause has yet to be demonstrated: it is still no more than an inference in this case. In the case of pyloric stenosis, however, we already have evidence of the raised incidence of the condition among the offspring of successfully treated parents,[3] though the genetical causation of the upset has not yet been made fully clear.

How serious a problem is medicine creating for itself by the changes it is gradually bringing about in these forces of natural selection? From the direct point of view it may be held that such an increase can hardly be regarded as menacing in so far as it springs from the success of a treatment which can be extended in its application—though in general one would prefer to see fewer rather than more individuals coming to require either the insulin injections of the diabetic or the sort of diet which is now being fed to phenylketonuriac children.

The problem may also be examined from another point of view. How rapid an increase in a genetically determined disability is to be expected from its successful treatment? As we have already seen, a rise in the mutation rate and a corresponding reduction in the unfitness will have the same effect on the incidence of disability when equilibrium has been re-established; but they do not result in the new equilibrium being approached at the same speed. This difference is illustrated in Figure 1 where doubling the mutation rate and halving the unfitness are compared in their effects on the incidence of achondroplasia, haemophilia and phenylketonuria. Achondroplasia, mediated by an autosomal dominant gene, reduced fitness to 0·2, *s* being thus 0·8. Haemophilia, due to a sex-linked recessive, reduces fitness to 0·125, *s* being thus 0·875. Phenylketonuria, due to an autosomal recessive gene, reduces fitness to 0, *s* being thus 1.

The effects of altering either μ or s are shown very much more rapidly for achondroplasia than for phenylketonuria, haemophilia being intermediate and nearer to achondroplasia. In all cases, however, the rise in f is much slower following the halving of s than after the doubling of μ. The reason is, of course, that although the increase in f is the same in the two cases, it comes about in different ways as can readily be seen if we look at the incidence of achondroplasia. Here every achondroplasia gene, being dominant, shows its effects in every individual carrying it, and since $s = 0{\cdot}8$ every generation sees 80 per cent of the genes lost, the loss being made good by new mutants. So, out of every hundred achondroplasiac individuals, twenty owe their causal genes to transmission from their parents and eighty owe them to mutation. If the mutation rate is doubled, 160 new genes come into being by mutation, with the result that $160+20 = 180$ affected individuals appear in the first generation for every 100 in their parental generation. Of these 180, 20 per cent, or 36, pass on their genes to their offspring who together with another 160 new mutants give $160+36 = 196$ in the second affected generation and so on. The approach to the new equilibrium with 200 achondroplasiacs for every 100 originals, is thus rapid and is virtually complete in four generations, that is in just over a century.

If, however, s is halved to $0{\cdot}4$, affected parents pass on sixty out of every 100 genes to their offspring. The contribution from mutation stays at 80, and $60+80 = 140$ achondroplasiacs for every 100 in the parental generation. Of these

FIGURE 1. (*Opposite*) The approaches to the new equilibrium, with double the incidence of affected individuals, produced by doubling the mutation rate (2μ) and halving the unfitness of affected individuals ($s/2$), for: A. achondroplasia (mediated by an autosomal dominant gene with $s = 0{\cdot}8$); B. haemophilia (mediated by a sex-linked recessive gene with $s = 0{\cdot}875$); and C. phenylketonuria (mediated by an autosomal recessive gene with $s = 1$). The rise in incidence of the condition in the population from its initial value, taken as 100, towards the new value at the new equilibrium, shown as 200, is given by the solid lines. In all three cases the rate of rise with $s/2$ is slower than that with 2μ though the ultimate equilibria are the same. The fall from the new equilibrium, brought about by restoration of the original mutation rate or unfitness as the case may be, is shown by the broken lines marked R.

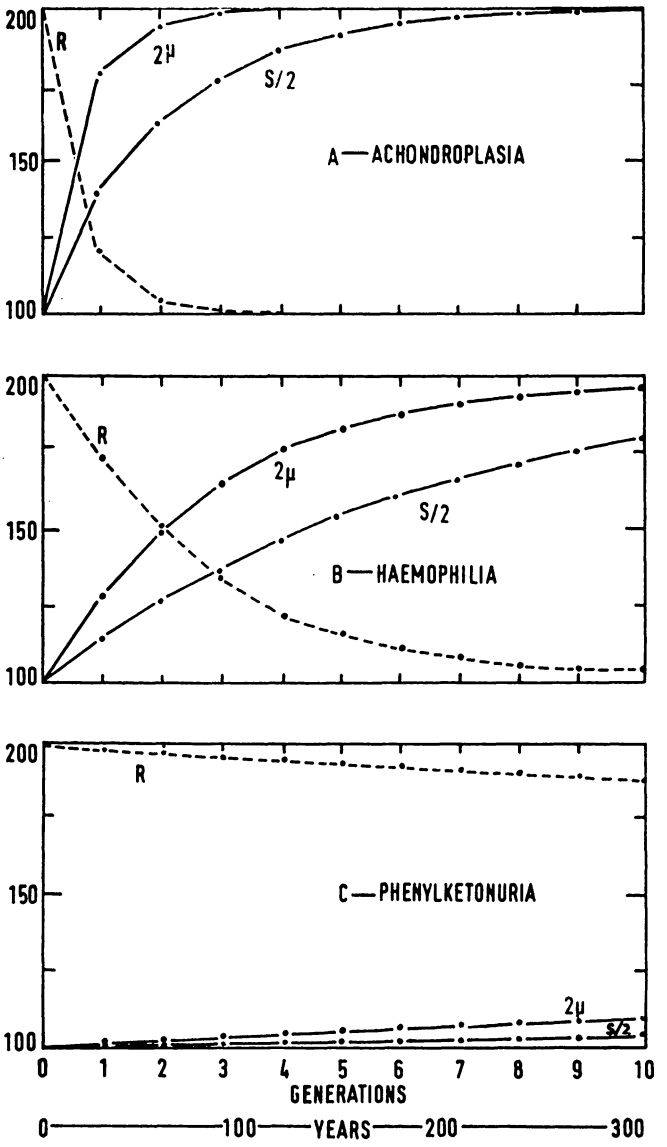
200
150
100
R
2μ
S/2
A—ACHONDROPLASIA
200
150
100
R
2μ
S/2
B—HAEMOPHILIA
200
150
100
R
C—PHENYLKETONURIA
2μ
S/2
0 1 2 3 4 5 6 7 8 9 10
GENERATIONS
0 100 YEARS 200 300

FIGURE 1

140 genes, 60 per cent, or eighty-four are transmitted and with another eighty new mutants give $84+80 = 164$ affected individuals in the second generation. The rise is slower and some nine or ten generations, or 250 to 300 years, are needed before it is virtually complete. It is noteworthy, however, that if, when the new equilibrium is attained, the original situation is restored (by halving the doubled mutation rate or doubling the halved unfitness as the case may be) the speed of return to the original equilibrium is the same in both cases and is equal to the speed of rise with a doubled rate of mutation.

With haemophilia and even more so with phenylketonuria, most of the causal genes, being recessive, are hidden in heterozygotes where they do not display their effects. They are thus transmitted to the offspring without any loss, only the minority of genes in affected individuals suffering loss in transmission because of the unfitness they cause. So reducing s has a less rapid effect than in the case of achondroplasia. The balancing increment added by mutation is also relatively smaller so that raising the mutation rate also has a less rapid effect. In all cases, however, the effect of doubling μ is a more rapid rise than is brought about by halving s, even though once reached the new equilibrium is the same in both cases. The rate of rise of a condition, like phenylketonuria, caused by a recessive gene is very slow. With a doubled mutation rate, ten generations or some three centuries would see an increase of only about 10 per cent and with a halved unfitness an increase of only 5 per cent.

We have seen how raising the mutation rate or alleviating the condition will bring about an increase in the frequency of the condition. How can this frequency be reduced? Lowering the mutation rate would of course effect such a reduction, but while we can raise mutation rates we do not yet know how to decrease them. Increasing the unfitness would in principle achieve the same end, but with s at 0·8 to 1·0, as in the cases of the disabilities we have been considering, no reduction or only a very small one, is possible by the most extreme measures such as sterilization, even if we were prepared to contemplate them. A reduction to 80 per cent in incidence

is the most that could be achieved with achondroplasia, until means are found of lowering the mutation rate.

With recessive genes, other possibilities are, however, open. Of these two may be mentioned, both of which depend on recognizing heterozygotes. If these could be recognized and persuaded or discouraged from reproducing or always mated to individuals homozygous for the normal gene, the incidence of the disability would be reduced. In the former case it would be reduced to zero, but in the latter case affected individuals would still appear because of mutation in the normal mates of the heterozygotes. The reduction would nevertheless be very great: in fact it would be the present incidence multiplied by about twice the square root of the mutation rate. With phenylketonuria, where f is now about 1/40,000 and $\sqrt{\mu}$ about 1/200, the reduced frequency would be about 1/4,000,000.

The two procedures would be alike in sharply reducing the incidence of the disability but they would differ in their effects on the frequency of the gene itself as opposed to the disability it causes when homozygous. Prevention of heterozygotes from breeding would lower the number of causal genes in the population as well as the number of affected individuals; but the mating of heterozygotes to homozygous normals would lead to a steady rise in the frequency of the gene, hidden behind the cloak of its recessiveness in the increasing number of heterozygotes.

Individuals heterozygous for some genes, like that for phenylketonuria, can already be recognized by appropriate tests and we may expect more of them to become recognizable. Heterozygotes mated to one another can, of course, always be recognized when they produce an affected child. If such a couple were to curtail their families as soon as an affected child was born one-quarter of them would have only one child, three-sixteenths would have only two (an unaffected followed by an affected child), nine out of sixty-four would have three (two unaffected followed by an affected child) and so on. The number of affected individuals would be thus reduced by a proportion depending on the size of the family that would have been in prospect had no affected child appeared. With prospective families of one, obviously no reduction would

ensue. With prospective families of two, the numbers of affected individuals would be reduced to 7/8 (= 0·875), with prospective families of three to 37/48 (0·771); with prospective families of four to 175/256 (= 0·684), and so on. This breeding test, if it may be so styled, thus has its effects but these become striking only where prospective families are relatively large.

POLYMORPHISM

Not all variants are maintained in populations by the balance of mutation and selection. The classical case of a disability which is not so maintained is sickle-cell anaemia which in some negro tribes occurs at frequencies far too high to be attributable to mutation pressure and which Allison [1, 2] has indeed shown to be determined by the balance of two selection pressures. Individuals homozygous for the sickling gene have abnormal haemoglobin, suffer from anaemia, develop abnormally and show a fitness of only about a quarter of the normal. Their haemoglobin confers on them, however, resistance to a malarial infection. Individuals homozygous for the normal allele have normal haemoglobin and develop normally but are subject to the malarial infection and so show reduced fitness in malarial regions. The heterozygotes have both kinds of haemoglobin and enjoy the best of both worlds: they both develop normally and are resistant to the malaria. The heterozygotes are thus fitter than both homozygotes, with the result that both the sickling gene and its normal allele are held in the population with frequencies depending on the balance of selective disadvantage of the two homozygotes. Where u is the frequency of the normal gene and v $(= 1-u)$ that of the sickling gene in the population, with s_N the unfitness of the normal homozygote and s_A that of the anaemic homozygote, when equilibrium is struck

$$us_N = vs_A.$$

This can be rewritten as $v = \frac{s_N}{s_A} u$

so that the frequency of the sickling gene will rise as the ratio

of unfitness of the normal homozygote to the unfitness of the sickler homozygotes falls, and *vice versa*. In other words the proportion of the population suffering from sickle-cell anaemia will depend not only on the level of unfitness which their condition brings about in them, but also on the incidence of malaria and the unfitness it causes in the normal homozygotes (Figure 2). Sickle-cell anaemia will become more common if the ravages of malaria increase and will diminish in its incidence as malaria is brought under control. Removal from their malarial homelands to a malaria-free country will bring about a reduction in sickling and this appears to have happened among the Negroes of the U.S.A.[1] Equally the elimination of the malaria carrying mosquitoes will reduce the incidence of

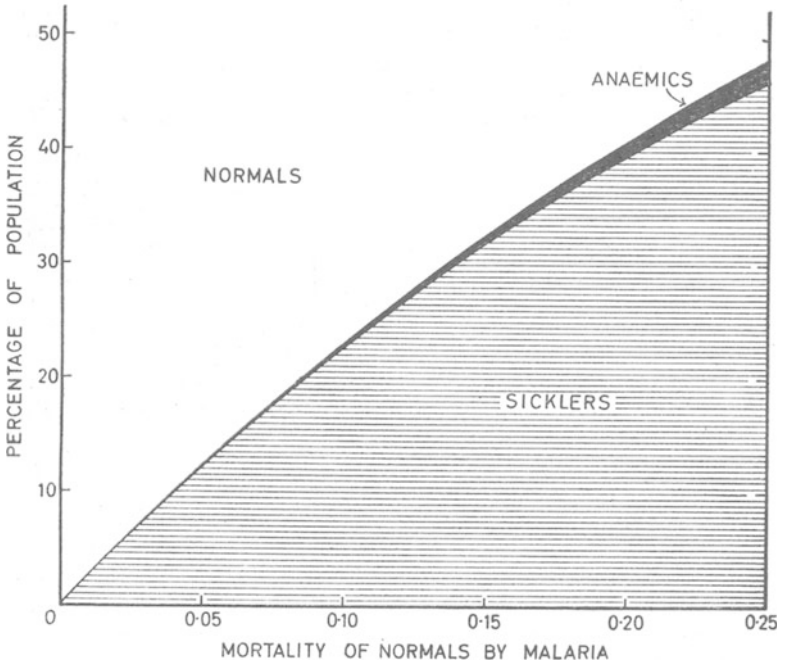

FIGURE 2. The frequencies of anaemic homozygotes, sickler heterozygotes and normal homozygotes in populations at equilibrium for the sickle-cell gene in relation to the loss of fitness brought about by malaria in the normal homozygotes (reproduced from Mather, 1964).

the sickling condition as well as that of malaria. The invention of DDT offered the means of controlling this anaemia!

How quickly will a change in balance of the selective forces produce its effect? Figure 3A shows the changes in frequency of the three types, anaemic homozygotes, sickler heterozygotes and normals, in a population in which 4·4 per cent of the children are born to be anaemics and 33 per cent are heterozygotes (frequencies which are at equilibrium when the fitness of the normal homozygotes is reduced to 0·8 by malaria), consequent on the elimination of malaria and hence the raising of the fitness of the normal homozygotes to equal that of the heterozygotes. The frequencies shown in the figure are not those among the children at birth but among the adults after three-quarters of the anaemics have been eliminated. After ten generations the anaemics have been reduced among the adults from 1·1 per cent to just under 0·2 per cent and the heterozygotes from 34 per cent to 15 per cent. The change is thus not a very rapid one: after ten generations, or something like 250 to 300 years, there is still a long way to go before the gene and with it the sickling condition is eliminated. Furthermore the speed of change is falling. Not, of course, that the gene would in any case be completely eliminated. Presumably it continues to arise from its normal allele by mutation and while mutation pressure can be neglected when we are dealing with the balancing effects of the two selective forces, it would ensure that the gene was held in the population when one of these forces had been eliminated. In fact as the gene became rare it would tend towards the equilibrium determined by the balance of mutation and the remaining force of selection, as discussed in the previous section.

The change in a population where s_N was not reduced to zero but halved is shown in Figure 3B. Here the approach is not to reduction of the sickling gene to the low level maintainable by mutation, but to the new equilibrium determined by the ratio of the changed s_N to s_A. After ten generations there is still some way to go, though not so far as in the population shown in Figure 3A. This same pattern of change would follow of course whenever the ratio s_N/s_A was halved, whether this was achieved by halving s_N, doubling s_A, quartering s_N

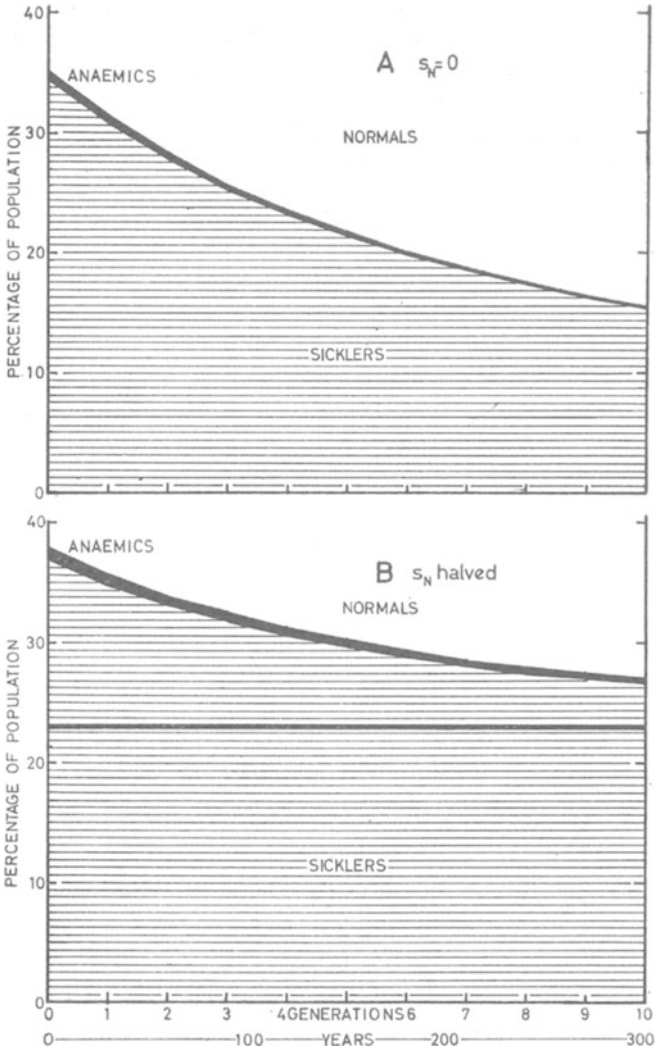

FIGURE 3. A. The changes in the frequencies of anaemics, sicklers and normals in a population initially at equilibrium with malaria reducing the fitness of normals by 0·2, when the unfitting effect of malaria is abolished. The approach is to a new equilibrium at which both anaemics and sicklers would be virtually absent.

B. The corresponding changes in the population where the unfitting effect of malaria is not abolished but halved. The approach is to new equilibrium frequencies shown by the heavy line horizontally across the diagram.

while halving s_A or by any other simultaneous adjustment of s_N and s_A in the right proportion. It is not the strengths of the individual forces of selection that matter, but the ratio that they bear to one another.

Variants maintained at reasonably high frequencies in the population by a balance of selective forces may be described as polymorphisms. Unlike sickle-cell anaemia, most human polymorphisms are not associated with conditions that result in marked reduction of fitness. The best known and most widely studied of the human polymorphisms are the blood group systems, especially the ABO system. Evidence is coming available associating these blood groups differentially with certain diseases.[7, 10] There can thus be little doubt that selection is involved in their maintenance in human populations, but just what these forces of selection are and how they interact, or even whether they maintain the polymorphism by endowing heterozygotes with an advantage or act in some other way, is not yet known. Of certain things, however, we can be sure; that whatever the forces of selection may be, they are being changed by medicine, preventive as well as curative; that these changes will result in corresponding readjustment of the polymorphism; and finally that, as with sickle-cell anaemia, the readjustments will be relatively slow in their progress. The blood group polymorphisms can still be used as short-term indications of human migration and crossing, even if in the long term their shapes are determined by selective balance rather than racial origins.

CONTINUOUS VARIATION

The variants considered so far have all been of the kind leading to clear differences by which individuals can be classified into sharply distinct categories. Much of our variation is, however, quantitative rather than qualitative, with the character showing every degree of expression between wide limits (Figure 4). Such continuous variation is partly non-heritable, reflecting the action of external agencies, and partly heritable, the genetical differences being, however, not simple as in the cases considered earlier but characteristically complex

in that they reflect the operation of polygenic systems. The genetical equilibria underlying continuous variation are therefore incapable of representation by the simple relations that suffice where inheritance is less complex. Nevertheless natural selection can be seen to be acting on continuous variation and changes in the forces of selection will produce changes in the distribution of the character in the population just as they will in the proportions of individuals falling into the classes distinguished in the simpler genetical situations.

The frequencies of the different expressions of a continuously

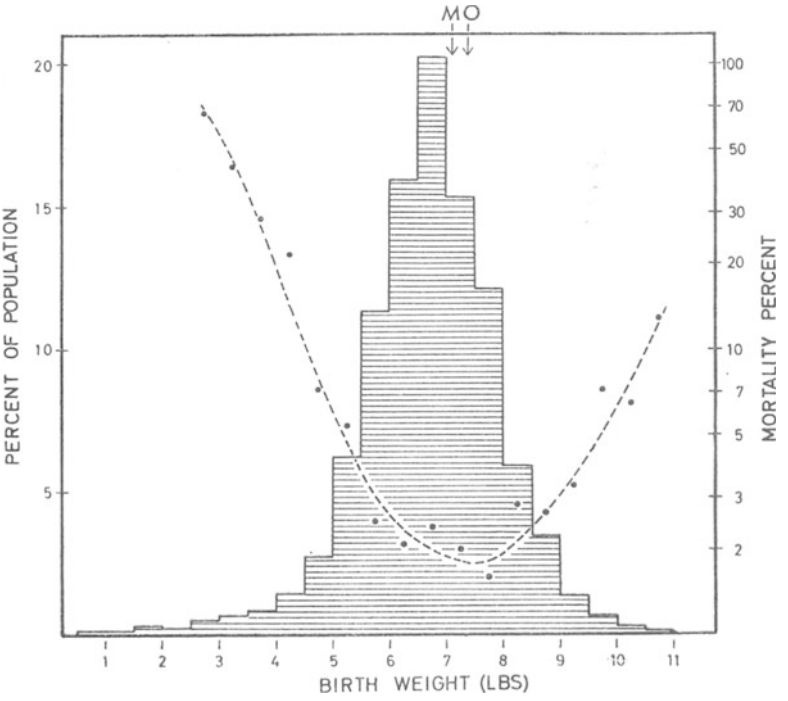

FIGURE 4. The distribution of birth weight (hatched histograms) and the rates of early mortality (broken curve) among 13,730 children. The percentage mortality is set out on a logarithmic scale for ease of presentation, the values actually observed for the classes of birth weight being represented by the points to which the curve is an approximation. M marks the mean birth weight and O the birth weight associated with the lowest mortality. (Reproduced from Mather, 1964. Based on data from Karn and Penrose, 1952.)

varying character characteristically follow a normal distribution or something akin to it, and for our present purposes we may consider selection as capable of acting on the character in two ways (Figure 5). The forces of selection may favour the central expressions of the character at the expense of the more extreme. The selection may then be said to be stabilizing since it tends to stabilize the mean expression, which is of course the most-favoured or optimum expression. Or the forces of selection may favour some expression other than the mean. Selection may then be described as directional since it will tend to move the mean expression towards the optimum with which, by definition, it does not coincide. This action of directional selection in shifting the expression of the character

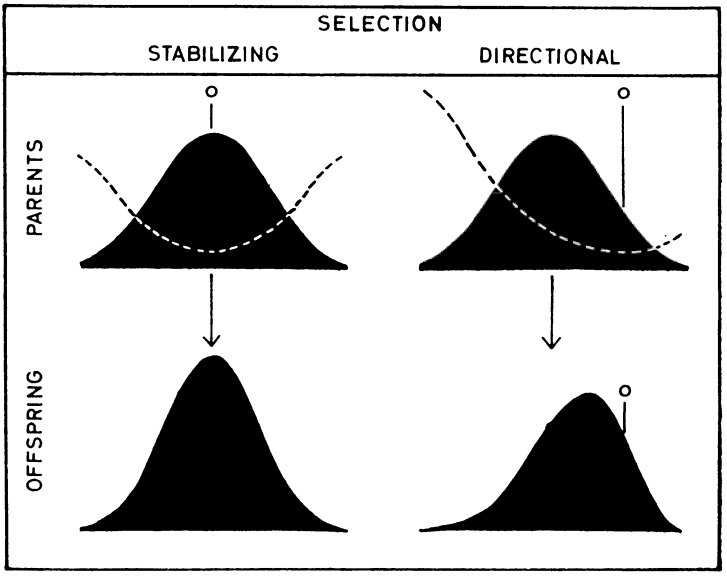

FIGURE 5. Stabilizing and directional selection. The solid figures represent the distribution of the character in the population and the broken curves the relative intensity of selection against the various expressions of it. O is the optimum expression, suffering the least action of selection. In stabilizing selection the average expression is the optimum and the action of selection is to narrow the range of expression. In directional selection the average is not the optimum and selection tends to shift it towards this optimum. (Reproduced from Mather, 1964.)

is well known from experiment and has been the basis of our breeding of improved strains of domestic animals and plants as well as of natural changes in evolution. Stabilizing selection is of course also practised by plant and animal breeders to maintain their strains once they have been achieved.

A clear case of stabilizing selection in man is provided by Karn and Penrose's [5] data on birth weight in relation to neonatal mortality (Figure 4). The frequency distribution of birth weight approximates to a normal curve with mean just over 7 lb (taking boys and girls together). Neonatal mortality is at its lowest for children with birth weights in the central range, the optimum weight being perhaps just a little above the mean but still less than 8 lb. Mortality rises sharply as weight moves away from the mean in both directions. The result of this selection is that the surviving babies show only 80 to 90 per cent of the variation (according to the way it is measured) of the babies born, but the mean is hardly affected. The genetical component of these differences in birth weight is complicated by dependence partly on the mother's genotype as well as on that of the child itself, but in total it would seem to account for some 40 per cent of the variation.[8] Assuming that selection does not distinguish between differences of genetic and non-genetic origin, the heritable variation will be reduced by 10 per cent or more, just as in the total variation.

Now selection will be weakened by any medical treatment which is successful in saving babies deviating from the mean in either direction, who in its absence would have died. If the treatments, though differing in nature for small and large babies are equally successful in saving their lives, the mean birth weight of the survivors will not be altered, but their variance will be less reduced by selection, that is it will be higher than in the absence of treatment; and if treatment were successful in wholly eliminating the differential mortality among babies of more extreme weight, no loss of variance after birth would occur at all. Nor would its effects stop at this point, as we can see if we look at a simple example.

Let us consider a system comprising two gene-pairs, A-a and B-b, which are unlinked, affect the same character, are equal and additive in their effects and show no dominance.

We assume that the alleles A and a are equally common in the population as are B and b, and that mating is at random. The character can show five degrees of expression, the degree associated with any genotype being proportional to the number of capital letters in that genotype. Taking the mean expression as the origin, aabb will have expression —2: Aabb and aaBb expression — 1; AAbb, aaBB and AaBb expression 0, AABb and AaBB expression 1; and AABB expression 2. Furthermore, simple Mendelism tells us that these five classes will respectively constitute 1/16, 4/16, 6/16, 4/16 and 1/16 of the population in the absence of selection. But let us suppose that stabilizing selection is acting so that only a proportion $1-s$ of the two extreme genotypes survive and $1-\frac{1}{2}s$ of the two intermediate classes, the central class having a fitness of 1. The variance of the survivors will obviously be reduced by an amount depending on the value of s.

There would, however, be a further effect expressed in the next generation, for the four possible types of gametes, AB, Ab, aB and ab, would not be produced in equal number by the selected population. The balanced types Ab and aB, originating preponderantly from the central classes, would be more frequent than the extreme types, AB and ab. There would thus be a short fall in the proportions of the next generation with the more extreme genotypes, and hence in the more extreme expression of the character, even before selection had begun to operate. So under stabilizing selection there is a dual reduction in the variation expressed by the survivors; it is reduced by the immediate action of selection in the generation under selection and also by the effect of past selection in distorting the relative frequencies of the different genetic classes of gamete. And if selection is relaxed in a population which has come to equilibrium under its stabilizing action, the variation will climb above that displayed even by the individuals of past generations before selection had acted on them.

The magnitudes of these two effects of selection will obviously depend on s. They are shown in Table I for four values of s. At all levels of selection, the long term effect expressed by the reduction of V_I below the value of 1 is somewhat less than the immediate effect of selection on the generation in question

which is expressed by the reduction from V_I to V_S. Now this latter reduction corresponds to the 10 per cent or so reduction in variation that we observed selection to be producing in birth weight. It is about 10 per cent in our simple model when $s = 0{\cdot}25$. So, if this model is any guide to the more complex reality of birth weight, abolition of the stablizing selection would raise the variance of surviving babies by nearly 20 per cent and the variance of the babies at birth by rather less than half this amount. There would be more babies showing extreme birth weights and to this extent an increased demand for special medical care; but save at the very extremes, the

TABLE I

s	0·25	0·50	0·75	1·00
V_I	0·93	0·86	0·78	0·70
V_S	0·84	0·66	0·49	0·32

The effects of stabilizing selection at equilibrium in a simple two-gene case, where V_I is the variance of the distribution at conception and V_S the variance after selection has acted, both being expressed as proportions of the variance which the population would show at equilibrium in the absence of selection.

numbers falling in the different classes of birth weight would not be greatly changed.

The conclusions derived from such a simple model are obviously to be treated with reserve. They can be varied in detail by altering the strength of the selection assumed to operate on the intermediate classes relative to the extreme and central groups; but the changes so brought about are quite small. Furthermore, taking a similar model but involving three gene-pairs gives surprisingly similar values of V_I and V_S at comparable levels of selection. More complex cases have not yet been investigated, but it would appear that the simple case considered may well present a picture not seriously misleading, provided that the assumption of no linkage is valid. Should there, however, be an excess of repulsion linkages—and stabilizing selection must favour such a situation—the relaxation of selection could have effects markedly larger than those that our model suggests.

The variance is raised but the mean is unchanged if stabilizing selection is relaxed symmetrically, that is equally on both sides of the distribution. If, however, relaxation is asymmetrical, a directional element appears in the selection and the mean must tend to change accordingly. It will shift to an extent proportional to the asymmetry and will continue to shift so long as the asymmetry of selection continues to maintain the directional pressure, provided the genes mediating the expression of the character have no other effects on fitness and are not closely linked with further genes affecting fitness by a different path. If on the other hand heterozygotic genotypes, which tend to be associated with more central expression of the character, are favoured over homozygotes by reason of either pleiotropic effects or linkage, the distribution will resist the directional selection and the mean will change to a disproportionately small extent, if at all, as Penrose [9] has pointed out in relation to intelligence. We may note, however, that such an assumption does not deny the possibility of the character being changed by alteration of the forces of selection; it merely shifts the emphasis on to the forces of selection from which the heterozygous advantage stems. These may not be the forces observable as acting on the character directly, but, however they are exerting their effects, any alteration in them will result in a corresponding shift of the character. Whatever the selection that is acting, and however it is doing so, whether directly or indirectly through genetic correlations springing from pleiotropy or linkage, changes in them will inevitably result in corresponding readjustments of the character and its distribution in the population though, if the action is indirect the readjustments may come as a surprise to us in both their kind and their magnitude.

CONCLUSION

All human populations are genetically variable and all are under the action of natural selection. Their characteristics reflect the interplay of this variation and these forces of natural selection, and characters tend towards genetical equilibria reflecting the balance between the agencies of variation and

the forces of selection. The equilibria may be of various kinds depending on the mode of inheritance of the character and the effects of the causal genes on the fitness of individuals carrying them. Any change in the agencies of variation, and especially in the forces of selection, must lead to shifts in the equilibria, and hence in the frequency distribution of the characters in the population, though in general the readjustments will be slow in coming about. Natural selection is acting on us all; medicine, and indeed many other human activities, are changing these forces; and these changes will inevitably, even if in general only slowly, alter the genetical constitutions of our population.

DISCUSSION

PROFESSOR MATHER made two points in reply to comments and queries:

(i) The use of techniques, such as selection, for changing the genetical constitution of a population would raise questions requiring careful consideration and, no doubt, lengthy discussion. One point might, however, be made at once. With other species we generally knew at what we were aiming: for example with dairy cattle we knew that we wished to raise the yield and quality of the milk. The criterion of merit by which one chooses parents for breeding is then clear. With man, however, we must expect divergences of view about aim. Even if a manipulation were possible technically, we still had the question of deciding for what purpose we would wish to use the technique. This could hardly fail to cause more dispute among the public than even discussion of the technique would cause among the geneticists.

(ii) When one adopted a quantity like s as a measure of unfitness one assumed it to be a comprehensive quality, covering for example, not merely biological infertility, but infertility arising from social, psychological or other causes also. From the point of view of the genetical consequences it did not matter for what reason children were not born: whatever the reason, the genes they would have carried will have been lost from the population just the same.

REFERENCES

1. ALLISON, A. C. 1955. Aspects of Polymorphism in Man. *Cold Spr. Harb. Symp. quant. Biol.* **20**, 239.
2. ALLISON, A. C. 1959. Recent Developments in the Study of Inherited Anaemias. *Eugen. Quart.* **6**, 155.
3. CARTER, C. O. 1961. The Inheritance of Pyloric Stenosis. *Brit. med. Bull.* **17**, 251.
4. CARTER, T. C. 1957. Ionising Radiation and the Socially Handicapped. *Brit. J. Radiol.* **30**, 641.
5. KARN, M. N. and PENROSE, L. S. 1952. Birth Weight and Gestation Time in relation to Maternal Age, Parity and Infant Survival. *Ann. Eugen. Lond.* **16**, 147.
6. MATHER, K. 1964. *Human Diversity*. Edinburgh. Oliver and Boyd.
7. MOURANT, A. E. 1959. Human Blood Groups and Natural Selection. *Cold. Spr. Harb. Symp. quant. Biol.* **24**, 57.
8. PENROSE, L. S. 1954. Some Recent Trends in Human Genetics. *Proc. 9th Int. Cong. Genet. Caryologia* Suppl. Vol. 521.
9. PENROSE, L. S. 1955. Evidence of Heterosis in Man. *Proc. roy. Soc. B.* **144**, 203.
10. SHEPPARD, P. M. 1959. Natural Selection and some Polymorphic Characters in Man. In *Natural Selection in Human Populations* (p. 35). Ed. D. F. Roberts and G. A. Harrison. London. Pergamon Press.
11. STEVENSON, A. C. 1961. Frequency of Congenital and Hereditary Disease with Special Reference to Mutations. *Brit. med. Bull.* **17**, 254.

INFECTIOUS DISEASES AS SELECTIVE AGENTS

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ALTHOUGH we may rightly be proud of the wonderful achievements in present day biology, we must realize that the main trends of thought and investigation in this science are still proceeding along the tracks marked by two fundamental discoveries of the last century, namely the cellular structure of organisms and evolution by natural selection.

In fact scientific investigation on selection only started about sixty years after the clear and complete statement made by Darwin of the role of natural selection as a directing force in evolution. During the decade 1920–30, three famous biologists, R. A. Fisher, J. B. S. Haldane and S. Wright, pointed out that selection can be investigated and measured almost as a physical force by means of experiments or observations properly devised to test theoretical models worked out in mathematical terms. The papers by Hardy[19] and by Weinberg[40] were ' rediscovered ', and the principle now commonly known as the Hardy-Weinberg law became the basis of all evaluation of selective forces. R. A. Fisher's book, *Genetical Theory of Natural Selection*[15] laid the foundations of the study of evolution on the factual basis newly provided by genetics.

During the subsequent decades great progress was made both in expounding the theoretical foundation and in testing the effects of selection in animals, plants and micro-organisms.

Only recently, about a decade ago, man came into the picture. Man, of course, has always been foremost in the thoughts of geneticists and evolutionists. Darwin himself, as is well known, after a brief but significant mention in the *Origin of Species* (1859), devoted a whole book to the problems of human evolution, *The Descent of Man and Selection in Relation to Sex* (1871).

Shortly thereafter—it is perhaps superfluous to remind you in this place—a movement was started by Galton, which led

to the establishment of eugenics as an autonomous discipline. It is well known that the original optimistic hopes as to the power of eugenics in ameliorating the lot of mankind have in the course of time gradually diminished, when confronted with the complex and difficult situation which was revealed by genetic studies in general and, especially, in the case of man.

It has been a common saying for a number of years that man is a very poor subject, quite unsuitable for genetical investigation. Luckily enough, later developments proved the contrary and we now realize that man represents very good material for several kinds of genetical investigations, including studies on population genetics. Thus the hope of succeeding in discovering selection at work, of grasping at least some of the mechanisms underlying human evolution appears to-day much better founded that one could have foreseen twenty years ago. In fact we may say that we have already acquired some good and firm landmarks in this field.

Geneticists of to-day have little doubt that mankind as it is now, with its interracial and intraracial variability, is the product of an evolutionary process controlled by external factors which have acted and are still operating as selective agents. They work on the possibilities offered by the variability of human genotypes. Clearly enough, as soon as man succeeded in making himself more and more independent of natural conditions by building and modelling his own artificial environment, the selective agents gradually became different: some of the old ones ceased to work, while new ones became active.

For the experimentally minded geneticist of to-day it is essential to demonstrate, and possibly to measure, at least some of the selective factors which are active in man. If he succeeds, this would put an end to merely speculative discussions pro and contra the effects of selection; it would give the key to a deeper insight in the causes which originated diversities between individuals, populations and races; and, last but not least, would probably open up the possibility of grasping some points of important practical—and eugenic—value.

If we think of the work of selection in the old traditional way—the elimination of the unfit by death—there is little doubt that diseases which are able to kill individuals early in their life, before or during the reproductive period, are strong selective agents. If on the other hand we consider selection, more properly, as differential reproduction, it is clear that any kind of illness which although not lethal, lowers the reproductive potentiality, is a selective agent. In both cases, needless to say, we must suppose that there is a genetic variability in susceptibility to disease and in the possibility of recovery. This assumption is supported by many facts known to pathologists and anthropologists.

Darwin himself was perhaps the first to recognize this principle while searching for the possible causes of differentiation in such racial characters as are well known to anthropologists. In Chapter VII of the *Descent of Man*, he first points out that among other distinctive characters of races, differences in liability to certain diseases must be taken into account. Later on in the same chapter, when discussing the origin of human races, he notes that apparently skin and hair colour sometimes show a correlation with total immunity towards poisons extracted from plants and towards infection by certain parasites. With his usual cautious attitude Darwin expresses this conjecture in dubitative form and explains how he had endeavoured, with but little success, to ascertain how far it holds good. In the spring of 1862 he obtained the permission of the Director-General of the Medical Department of the Army to transmit to the surgeons of the various regiments on foreign service a questionnaire asking whether in Europeans living in the tropics there is any relation between the colour of their hair and their liability to tropical diseases such as malaria, yellow fever or dysentery. Darwin received no returns.

Thus the question as to the possible action of infectious diseases as selective agents was put clearly enough by Darwin.

The attempts to find instances of selection in man were repeated from time to time in the post-Darwinian period, but we must come to recent years, as I said, to find some penetrating and successful investigations.

As F. Vogel [37] points out, several authors between 1924 and 1928 tried to find out whether there was a relationship between the ABO blood group and syphilis. The data, which had been reconsidered and statistically worked out by Vogel, Pettenkofer and Helmbold,[38] do not show any indication that people belonging to any one of the four groups are more susceptible to infection by *Treponema*. They show, however, that with antiluetic treatment Wasserman reaction becomes negative after a shorter time in patients belonging to groups O than in patients belonging to the other groups.

The first precise suggestion of a selective mechanism due to an infectious disease was put forward by J. B. S. Haldane,[18] who suggested a possible explanation of the high frequency of heterozygotes for thalassaemia in certain populations. Thalassaemia is due to a gene (*Th*), which in homozygous conditions is lethal: it determines Cooley's disease which causes death of practically all individuals before reproductive age. The heterozygotes (*Thth*) show only a comparatively mild form of anaemia (Thalassaemia minor), fully compatible with life. Haldane supposed that in regions in which malaria is endemic the heterozygotes might be at advantage in respect to normal homozygotes, because they might be less susceptible to infections by *Plasmodium falciparum*, the agent of pernicious tertian malaria. The advantage of heterozygotes over both homozygotes (a fact known as *heterosis*) would be sufficient to counteract the constant loss of the thalassaemia gene through the production at every generation of a certain number of lethal homozygotes. This mechanism would maintain in a population a variability which otherwise would rapidly disappear. This condition is well known to geneticists under the name of *balanced polymorphism*, and its theoretical aspects and factual basis in animals and plants have been discussed, among others, by E. B. Ford [16] and Julian Huxley.[21]

The first demonstration of Haldane's assumption, however, did not come from thalassaemia (to which I will come back later) but on another blood disease, *sickle cell*. It is well known that this disease depends on a minute anomaly in one of the four poly-peptide chains of the adult haemoglobin (beta chain), that is, to a single amino-acid substitution, among the few

hundreds amino-acids which form the Hb molecule.[20] The genetic control of this anomaly is due [27] to a single autosomal gene pair. The situation here is slightly different from thalassaemia, because the homozygotes *Ss* do not necessarily die before reproduction. Their reproductive fitness has been estimated as 20–25 per cent of the normal. In spite of that it was found that the heterozygotes, showing the sickle-cell trait, but being otherwise normal, are very frequent in some populations. In American Negroes, where the character was discovered, heterozygotes are about 9 per cent, but in African populations their frequency may be as high as 30 per cent or even 40 per cent in some localities, while in others it is as low as 1 per cent. Since the highest incidence coincides with the zones where malaria is endemic, and since explanations based on a high mutation rate of locus S, or on genetic drift, are untenable for many reasons, the most probable explanation is the same as for thalassaemia, i.e. the selective advantage of heterozygotes, which would be in some way protected from malarial infection.

To Allison [2, 3] must go the credit of having examined the problem from all points of view and of bringing it to an experimental test. He found that among populations living in countries with high malarial endemicity, the percentage of heterozygotes for gene *S* was over 10 per cent, while in populations living in regions with low malarial infection, it is under 10 per cent. He found a statistically significant difference between *Ss* and *ss* children as to the frequency of malarial infection: it is lower in heterozygotes. He finally performed the well known and much discussed experiment of inoculating *Plasmodium falciparum* in fifteen normal individuals and in fifteen bearers of the sickle-cell trait, with the result that fourteen of the first group and only two of the second showed parasites in the blood. In subsequent papers Allison [4] showed that the sickle-cell trait is significantly more frequent in grown-up people than in children of the same population, and that—in agreement with former data of Hiernaux [20]—the fertility of the three types of crosses is different. The reproductive fitness of the three possible crosses, namely heterozygous by heterozygous, heterozygous by normal and normal by normal

being in the ratio: 0·203 : 1·195 : 0·951. Thus, in spite of some criticisms and exceptions (see Livingstone [24] for an explanation) the Haldane hypothesis seems well substantiated in the case of the sickle-cell gene.

I would like now to come back to the thalassaemia (beta-thalassaemia). This gene, which was discovered in America on people of Sicilian or Greek origin, has a diffusion area which in the Euro-Afro-Asiatic continent is largely overlapping with the diffusion of malaria.[23] It is particularly frequent in Mediterranean populations, sometimes together with *S*, sometimes without it as in most regions of Italy. Its distribution in Italy has been studied quite thoroughly, [8, 25, 26] especially by Silvestroni and Bianco.[32] The distribution of high frequencies of heterozygotes (from 10 to 30 per cent) is coincident with heavy malarial endemicity. In this case another possible explanation, namely the hypothesis of selective mating, was tested by Montalenti, Silvestroni and Bianco in 1953 and disproved.[27] High mutability of the *Th* locus and genetic drift must be discarded on several grounds, as in the case of the *S* gene. The ' ethnic ' explanation of Silvestroni, Bianco and Alfieri,[33] should also be discarded because it may well account for the penetration of the gene into a population, but not for its maintenance at a high level in spite of continuous elimination by lethal zygotes. Thus the only explanation left is the selection in favour of the heterozygotes in malarial regions, as assumed by Haldane.

The first investigations to test this hypothesis were by Ceppellini [11] and Carcassi, Ceppellini and Pitzus.[10] They found that in four Sardinian villages not far from each other, but two situated in the plain, that is in a formerly malarial region, and two on the mountains, a non-malarial region, the frequency of the thalassaemias (i.e. of heterozygotes for the gene *Th*) was very different, much higher (about 20 per cent) in the former than in the latter populations (about 4 per cent).

The correlation between ' malaricity ' and frequency of the *Th* gene in Sardinia was then extensively studied by a team of workers of the Istituto di Genetica of Rome, with other co-workers under the guidance of Professor M. Siniscalco. A third gene, however, came into the picture. This mutation

is responsible for glucose-6-phosphate dehydrogenase deficiency in the red blood cells. The gene is indicated by the symbol G6PD, shortened in $Gd^{(-)}$. It was discovered recently and it was found that the gene is responsible for the sensitivity of certain people to fava beans (being the cause of a disease known under the name of 'favism') and to primaquine (see studies by Sansone, Piga and Segni,[31] and Butler [9]). The $Gd^{(-)}$ gene, unlike the others mentioned so far, which are autosomic, is sex-linked,[13, 36] i.e. localized in the *X* chromosome.

The distribution of favism in Italy was investigated many years ago (1905) by Fermi and Martinetti. [14] It again shows a remarkable overlapping with the distribution of malaria. It was therefore decided to study the distribution of this gene in Sardinia where favism is a fairly frequent occurrence. Motulsky [28] has elaborated a method which enables the worker quickly to determine the genotype of an individual and thus makes it possible to screen fairly large populations.

In Sardinia, as in continental Italy, malaria has been completely eradicated since the end of World War II. Fortunately we have a survey made by Fermi [13] in 1938, which gives a measure of malarial morbidity in the years between 1929 and 1938. This greatly facilitated the work of Siniscalco and his collaborators.[34]

The results of surveys made in the years between 1958 and 1964 are presented in Table I. The samples of populations examined (except for some villages, in which a complete ascertainment was made) are represented by boys of school age. It is quite evident that both for *Th* and $Gd^{(-)}$ genes there is a negative correlation with altitude and a positive one with the intensity of malarial morbidity in former times. This is another strong, though indirect proof that malaria acts as a selective agent in favour of heterozygotes for the thalassaemia gene and of carriers of $Gd^{(-)}$ gene. Table I shows some anomalies in the correlation indicated. Most of them have an explanation. For instance, the population of the little island Carloforte is not a Sardinian one; it is composed of people coming from Liguria (Genoa), who migrated to Tunisia about four centuries ago and settled down in the island Tabarca.

In the eighteenth century they were given, by Carl Emmanuel III of Savoy, king of Sardinia, the island San Pietro, where they founded the town Carloforte. They settled down as a highly inbred unit, and they have preserved their language, habits and customs, with very little outbreeding until very recent times. In fact it was ascertained that the few people who showed either *Th* or *Gd* genes had received them from a Sardinian parent or grandparent.

Usini is a recently formed village, with probable infiltration from Catalogna. In many places (e.g. S. Gavino, Capoterra, Guspini, Senorbi, Sedilo, Arbus, Dualchi, Borore) the incidence of malaria was known to be very high, but at the time of Fermi's report a great deal of prophylaxis had already been used, and malaria was almost defeated. Some places located at a certain altitude, like Alà dei Sardi, have a comparatively high index of ' malaricity ' because the active section of the population used to spend a great part of the year working in the plain, where agriculture is possible, and still do so. Therefore we consider that, on the whole, the positive correlation between intensity of malarial incidence in recent times, and frequency of $Th^{(+)}$ and $Gd^{(-)}$ genes is proved.

The last mentioned gene is certainly less unfavourable than thalassaemia, because only a minority of its carriers may be subject to severe haemolytic crises if they eat fava beans. The mortality among people affected by favism has been recorded by several authors: the figures vary from 2 per cent to 18 per cent, before blood transfusion became a common practice.[31] Since not all the gene carriers eat fava beans and not all the carriers who eat them necessarily become ill (individual variability in response), it is clear that the gene is much less dangerous than thalassaemia.

An interesting consequence of these facts is the correlation between the frequencies of the two genes. As shown in the diagram published by Siniscalco, Bernini, Latte and Motulsky[34] in 1961, it is linear up to a certain value, beyond which the frequency of *Th* decreases, while $Gd^{(-)}$ continues to increase. The data collected recently and tabulated in Table I confirm this trend. It may be due to the fact that *Th* is more deleterious than $Gd^{(-)}$, so that the intensity of selection *against Th* is stronger

than that *against* $Gd^{(-)}$. A similar condition is found by Allison *et al.*[5] in a Greek population. Motulsky [28] found essentially the same kind of correlation between *S* and $Gd^{(-)}$ in Negroes, with the exception of a group of pygmies, in which the correlation was negative. Barnicot *et al.*[6], on the contrary, found an essentially negative correlation between *S* and *Th* among Greeks. The dynamics of two or more genes selected with various intensities by the same ecological factor have still to be worked out.

A different problem is the interaction between two non-allelic genes, an indication of which may be found in the frequency of their presence in the same individual. The available data for *Th* and $Gd^{(-)}$ (Table I) show that on the whole there is a significant excess of the bearers of the two genes. As a tentative explanation Siniscalco (private communication) offers either a higher selective advantage of the double heterozygote, or some kind of meiotic drive, as was found by Went [41] between *HbA* and *C* genes.

A further step in the investigation in Sardinia was made by considering the pattern of mortality before the age of reproduction, namely during the first year of life, in villages of the two types, i.e. with high and low incidence of malaria, before and after the eradication of the disease in Sardinia. From the data available at the Istituto Centrale di Statistica it is clear that in villages with a high incidence of malaria there has been a considerable drop of mortality after the anti-malaria campaign, whereas in non-malarial centres no such great variation is detectable. The vital statistics of the two kinds of centres before and after eradication are quite different (Siniscalco, unpublished data). Thus there is little doubt that malaria was the primary cause of the death of a number of people early in life, and therefore it can work as a selective agent. This is an answer to a question put by Neel [30] as to the actual efficiency of malaria as a killing agent.

The evidence accumulated so far in favour of the selective hypothesis is thus quite consistent. I believe that the data collected by Siniscalco and collaborators on Sardinian populations have a particular value in so far as they are drawn from a fairly homogeneous population, whose villages have

TABLE I

GENE FREQUENCIES OF THALASSAEMIA [Th (+)] AND GLUCOSE-6-PHOSPHATE DEHYDROGENASE DEFICIENCY [Gd (−)] IN 52 SARDINIAN VILLAGES

VILLAGE	Altit. m.	Malar. freq. Per cent	Gd (−) N.	Gd (−) Per cent	Th (+) N.	Th (+) Per cent	Gd (−) and Th (+) N.	obs.	exp.	diff.
Assemini	6	33	108	20·4	108	5·5	108	1	2·04	−1·04
Marrubiu	7	40	98	32·6	98	14·3	98	8	7·80	+0·20
Cabras	9	97	200	35·0	100	14·0	100	11	8·42	+2·58
Terralba	9	97	100	30·0	...	...	...	...	...	...
Carloforte	10	90	99	5·0	99	2·5	99	...	0·24	−0·24
Decimomannu	10	100	100	26·0	100	12·5	100	6	5·68	+0·32
S. Giusta	10	97	42	30·9	...	...	...	...	...	...
Pula	15	50	100	15·0	100	8·0	100	1	2·20	−1·20
Tortolí	15	90	50	16·0	...	...	...	...	...	...
Orosei	19	93	180	13·0	308	9·4	...	...	...	...
Torpé	24	98	100	22·0	100	19·0	100	5	6·76	−1·76
Irgoli	26	95	100	15·0	100	16·0	100	4	4·03	−0·03
Galtellí	40	96	175	12·0	235	10·6	...	...	...	...
Siniscola	42	55	195	11·3	97	12·2	97	2	2·44	−0·44
Barisardo	50	66	98	15·3	98	9·2	98	3	2·49	+0·51
Teulada	50	90	101	16·9	100	12·5	100	3	3·68	−0·68
S. Gavino	53	18	100	26·0	...	...	...	...	...	...
Capoterra	54	15	92	16·3	92	10·3	92	1	2·77	−1·77
Siliqua	66	50	100	26·0	100	11·0	100	3	5·09	−2·09
Vallermosa	70	60	86	20·9	86	10·5	86	5	3·37	+1·63
Monastir	83	50	94	23·4	94	10·6	94	6	4·16	+1·84
Nuraminis	86	100	100	25·0	100	8·5	100	4	3·88	+0·12
Villamar	108	21	100	23·0	100	9·0	100	6	3·76	+2·24
Guspini	137	10	99	28·2	99	17·2	99	12	7·90	+4·10
Domusnovas	152	33	100	22·0	100	9·5	100	3	3·78	−0·78
Gonnosfan	156	50	49	24·5	...	...	...	...	...	...
Ottana	185	83	72	8·0	72	14·0	72	4	1·38	+2·62
Tresnuraghes	257	50	86	12·7	86	15·7	86	...	2·88	−2·88
Usini	190	95	99	6·1	99	7·1	99	3	0·79	+2·21
Senorbi	204	12	101	24·7	101	6·9	101	2	3·17	−1·17
Sedilo	288	10	100	22·0	96	9·4	96	3	2·63	+0·37
Serrenti	207	50	100	21·0	100	12·5	100	4	4·59	−0·59
Arbus	311	5	95	35·7	95	16·4	95	9	9·31	−0·31
Abbasanta	315	73	97	18·5	92	11·4	92	3	3·44	−0·44
Dualchi	321	3	75	21·3	100	9·0	75	2	2·61	−0·61
Suni	333	85	98	14·3	100	12·5	98	4	3·13	+0·87
Lodé	395	90	820	28·2	820	13·8	820	65	55·00	+10·00
Gergei	374	66	92	18·5	92	6·6	92	2	2·10	−0·10
Borore	399	15	100	9·0	99	16·6	99	2	0·24	+1·76
Benetutti	406	50	100	9·0	100	6·0	100	2	1·00	+1·00
Bolotana	472	80	93	11·8	93	10·7	92	3	2·09	+0·91
Luras	508	78	100	7·0	98	11·5	98	2	1·42	+0·58
Lula	521	81	100	7·0	100	9·5	100	2	1·54	+0·46
Isili	523	17	100	9·0	100	8·5	100	1	1·38	−0·38
Bitti	549	18	193	5·1	196	6·1	193	3	0·96	+2·04
Lanusei	595	25	100	4·0	...	...	...	...	...	...
Alà dei Sardi	663	75	80	22·5	80	10·0	80	3	3·24	−0·24
Orune	745	80	97	6·1	97	7·2	97	...	0·79	−0·79
Gavoi	777	90	98	3·0	98	5·1	98	1	2·84	−1·84
Desulo	891	33	313	3·0	320	1·9	...	...	...	...
Tonara	935	10	148	4·0	102	2·4	...	...	...	...
Fonni	1·000	19	100	3·0	...	...	...	...	...	...

remained isolated from each other for a very long period of time. Other genetical and ecological factors being equal, it appears that the incidence of malaria is the only differential selective factor that could explain heterogeneity of Sardinian villages in respect of thalassaemia and G6PD-deficiency.

Data suggesting a similar protective effect by enzyme deficiency have also been accumulated by other authors on other populations. It might also be conceived that the selective agent is something other than *Plasmodium*, which would be closely connected with malaria; but this hypothesis, which was also considered by Haldane,[18] has lost ground in recent years.

Summing up: we have fairly good evidence that infection by *Plasmodium falciparum* selects for at least three genes slightly or heavily deleterious: *Gd*, *S* and *Th*. Probably other abnormal haemoglobins, e.g. haemoglobin C, are also selected for.[39] The mechanism underlying this process should be similar : in all cases the biochemical conditions of the red blood cell, which is the ultimate host of *Plasmodium*, is altered and it is conceivable that the parasite does not find conditions favourable to its nutrition and development in abnormal blood corpuscules. In the case of $Gd^{(-)}$ we know a little more. Plasmodia require glutathion for growth *in vitro*; enzyme deficient red cells have a diminished content of reduced glutathion. This might be the reason why the parasite does not develop well in such cells, and hence the protective effect of enzyme deficiency.

For infectious diseases other than malaria we have only scanty and at the moment inconclusive data. Perhaps the best case is that of plague and ABO groups, which has been worked out by Vogel and collaborators.[38] The frequency of O

TABLE I

The observed number of Gd (−) Th (+) individuals tends to exceed the expected one calculated on the basis of the gene frequencies found in each village. When the data from all villages are pooled together the difference becomes highly significant (2·57 times its sampling variance) and points to the possibility of a higher fitness of the combined genotype.

The absence of a figure in the table (...) means that for one reason or another the corresponding data for that particular village were not available.

gene is higher in regions in which there is no record of plague epidemics (American Indians, Australia, Polynesia), lower where plague is or has been endemic until recently, or where there have been epidemics in recent times. Vogel found that the specific agent of plague, *Pasteurella pestis* contains H antigen, the same as is present in *O* blood. Patients belonging to *O* group would therefore be able to produce less anti-*H* substance than those of other blood groups, and would therefore have a heavier manifestation of the disease.

According to the same author vaccination against smallpox would produce a strong anti-*A* serum. The blood of *A* and *AB* individuals would contain little or no anti-*A* serum; therefore one should expect that gene *A* would be selected against smallpox.

The idea that infectious diseases are effective factors of selection in man—and, of course, in animals as well—is now substantiated by conclusive proofs in a small number of cases and by more or less strong indications in others. It is certainly worth while to extend investigations to other cases, in order to get a more complete picture.

Needless to say, if we think of the diffusion of infectious diseases in old as well as in recent times, of the great epidemics of plague, smallpox, syphilis, tuberculosis, influenza, etc., of the endemicity of malaria, trypanosomiasis, yellow fever, etc., we realize how great their influence has been in controlling the present composition of the gene pool of different populations. The effects of the introduction of new diseases into populations which did not have them before, and therefore had only a few resistant genotypes, are well known in several cases, such as in many populations of Polynesia. The terribly high mortality, which entirely destroyed some populations or reduced them to a very small number, is the most dramatic picture of this kind of selection, which operates drastically by killing the unfit.

We may now turn back for a moment to the point raised by Darwin: Is there any correlation with genes for resistance to diseases and anthropological character? There is little doubt that there is. Blood groups are anthropological characters, and, besides the example of the plague which I have

mentioned, there is ever-increasing evidence that blood groups are not neutral characters, but subject to selection.

But external characters may also be connected with genes, or genotypes controlling primarily microscopic traits. For example Siniscalco, Montalenti, Silvestroni and Bianco [35] were able to show that thalassaemia has some effect on the pattern of some body measurements, mainly on weight and stature. Probably racial characters, to which it is not easy to attribute a selective advantage, are the visible expression of genes, or genotypes whose selective value is due to phenotypical manifestations concealed from the human eye, a well known fact in animals and plants.

The examples I have quoted in a very schematic way, deliberately ignoring many interesting details and complications, are, I think, the best known and more elaborated cases in the field of natural selection in man, in which, as Haldane says, our ignorance is enormous. A great deal of further study on these cases is still wanted, especially on the quantitative side, in order to measure more accurately the actual fitness of different genotypes. However, even so, I consider them extremely significant, because they give consistency to the assumption that for some tens of thousands of years infectious diseases may have been one of the major factors operating in human populations. The facts summarized here also open the way to several interesting considerations both from a biological and from a social point of view. I will mention a few of them.

From the biological viewpoint it is interesting to note that in different areas of the world the same selecting factor—malignant malaria—apparently picked out different genes. For instance in Italy thalassaemia was favoured, haemoglobin *S* is rare, in some places extremely rare, while in other Mediterranean and in African populations it is very frequent. The distribution of haemoglobin *C* according to data collected mainly by Lehmann and his co-workers [23] is limited to a comparatively small area in West Africa.

There are obviously two facts which may account for such inequalities. It may be pure chance, i.e. the occurrence of one or the other mutation, or the introduction

of the mutated allele from another population in which it is already frequent, which determines the selection for one instead of the other gene. Or else, as seems perhaps more probable, one gene is fitter than the other in a given genetical and environmental background.

Another interesting aspect which the biologist should not forget is the evolution of the infectious agent itself. We know from immunological and epidemiological work how virulence of parasites may change in a short time, and there is no doubt that new infectious diseases have developed in the course of human history and will probably develop in the future. Evolution is a continuous process, and we should expect that there is a mutual, continuous adjustment, in the long run, between the two organisms concerned, host and parasite.

From a social point of view it is interesting to note that the heterozygotes of genes which may be severely detrimental in homozygous condition are selected for. This is well known in animals, as I said, but obviously it opens quite particular problems in man. The case of thalassaemia is the most striking: the defence against malaria implies a lot of deaths by homozygosity of the *Th* gene. Clearly enough total eradication of malaria would in the long run greatly reduce the frequency of the gene. This is indeed a long process: we may be able to see the beginning of a decline in the next generation. The generation examined in Sardinia is the first after the disease has been defeated, and we cannot expect a sudden diminution of the gene.

As to the eugenic practice to be suggested to populations with a high frequency of *Th* gene, I may confine myself to the following purely biological considerations. If the marriage of two heterozygotes is discouraged because it gives rise to 25 per cent of the progeny being affected by Cooley's disease, the frequency of the gene tends to rise if the selecting agent is still operating and favourir g heterozygotes; if, on the contrary, the selective factor is no longer in action, i.e. malaria is exterminated, the gene fre ¡uency would be kept at the original level, supposing that no marriages between heterozygotes occur.

From a purely biological viewpoint a quick way to get rid

of the gene in the population would be to increase the number of marriages between heterozygotes, so as to produce the highest possible number of infants dying by Cooley's disease—thus eliminating *Th* genes. A less radical and even more effective measure would be to obtain that *Thth* × *Thth* crosses be sterile.

Thus far goes the biologist; further comments, and possible action, are to be expected from the sociologist.

In conclusion I would like to stress the urgent need for further investigations, into the problems of selection in mankind, investigations which are so much more urgent nowadays, because we have entered an era in which rapid changes are occurring not only in our environment, but presumably also in our gene pools, subjected as they are to the increase of environmental radioactivity. However, in order to start such a programme of research with a reasonable hope of getting valuable and useful results, it is obviously necessary to get a much wider knowledge of basic human formal genetics and phenogenetics (relations between genotype and phenotype). It would then be possible to achieve rapid progress along the lines indicated by Darwin a century ago.

REFERENCES

1. ADINOLFI, M., BERNINI, L., CARCASSI, U., LATTE, B., MOTULSKY, A. G. and SINISCALCO, M. 1960. Indagini genetiche sulla predisposizione al favismo. I. Il problema e i metodi. *Rend. Acc. Naz. Lincei*, ser. 8*a*, **28**, 716.
2. ALLISON, A. C. 1954*a*. The Distribution of Sickle Cell Trait in East Africa and Elsewhere and its Apparent Relationship to Incidence of Subtertian Malaria. *Trans. roy. Soc. trop. Med. Hyg.* **48**, 312.
3. ALLISON, A. C. 1954*b*. Protection afforded by Sickle-Cell Trait against Subtertian Malarial Infection. *Brit. med. J.* i, 290.
4. ALLISON, A. C. 1956. The Sickle and Haemoglobin C Genes in some African Populations. *Ann. hum. Genet.* **21**, 67.
5. ALLISON, A. C., ASKONAS, B. A., BARNICOT, N. A., BLUMBERG, B. S. and KRIMBAS, C. 1963. Deficiency of Erythrocyte Glucose-6-phosphate Dehydrogenase in Greek Populations. *Ann. hum. Genet.* **26**, 237.
6. BARNICOT, N. A., ALLISON, A. C., BLUMBERG, B. S., DELIYANNIS, G., KRIMBAS, C. and BALLAS, A. 1963. Haemoglobin Types in Greek Populations. *Ann. hum. Genet.* **26**, 229.

7. BERNINI, L., CARCASSI, U., LATTE, B., MOTULSKY, A. G., ROMEI, L. and SINISCALCO, M. 1960. Indagini genetiche sulla predisposizione al favismo. III. Distribuzione delle frequenze geniche per il locus Gd in Sardegna. Interazione con la malaria e la talassemia al livello popolazionistico. *Rend. Acc. Naz. Lincei*, ser. 8*a*, **29**, 115.
8. BIANCO, I., MONTALENTI, G., SILVESTRONI, E. and SINISCALCO, M. 1952. Further Data on the Genetics of Microcythaemia or Thalassaemia minor and Cooley's Disease or Thalassaemia major. *Ann. Eugen. Lond.* **16**, 299.
9. BUTLER, E. 1959. The Haemolytic Effect of Primaquine and Related Compounds: a review. *Blood* **14**, 103.
10. CARCASSI, U., CEPPELLINI, R. and PITZUS, F. 1957. Frequenza della talassemia in quattro popolazioni sarde e suoi rapporti con la distribuzione dei gruppi sanguigni e della malaria. *Boll. Ist. Sieroterap. Milanese* **36**, 207.
11. CEPPELLINI, R. 1955. Discussion of A. C. Allison's paper: Aspects of Polymorphism in Man. *Cold. Spr. Harb. Symp. quant. Biol.* **20**, 252.
12. CHILDS, B. and ZINKHAM, W. H. 1959. The Genetics of Primaquine Sensitivity of the Erythrocytes. Chapter of: *Ciba Foundation and IUBS Symposium on Biochemistry of Human Genetics* London.
13. FERMI, C. 1938. *La malaria in Sardegna.* Sassari.
14. FERMI, C. and MARTINETTI, P. 1905. Studio del favismo. *Ann. Igiene (sper.)* **15**, 75.
15. FISHER, R. A. 1930. *The Genetical Theory of Natural Selection.* London. Oxford University Press.
16. FORD, E. B. 1945. Polymorphism. *Biol. Rev.* **20**, 73.
17. HALDANE, J. B. S. 1931. Natural Selection in Man. *Progress in Medical Genetics* **1**, 27–37.
18. HALDANE, J. B. S. 1949. The Rate of Mutation of Human Genes. Proc. VIII Int. Congr. of Genetics. *Hereditas, Lund* Suppl. **35**, 267.
19. HARDY, G. H. 1908. Mendelian Proportion in a Mixed Population. *Science* **28**, 49.
20. HIERNAUX, J. 1952. La génétique de la sicklémie et l'intérêt anthropologique de sa fréquence en Afrique noire. *Ann. Mus. Congo belge Anthropologie* **2**, 42.
21. HUXLEY, J. S. 1955. Morphism and Evolution. *Heredity* **9**, 1.
22. INGRAM, V. M. 1957. Gene Mutation in Human Haemoglobin: The Chemical Difference between Normal and Sickle Cell Haemoglobin. *Nature, Lond.* **180**, 326.
23. LEHMANN, H. 1959. Variations in Human Haemoglobin Synthesis and Factors Governing their Inheritance. *Brit. med. Bull.* **15**, 40.
24. LIVINGSTONE, F. B. 1958. Anthropological Implications of Sickle Cell Gene Distribution in West Africa. *Amer. Anthropologist* **60**, 533.
25. MONTALENTI, G. 1954. The Genetics of Microcythemia. *Atti IX Congr. Int. Genet.* in *Caryologia*, Suppl. vol. VI, 554.
26. MONTALENTI, G. 1959. Polymorphisme et gènes létaux et sublétaux chez l'homme. *Arch. d. Julius Klaus-Stiftung* **34**, 279.

27. MONTALENTI, G., SILVESTRONI, E. and BIANCO, I. 1953. Nuove indagini sul problema della microcitemia. *Rend. Acc. Naz. Lincei*, ser. 8*a*, **14**, 183.
28. MOTULSKY, A. G. 1960. Metabolic Polymorphism and the Role of Infectious Diseases in Human Evolution. *Hum. Biol.* **32**, 28.
29. NEEL, J. 1949. The Inheritance of Sickle Cell Anemia. *Science* **110**, 64.
30. NEEL, J. 1956. The Genetics of Human Haemoglobin Differences: Problems and Perspectives. *Ann. hum. Genet.* **21**, 1.
31. SANSONE, G. A., PIGA, M. and SEGNI, G. 1958. *Il favismo*. Torino.
32. SILVESTRONI, E. and BIANCO, I. 1963. " Le emoglobine umane: Biochimica, Genetica, Popolazionistica, Patologia e Clinica." Chapter in: L. Gedda, *De Genetica Medica*, IV, pp. 205.
33. SILVESTRONI, E., BIANCO, I. and ALFIERI, N. 1952. Sulle origini della microcitemia in Italia e nelle altre regioni della terra. *Medicina* (Parma) **2**, 187.
34. SINISCALCO, M., BERNINI, L., LATTE, B. and MOTULSKY, A. G. 1961. Favism and Thalassaemia in Sardinia and their Relationship to Malaria. *Nature, Lond.* **190**, 1179.
35. SINISCALCO, M., MONTALENTI, G., SILVESTRONI, E. and BIANCO, I. 1959. Effect of a single gene difference on the pattern of some physical measurements. A chapter in: *Ciba Found. Symp. on Medical Biology and Etruscan Origin*, pp. 205–19. London.
36. SINISCALCO, M., MOTULSKY, A. G., LATTE, B. and BERNINI, L. 1960. Indagini genetiche sulla predisposizione al favismo. II. Dati familiari. Associazione genica con il daltonismo. *Rend. Acc. Naz. Lincei*, ser. 8*a*, **28**, 903.
37. VOGEL, F. 1961. *Lehrbuch der Allgemeinen Humangenetik*. Springer Verlag, Berlin-Göttingen-Heidelberg.
38. VOGEL, F., PETTENKOFER, H. J. and HELMBOLD, W. 1960. Über die Populations genetik der ABO-Blut gruppen. 2. Mitt.: Genhaüfigkeit und epidemische Erkrankungen. *Acta Genet.* **10**, 267. Basel.
39. WALTERS, J. H. and LEHMANN, H. 1956. Distribution of the S and C Haemoglobin Variants in two Nigerian Communities. *Trans. R. Soc. trop. Med. Hyg.* **50**, 204.
40. WEINBERG, W. 1908. Über den Nachweis der Vererbung beim Menschen. *Jahresh. Verein. vaterl. Naturk. in Württemberg*, **64**, 368.
41. WENT, L. N. 1961. Segregation-ratio Advantage of Abnormal Haemoglobins. *Nature, Lond.* **192**, 382.

CHROMOSOMAL ABERRATIONS IN DEVELOPMENTAL DISEASE AND THEIR FAMILIAL TRANSMISSION

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GROSS chromosomal aberrations must now be considered an important cause of human developmental disease. Thus, some 20 per cent of spontaneous abortions,[4] 25 per cent of women with primary amenorrhoea,[19] 11 per cent of high-grade male subfertility [13] and about 6 per cent of the institutionalized mentally defective have chromosomal abnormalities. These figures refer, of course, to chromosome aberrations readily detected under the oil immersion lens of an ordinary laboratory microscope. The limitations of cytology are such that quite severe degrees of chromosomal duplication, deficiency and re-arrangement must pass unrecognized, and there is a strong possibility that these lesions, just beyond the resolution of the microscope, may be responsible for an even greater amount of congenital disease. This seems to be confirmed by some of the more recent studies in which progressively smaller abnormalities are being confidently diagnosed.

Chromosome abnormalities are of two distinct types. First are the simple *numerical* abnormalities, where the affected person has an abnormal number of chromosomes, a defect which is usually the result of an accident of cell division, termed non-disjunction, in which the dividing chromosomes have failed to segregate equally between two daughter cells. The regular variety of mongolism is the best known example of this type; those affected have forty-seven chromosomes, the extra one being a normal chromosome 21, so that there are three chromosomes 21 instead of two, a situation termed trisomy. For a person to have an abnormal number of chromosomes in every somatic cell, the abnormal cell division must occur during gametogenesis, or during the early division of the fertilized egg.

In the second type of chromosome aberration there are abnormalities of *structure*, affecting the length and centromere position of individual chromosomes. These abnormalities result from multiple chromosome breakage with loss of chromosome segments, or from breakage with exchange of chromosome fragments between non-homologous chromosomes. The translocation variety of mongolism is the most familiar example of this type.

From the standpoint of clinical medicine it is often useful to subdivide human chromosome aberrations in another way, into those involving the sex chromosomes, and those involving the non-sex chromosomes, or autosomes. For autosomal anomalies are associated with severe mental and physical abnormalities affecting many of the systems of the body, whereas patients with sex chromosome aberrations have much less disability, often solely confined to the reproductive system; many of these patients are useful members of society.

In our present state of knowledge, the clinical defects associated with chromosome aberrations are irreversible, and so prevention remains the primary aim. As most chromosome aberrations occur sporadically, a knowledge of predisposing factors is essential but, unfortunately, this knowledge is scant, and at present only two possible factors are known to increase the frequency of chromosome aberrations. These factors are increasing maternal age and exposure to radiation. This experience warns us to have our children when we are young, and to avoid harmful irradiation, but this sort of advice can hardly be expected to influence the frequency of chromosome aberrations in the general population. Clearly much more information about aetiological factors is required. At this point it might be mentioned that in a small proportion of patients with sex chromosome aberrations it is now possible, by a careful study of the segregation of X-linked markers, such as colour-blindness and the Xg blood group, to determine in which parent the chromosome aberration originated. In this way aetiological factors operating on the mother can be separated from those operating on the father. For example, in cases of XXY Klinefelter's syndrome, an increased maternal age effect is apparent in the cases in which the non-disjunctional

event occurs in the mother, but cannot be demonstrated in those cases where the non-disjunction is paternal.[14]

Although most chromosome aberrations appear to arise spontaneously, a small percentage of cases are familial. Our present state of knowledge and our ability to distinguish these cases should at least ensure that a mother who has had a malformed child due to a chromosome aberration is advised correctly about the risk involved in undertaking a further pregnancy. It is the purpose of this paper to consider the question of transmission of developmental disease in persons with chromosome aberrations.

FERTILITY IN PERSONS WITH AUTOSOMAL ABERRATIONS

It is well known that the majority of autosomal aberrations reported to date are associated with such mental and physical defects that reproduction is impossible, even if the patient survives to reproductive age. However, these patients all seem to have normal gonads and genital ducts, and are thus potentially fertile. No patients have been known to survive to reproductive age in either the group 13–15 (D) trisomy or trisomy 18 syndromes, and trisomy 16 has only been found in spontaneous abortions (Table I). About 40 per cent of cases of mongolism with trisomy 21, on the other hand, survive to reproductive age but seldom reproduce. Reproduction is known in mongoloid women, and of the twelve pregnancies recorded, five have resulted in mongoloid offspring.[21] This gives a ratio near enough to the 1:1 ratio expected from the random segregation of the extra chromosome 21 to half of the mother's ova. As some spontaneous abortions have been found with trisomy 21[4] a slight deficiency of trisomic offspring to mongoloid mothers is probably to be expected.

Another very important type of familial mongolism is that which occurs in phenotypically normal women who are chromosomal mosaics for normal and trisomic 21 cell lines.[3] Penrose [27] has estimated that some 10 per cent of mongoloid individuals arise in this way. As it is impossible to exclude mosaicism completely by cytological techniques, this raises a very serious

difficulty in advising mothers who have given birth to a mongoloid child whether or not to have another pregnancy.

The third type of situation in which an autosomal aberration is transmitted, is that in which the normal parent carries a structural abnormality in the form of a balanced translocation, and gives rise to unbalanced translocations in abnormal offspring. Translocation mongolism is numerically the most important example of this type of transmission. About 5 per cent of all cases of mongolism [27] are associated with a chromosomal translocation, although more than half of these appear

TABLE I

CHROMOSOME ABERRATIONS—THE POSSIBILITY OF TRANSMISSION

	VIABLE	SURVIVAL TO REPRODUCTIVE AGE	OFFSPRING
Trisomy 16	—	—	—
Trisomy 13–15	+	—	—
Trisomy 18	+	—	—
Trisomy 21	+	40 per cent	1/2 Affected
Trisomy 21/Normal Mosaicism	+	+	Variable number affected
Translocation carrier 13/21	+	+	1/3 Affected, 1/3 Carrier 1/3 Normal
Translocation carrier 21/21	+	+	All affected
XXY	+	+	—
XO	+	+	One normal described
XXX, XYY	+	+	All unaffected

to arise sporadically because both parents can be shown to have normal chromosomes.[8] In these cases of mongolism, the translocation takes place between a chromosome 21 and another chromosome, often one in the 13–15 group. The balanced translocation heterozygote thus has a chromosome abnormality consisting of a reciprocal exchange of fragments between, for example, the short arm of a number 13 chromosome and the long arm of a number 21 chromosome. Two abnormal chromosomes are formed; the large one is similar in appearance to a chromosome in the 7–12 group and is composed of the long arms of both chromosomes 13 and 21; the small one is composed of the short arms of these chromosomes, and is apparently of little genetic importance because

it is readily eliminated without producing any obvious effect on the phenotype. The typical carrier of the 13/21 translocation has (after losing the small chromosome) forty-five chromosomes including only one number 13 and only one number 21, and appears to have an extra chromosome in the 7–12 group. A female carrier of this sort produces three types of viable ova; one with twenty-two chromosomes including the 13/21 chromosome but without a normal number 21; one with twenty-three chromosomes including the 13/21 and a normal 21; and one with twenty-three chromosomes including a normal 13 and a normal 21. When fertilized with normal sperm these ova will produce carrier, mongol and normal offspring in equal proportions. The risk of a translocation carrier producing a mongoloid child is therefore one in three, a risk which would not normally be advised.

In view of these findings it is clearly of advantage to parents who have had a child with an autosomal anomaly, and who contemplate a further pregnancy, to have chromosome analysis to determine whether or not they have a transmissible chromosome defect. As most medical schools in this country now have laboratory facilities capable of handling this service, there need be no excuse for omitting this investigation.

INFERTILITY IN PATIENTS WITH SEX CHROMOSOME ABERRATIONS

In contrast to the patients with autosomal aberrations, those with abnormal sex chromosome constitutions do not usually show severe physical and mental disability. In spite of this, there is no recorded instance of affected individuals transmitting the disorder to their offspring. Transmission of the defect appears to be prevented solely by a fortunate mechanism which prevents the successful maturation of germ cells with abnormal sex chromosome complements.[11] The nature of this mechanism will be considered below.

From the clinical standpoint there are four types of sex chromosome aberration, the Klinefelter group, the Turner group, and what might be called the extra-X group, and the extra-Y group.

Those in the Klinefelter group are male patients whose sex chromosome complement includes at least one normal Y chromosome and more than one X chromosome. The clinical syndrome is characterized in the adult by small testes, increased urinary excretion of gonadotrophins and skeletal disproportion; variable features include gynaecomastia, mental defect and androgen deficiency. At all ages from birth there is a marked deficiency of germinal cells, in many cases amounting to complete absence.[9] Disability as judged by the severity of mental defect, the frequency of cryptorchidism and the frequency of proximal radio-ulnar synostosis, appears to increase with the number of extra X chromosomes.[10] Only one 'fertile' case is known which is adequately supported by paternity studies,[28] but the pathological findings in the testes in this case seem to contradict this claim. It seems that mosaic Klinefelter patients with an XY cell line, may have normal testicular function [2] and so may reasonably be expected to father normal children.

Patients with Turner's syndrome and its variants show variable deficiency of the sex chromosome complement intermediate between XX and XO, and XY and XO respectively.[12] The commonest aberration is monosomy for the X chromosome (XO), but X and Y chromosome deletions, and XO/XX, XO/XY and other types of mosaicism are also described. The typical adult patient with Turner's syndrome has primary amenorrhoea, infantilism, high urinary gonadotrophins, is of short stature, and usually has several of the associated congenital abnormalities, such as webbing of the neck, peripheral lymphoedema at birth and co-arctation of the aorta. There are no true ovaries, and all that can be found at the ovarian sites are thin streaks of ovarian stroma without germ cells. However, spontaneous menstruation is described in a few cases, and one XO individual is known to have had a normal child,[1] so that occasionally some germ cells are present.

Female patients with XXX, XXXX and XXXXX sex complements (and with no Y chromosome) form a third group of sex chromosome aberrations. There seems to be no distinct clinical syndrome associated with these chromosomal findings, and those affected are at least potentially fertile,[20] although

mental deficiency is more common that in the general population.[6]

It is of great interest that no XXX or XXY offspring have been born to XXX mothers, although at least eight normal children are known. By analogy with the situation observed in mongoloid mothers,[6] one would expect half the offspring to have a chromosomal aberration. In the XXX cases there thus seems to be evidence of selection against abnormal ova. This seems to be supported by the observation of reduced numbers of ova in the gonads of some XXX patients [20] and also by the findings that some patients have secondary amenorrhoea.[18]

At least eleven individuals are now known to have an XYY sex chromosome constitution.[17] Five of these patients are mentally retarded and seven show some genital abnormality, five having cryptorchidism. Only two are of reproductive age, one being childless and the other fathering ten pregnancies. Among the ten were two spontaneous abortions and one mongoloid child, but apparently no XYY children.

THE CAUSE OF THE FAILURE TO TRANSMIT THE DEFECT IN THE SEX CHROMOSOME ABERRATIONS

A possible explanation for the finding that patients with sex chromosome aberrations do not transmit their anomaly to their offspring is suggested by observations relating to the Lyon hypothesis of X-inactivation.[23]

The hypothesis postulates that one of the two X chromosomes in each female somatic cell becomes genetically inert during embryogenesis, the process of inactivation occurring in either of the X chromosomes randomly in different cells. The inactivated X chromosome becomes condensed in intermitotic nuclei and is visible under the microscope as the sex chromatin body. One of the consequences of X-inactivation is that the somatic cells of females heterozygous for X-linked genes, show a mosaic pattern of activity with respect to those genes. This has been very clearly demonstrated for the X-linked locus for glucose-6-phosphate dehydrogenase.[7] The hypothesis also accounts for the phenomenon of ' dosage compensation ', which refers to the observation that the product

of an X-linked gene is quantitatively the same when it is present in single dose in the XY male as it is when present in double dose in the XX female. In the Klinefelter and extra-X cases, the extra-X chromosomes are inactivated, and this explains the comparatively small effect on the phenotype as compared with that of autosomal aberrations.

There is convincing evidence that X-inactivation does not occur in oogonia,[26] and this may be the important factor in the differentiation and development of these cells. In other words, the complete genetic activity of two X chromosomes may be essential for the normal maturation and proliferation of oogonia. It might follow that embryonic germ cells are more susceptible to the genetic imbalance produced by abnormal sex chromosome complements than are somatic cells, because the imbalance is not masked by genetic inactivation. This genetic imbalance could lead to embryonic germ cell degeneration, which seems a satisfactory explanation for the usual absence of germ cells in patients with the Turner and Klinefelter syndromes. The occasional survival of a limited number of germ cells in these patients, and the observations of fertility in other types of sex chromosome aberrations without mosaicism for normal cell lines, requires further explanation. It has already been noted that all known offspring of these cases have had normal sex chromosome complements, and it is possible that non-disjunctional events involving the sex chromosomes during proliferation of the germ cells have led to the selection and survival of a small proportion of germ cells with normal sex chromosome complements. Support for this view is provided by a recent study of oogonia in the creeping vole, *Microtus oregani*.[25] The sex chromosome constitution of the normal female in this species is XO, but XX oogonia are produced by a process of selective non-disjunction.

This concept of embryonic germ cell degeneration as a cause of the gonadal defects in the Klinefelter and Turner syndromes is also supported by animal experiments. Hemsworth and Jackson [15,16] have demonstrated that if Busulphan is administered to pregnant rats on or about the thirteenth day after insemination, i.e. at the time of migration and proliferation of the embryo's germ cells, these are destroyed, so that

female offspring are born with sterile atrophic ovaries and males with sterile testes. The similarity of such ovaries to the gonads of Turner's syndrome, and of the testes to the prepubertal testes with germinal aplasia in Klinefelter's syndrome is striking, and suggests that some abnormal sex chromosome complements lead to germ cell degeneration. Evidence from other species is scant, and to some extent conflicting. Thus the XXY mouse [24] and the XXY cat [5] are like the human XXY and are sterile, having testicular germinal aplasia. However, the XO mouse is fertile and produces both XO and normal offspring. It seems that studies of hybrids in mammals may also provide confirmatory evidence, for sterility is usual in this particular type of chromosome aberration. It is of interest that recent histological studies of mule ovaries show changes similar to the streak gonads of Turner's syndrome.[22]

SUMMARY

The various types of human chromosome aberrations are discussed in relation to their familial transmission. Direct transmission from parent to half the offspring is theoretically possible in the autosomal trisomy syndromes, because all cases are potentially fertile. However, the severity of the physical and mental defect in these conditions usually prevents reproduction, even if it has not led to death before the reproductive age. The occurrence of mosaicism for normal and trisomic cell lines in apparently normal parents, is a more important cause of familial transmission, and presents difficulties because one cannot assess the risk of producing completely trisomic children. On the other hand, a parent with a balanced autosomal translocation can be advised about the chances of producing abnormal offspring; in the case of the translocation mongol, the theoretical risk is one in three.

Individuals with sex chromosome aberrations do not show the severe disability associated with autosomal aberrations. Despite this, transmission of the abnormality from parent to offpsring is unknown. This seems to be due to a failure to produce germ cells with abnormal sex chromosome complements. Evidence is presented in favour of a hypothesis that

explains this on the basis that genetic inactivation of the X chromosome does not occur in germ cells.

DISCUSSION

DR FERGUSON-SMITH, when asked if it was known by what mechanism embryos with gross chromosome aberrations, such as trisomy 16, were aborted spontaneously, replied that trisomy 16 had been demonstrated on at least three occasions from products of spontaneous abortions, but was unknown in live-born children. Presumably this aberration was compatible with only a short period of embryonic development. Death of the foetus initiated in some unknown way the expulsion of the whole conceptus, even although part of the membranes were still viable and could be grown in culture. In answer to a further question, he knew of no evidence that thalidomide acted by preventing the expulsion of abnormal embryos; on the contrary, the evidence showed that the drug was actively teratogenic. (Incidentally, chromosome aberrations are not induced *in vitro* by treating growing human cells with thalidomide.)

Asked if chromosome abnormalities could be detected *in vitro* by culture of embryonic cells in view of the possibility that birth of an abnormal child might be prevented by therapeutic abortion, the speaker thought that in pregnancies where there was a high risk of a chromosomally abnormal child, it was doubtful whether uterine puncture and withdrawal of viable embryonic cells could be accomplished without risk to mother and foetus early enough in pregnancy to make therapeutic abortion feasible. (In such a situation cell culture might have to be continued for several weeks before chromosome analysis was possible, and even then there was no assurance that maternal tissue was not being grown.) It was of interest that a Danish group had had a similar idea, applying the much simpler technique of nuclear sexing to amniotic cells obtained by uterine puncture at the fourth month of pregnancy in two mothers heterozygous for the X-linked haemophilia gene (*Lancet*, 1960, ii: 180). The object was to

terminate the pregnancy if the foetus was found to be male, because of the expectation that half the male offspring would have haemophilia.

REFERENCES

1. BAHNER, F., SCHWARZ, G. and HEINZ, H. A. 1960. Turner-Syndrom mit voll ausgebildeten sekundären Geschlechtsmerkmalen und Fertilität. *Acta endocrinol.* **35**, 397.
2. BARR, M. L., CARR, D. H., MORISHIMA, A. and GRUMBACH, M. M. 1962. An XY/XXXY Sex Chromosome Mosaicism in a Mentally Defective Male Patient. *J. ment. Defic. Res.* **6**, 65.
3. BLANK, C. E., GEMMELL, E., CASEY, M. D. and LORD, M. 1962. Mosaicism in a Mother with a Mongol Child. *Brit. med. J.* ii, 378.
4. CARR, D. H. 1963. Chromosome Studies in Abortuses and Stillborn Infants. *Lancet* ii, 603.
5. CHU, E. H. Y., THULINE, H. C. and NORBY, D. E. 1964. Triploid-diploid Chimerism in a Male Tortoiseshell Cat. *Cytogenetics* **3**, 1.
6. COURT BROWN, W. M., HARNDEN, D. G., JACOBS, P. A., MACLEAN, N. and MANTLE, D. J. 1964. Abnormalities of the Sex Chromosome Complement in Man. M.R.C. Special Report Series No. 305. London. H.M.S.O.
7. DAVIDSON, R. G., NITOWSKY, H. M. and CHILDS, B. 1963. Demonstration of Two Populations in the Human Female Heterozygous for Glucose-6-Phosphate Dehydrogenase Variants. *Proc. nat. Acad. Sci., Wash.* **50**, 481.
8. EDWARDS, J. H., DENT, T. and GULI, E. 1963. Sporadic Mongols with Translocations. *Lancet* ii, 902.
9. FERGUSON-SMITH, M. A. 1959. The Prepubertal Testicular Lesion in Chromatin-Positive Klinefelter's Syndrome (Primary Micro-Orchidism) as seen in Mentally Handicapped Children. *Lancet* i, 219.
10. FERGUSON-SMITH, M. A. 1963. Chromosome Studies in Klinefelter's Syndrome. *Proc. roy. Soc. Med.* **56**, 577.
11. FERGUSON-SMITH, M. A. 1964. Karyotype-phenotype correlations in gonadal dysgenesis and their bearing on the pathogenesis of Malformations. *J. med. Genet.* (In Press.)
12. FERGUSON-SMITH, M. A., ALEXANDER, D. S., BOWEN, P., GOODMAN, R. M., KAUFMANN, B. N., JONES, H. M. and HELLER, R. H. 1964. Clinical and Cytogenetical Studies in Female Gonadal Dysgenesis and their bearing on the Cause of Turner's Syndrome. *Cytogenetics* **3**. No. 6.
13. FERGUSON-SMITH, M. A., LENNOX, B., MACK, W. S. and STEWART, J. S. S. 1957. Klinefelter's Syndrome: Frequency and Testicular Morphology in relation to Nuclear Sex. *Lancet* ii, 167.

14. FERGUSON-SMITH, M. A., MACK, W. S., ELLIS, P. M., DICKSON, M., SANGER, R. and RACE, R. R. 1964. Parental Age and the source of the X Chromosomes in XXY Klinefelter's Syndrome. *Lancet* i, 46.
15. HEMSWORTH, B. N. and JACKSON, H. 1963. Effect of Busulphan on the Male Rat. *J. Reprod. Fertil.* **5**, 187.
16. HEMSWORTH, B. N. and JACKSON, H. 1963. Effect of Busulphan on the Developing Ovary in the Rat. *J. Reprod. Fertil.* **6**, 229.
17. HUSTINX, T. W. J. and VAN OLPHEN, A. H. F. 1963. An XYY Chromosome Pattern in a Boy with Marfan's Syndrome. *Genetica* **34**, 262.
18. JACOBS, P. A., BAIKIE, A. G., COURT BROWN, W. M., MACGREGOR, T. N., MACLEAN, N. and HARNDEN, D. G. 1959. Evidence for the Existence of the Human ' Super Female '. *Lancet* ii, 423.
19. JACOBS, P. A., HARNDEN, D. G., BUCKTON, K. E., COURT BROWN, W. M., KING, M. J., MCBRIDE, J. A., MACGREGOR, T. N. and MACLEAN, N. 1961. Cytogenetic Studies in Primary Amenorrhoea. *Lancet* i, 1183.
20. JOHNSTON, A. W., FERGUSON-SMITH, M. A., HANDMAKER, S. D., JONES, H. W. and JONES, G. S. 1961. The Triple-X Syndrome: Clinical Pathological and Chromosomal Studies in Three Mentally Retarded Cases. *Brit. med. J.* ii, 1046.
21. JOHNSTON, A. W. and JASLOW, R. I. 1963. Children of Mothers with Down's Syndrome. *New Engl. J. Med.*, **269**, 439.
22. JONES, H. W. and HELLER, R. H. Personal Communication.
23. LYON, M. F. 1963. Attempts to test the Inactive-X Theory of Dosage Compensation in Mammals. *Genet. Res., Camb.* **4**, 93.
24. LYON, M. F. Personal Communication.
25. OHNO, S., JAINCHILL, J. and STENIUS, C. 1963. The Creeping Vole (*Microtus oregoni*) as a Gonosomic Mosaic. I. The OY/XY constitution of the male. *Cytogenetics* **2**, 232.
26. OHNO, S., KAPLAN, W. D. and KINOSITA, R. 1960. On the Isopycnotic Behavior of the XX-Bivalent in Oocytes of *Rattus Norvegicus*. *Exp. Cell. Res.* **19**, 637.
27. PENROSE, L. S. 1964. Mongolism as a Problem in Human Biology. Proceedings of the Symposium on The Early Conceptus, Normal and Abnormal, held at Queen's College, Dundee, Sept. 1964.
28. WARBURG, E. 1963. A Fertile Patient with Klinefelter's Syndrome. *Acta endocrinol.* **43**, 12.

SOME PRACTICAL APPLICATIONS

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IT was recommended that this session might include one rather bread-and-butter paper, namely, a brief review of some of the contributions that genetics can make to the practice of medicine and surgery here and now, or at least in the reasonably foreseeable future. The first contribution I should like to mention is the help a knowledge of genetics can give in facilitating early diagnosis when this is important if effective treatment is to be instituted. Early diagnosis is important in a number of genetically determined conditions. A well-known example is multiple polyposis of the colon and rectum, a condition determined by a dominant gene.[4] This is a pre-cancerous condition and early operation has saved many lives. Dr Dukes and his colleagues at St Mark's Hospital have kept in touch with very many family groups, and as individuals of the younger generations grow up they are examined and, if polyposis is found, are operated on.

Galactosaemia is due to a deficiency or abnormality of the enzyme galactose 1-phosphate uridyl transferase. The body is unable to utilize lactose and toxic products cause serious damage, including liver damage, cataracts and severe mental deficiency. Early death is common. The condition is not easily diagnosed, but, if this is done sufficiently early, withholding lactose from the diet can ensure more or less normal development. Galactosaemia is due to a recessive gene, so when an affected child has been born and has perhaps died, it is at least known that the risk of recurrence in any subsequent child is one in four, so that all concerned are ready to detect abnormality at the earliest possible moment. The same considerations apply to phenylketonuria, where it is most important to put the child on a diet very low in phenylalanine as early as possible. With this condition the test is so simple, however, that screening the whole population of new births is practicable and is being practised in many areas.

Diabetes insipidus is due to a sex-linked gene. Once again early diagnosis may be difficult and in the absence of treatment the results are often disastrous, with a high proportion of early deaths. The maintenance of a proper fluid and electrolyte balance makes it possible to secure fairly normal development. So when a woman has had an affected boy, it is known that the risk to any subsequent boy, barring a mutation, is 1 in 2.

A number of commoner conditions could be mentioned which are not wholly genetic, but are at least partly genetic, and where there is an appreciable or fairly substantial incidence in close relatives, these incidences being estimated by making appropriate surveys. With congenital pyloric stenosis, the commonest reason for surgery in very early life, about 8 per cent of the brothers or sons of affected men are affected and no less than about 20 per cent of the brothers and sons of affected women. The results of treatment are excellent and the cure lifelong. In fact there is evidence that those affected are above the average in muscular efficiency. Other conditions include congenital dislocation of the hip and Hirschsprung's disease. With Hirschsprung's disease very early operation offers the best hope of survival in many desperate cases. Meconium ileus is one of the forms that fibrocystic disease of the pancreas may take, and very early operation is required if life is to be saved. Fibrocystic disease in an older sib is a warning that this complication must be watched for in any subsequent child.

Help in early diagnosis is by no means confined to the diseases of infancy and childhood, however. I will mention two striking examples concerning adult diseases. Miller and Paterson [5] carried out a family study of glaucoma simplex. The incidence of frank disease in relatives was quite low, namely, four out of 50 sibs and two out of 75 children. But a measure of the coefficient of outflow from the anterior chamber of the eye, the Friedenwald index, gave very striking results. The values were abnormally high in just about half the sibs, and in just about half the children, whereas in the controls there was but a single instance of a value above the accepted upper limit of normality. This strongly suggests a dominant

gene, though of course it is only a fairly small proportion of the bearers of the gene who will develop frank glaucoma. A remarkable finding was that the index showed little if any change with age, the proportion of abnormal values being about the same in relatives of all ages down to fifteen years, the youngest studied. If all this works out, and it looks very convincing, it would be well worth while examining the sibs and children of people with glaucoma simplex, and perhaps their nephews and nieces and first cousins too, and keeping under periodical observation those who showed a raised index.

The second example is idiopathic haemochromatosis, a defect of iron metabolism. In the past the evidence for genetic determination was not very strong and there have been divided views. The practical difficulties of carrying out family studies are formidable. This is largely because liver biopsies on normal control subjects are hardly admissible and those with actual or suspected liver disease are not a very good substitute as controls. In spite of the difficulties, however, Williams, Sheuer and Sherlock [8] have been able to produce strong evidence that an intermediate gene is responsible. One dose of the gene gives lesser manifestations, two doses the full disease, or something approaching it. Treatment by multiple venesection, instituted as early as possible, is most important, and these authors have already diagnosed the disease at a relatively early stage in some sibs of affected persons and have commenced to treat them.

I must now turn to another, but related subject, namely drug-induced disease where the susceptibility has a genetic basis. A very striking example is probably familiar to you, but I think the story bears repetition. A form of hereditary porphyria, porphyria variegata, is common in the white and coloured populations of South Africa and it is estimated that in that relatively small population there are about 8000 affected individuals. It is due to a dominant gene and, thanks to intensive work by Dean,[3] it now seems certain that all these abnormal genes derive from a single mutation, for the evidence is very strong that all 8000 are descended from a single couple who married in 1688. Under natural conditions the gene does not do very much harm. Some affected people suffer from

rather troublesome skin lesions and some have acute episodes. Some, however, remain free of all symptoms, though the presence of the gene can always, or practically always, be detected, at least in adults, by a very simple chemical test. It was the introduction of the barbiturates, and above all of barbiturate anaesthetics, that produced a transformation. These drugs are very apt to cause severe reactions and there have been many deaths. The danger is now well recognized. At some South African hospitals it is routine to test for porphyrin before giving a general anaesthetic, and those known to carry the gene are given cards warning doctors that the dangerous drugs must not be given.

Another example is provided by an enzyme defect, a deficiency or abnormality of pseudocholinesterase. Under natural conditions there seem to be no harmful effects at all, at least none have been discovered. But if suxamethonium is given as a muscle relaxant during anaesthesia there may be a prolonged and dangerous apnoea. The genetics are not simple. Several genes are involved; they may well be alleles. In general, inheritance is recessive. The frequency of susceptible persons in our population is about one in 2000. Already during the course of family studies susceptible persons have been identified and given cards warning doctors that suxamethonium must not be used. Other examples could be given and it seems certain that the list of drug sensitivities known to have a genetic basis will continue to grow.

The next topic I should like to mention is the detection of carriers. From the practical point of view this is most important with sex-linked genes. Apart from a recent mutation the chance that the sister of an affected boy will be a carrier is 1 in 2. It is of the greatest importance to distinguish between the two kinds of sisters if possible. If it is possible, then half the sisters of such boys must be warned that the chance that any son will be affected or any daughter a carrier is one-half. But the other half of the sisters can be assured that they are not carriers and have nothing to fear. Fortunately, it is precisely with sex-linked genes that the carrier state is most easily detected. This might be expected on the Lyon hypothesis. If indeed only one X chromosome is active in the cell,

some of these will be the X bearing the abnormal gene, and so it may be expected that lesser manifestations, usually perhaps of no clinical significance, or perhaps detectable only by the use of special tests, will in fact frequently be found. And this seems to be coming true.

Carter and Simpkiss [1] showed that carriers of nephrogenic diabetes insipidus can almost always, or perhaps always, be detected by their failure to produce a normally concentrated urine. Retinitis pigmentosa is usually recessive, but in some families it is due to a sex-linked gene. The carrier woman can be detected by a symptomless anomaly visible on examination with the ophthalmoscope, the tapetal reflex. In choroideraemia, another sex-linked eye condition, the carrier women also show a symptomless lesser manifestation.

The most important breakthrough is quite recent. This is in the Duchenne type of progressive muscular dystrophy, which, as simply inherited conditions go, is relatively common. In the affected boys there is a rise in the level of creatine kinase in the blood. Now it has been discovered that the great majority of carrier women also show a rise of lesser degree, but nevertheless outside the normal range.[9] It seems likely that diagnosis of the carrier state can be made with about 90 per cent efficiency. This is a most important advance and will be a boon to many of the female relatives of affected boys, for they can be assured with high probability that they have been lucky and do not carry the gene.

Unfortunately, with haemophilia, this advance has not yet been made, though it may be hoped that an efficient method for the detection of carriers will be discovered in the not too distant future. I am no haematologist, but if I understand it correctly the present difficulty may be largely technical. Some carrier women do show a significant lowering in the level of anti-haemophilic globulin, but in the opinion of most observers the test is not at present of much practical use. One difficulty is that there is a considerable margin of error in the estimation. Another is that among normal people there are considerable variations in level and these seem to be genetically controlled. Hence a carrier woman might reveal a level which with her genetic background was in fact low, but yet was higher than

that in a normal woman coming from a family with a tendency to have relatively low levels of anti-haemophilic globulin. Still, as I have said, it may be hoped that the difficulties will be overcome and that haemophilia will join the growing array of sex-linked conditions in which the carrier woman can be detected, either unequivocally, or at least with sufficient probability to be of use for giving genetic advice.

The detection of the carriers of recessive genes is of much less practical importance, unless the gene should happen to be relatively common, which it seldom is. The detection of the carriers of recessive genes has indeed become possible with a growing list of conditions, though usually only when investigations can be carried out at the enzyme level; and then it is often found that the efficiency of separation of normals and heterozygotes is only partial. It is of course quite different with those intermediate genes which are common in some parts of the world. A very practical application of genetics has been proceeding for a considerable time in Italy. In parts of Italy the thalassaemia gene, which in double dose gives the fatal Cooley's anaemia, is very common rising to a frequency of one person in five in some townships in the district of Ferrara. Given random mating in those townships, one child in every hundred will die of Cooley's anaemia. A thorough system of testing has been instituted, especially for school children, and those found to carry the gene, that is those who display the lesser anomaly of thalassaemia minor, are warned of the danger of marrying each other.

The sickling gene is even commoner in parts of Africa. In fact in the areas of highest frequency something not far short of 10 per cent of children may be expected to suffer from sickle cell anaemia. When other more important problems of public health have been tackled, a scheme on the lines of the Italian one is likely to make an important contribution.

Most recessive genes are not sufficiently common to make it likely that the screening of the population would be a practical proposition even if efficient methods of detection were available. The commonest recessive defect in our own population is fibrocystic disease of the pancreas, with a frequency of about one in 2000 births. If it is always due to the same

gene—and there is some evidence that it is—this means that something like one person in twenty-five carries the gene. At present there is no method for detecting carriers. If there were, it might perhaps be considered whether general screening was worthwhile.

There might perhaps be some reason for testing the close relatives, particularly the sibs, of those suffering from a recessive defect, if, of course, a test were available, and then with those found to be carriers tests could be offered to prospective marriage partners. But all this is rather speculative and not much is to be expected along these lines for some time to come.

A number of other practical applications could be listed. There is the study of the genetic effects of ionizing radiation. There is the treatment of haemolytic disease of the foetus and newborn, which is largely an essay in applied genetics. Here recent work [2] holds the promise that one day it may be possible to prevent the sensitization of Rh-negative women bearing their first Rh-positive child. Then there are the medico-legal applications of genetics, such as the use of blood-grouping in cases of disputed paternity.

When these and all other applications are considered, however, there is no doubt in my mind that far the most important single application of genetics in medicine is the provision of genetic advice for those who need it. Not many couples really need genetic advice; but those who do, need it badly. Experience shows that in fact useful advice can be given in a very high proportion of instances. I will not try to develop this theme, however, as I have attempted to do in several publications.[6, 7]

DISCUSSION

Replying to a questioner who, referring to recent research in India, suggested that there might be a considerable dietary influence in the incidence of glaucoma simplex, Dr FRASER ROBERTS said that the ratio of 1:1 which was strongly suggested, both in sibs and in children, did not seem to be compatible with an environmental explanation. Needless to say, however, it might well be that environmental influences were of great

importance in determining whether the genetic predisposition was translated into frank disease.

Asked to explain further his reference to 'genetic advice to couples' Dr Fraser Roberts said that he and his colleague, Dr C. O. Carter had been running regular genetic clinics at Great Ormond Street since 1946 and that there were some others now in other parts of the country. It was definitely a matter of offering advice to people who felt they needed it. In their experience 90 per cent of all queries came from couples who had had a child with something the matter with it and were afraid of repetition in a subsequent child. They never advised people whether to have children or not; they worked out the risks to the best of their ability and then explained these risks to the couple. The cases fell very neatly into two groups: the bad risks and the good ones. Most couples really were deterred when they heard that unfortunately the risk was high, but in a considerable proportion of instances the risk was small. They felt that if it was less than one in twenty, people really had not very much to worry about.

REFERENCES

1. Carter, C. O. and Simpkiss, M. 1956. *Lancet* ii, 1069.
2. Clarke, C. A. 1964. *Genetics for the Clinician.* 2nd edn. Oxford. Blackwell.
3. Dean, G. 1963. *The Porphyrias.* London. Pitman.
4. Dukes, C. E. 1952. *Ann. Eugen. Lond.* **15**, 135.
5. Miller, S. J. and Paterson, G. D. 1962. *Brit. J. Ophthal.* **46**, 513.
6. Roberts, J. A. F. 1962. *Brit. med. J.* i, 587.
7. Roberts, J. A. F. 1963. *An Introduction to Medical Genetics.* 3rd edn. London, Oxford University Press.
8. Williams, R., Sheuer, P. J. and Sherlock, S. 1962. *Quart. J. Med.* N.S. **31**, 249.
9. Wilson, K. M., Evans, K. A. and Carter, C. O. *Brit. med. J.* (In the Press.)

ASPECTS OF FERTILITY CONTROL

Chairman: Sir Julian Huxley

INTRODUCTION

SIR JULIAN HUXLEY

ALTHOUGH this discussion has already roamed very widely, there are still certain aspects which I do not think we can hope it will be able to cover in two days. For instance, on the physiological side, nothing, so far as I remember, has been said about the stress effect of overcrowding and frustration, a stress which has been experimentally demonstrated in a number of animals. This is undoubtedly operating on the inhabitants of any city with over a million inhabitants in the world to-day, and has, to my mind, very serious implications.

We have had one paper by Dr Gille—and a very valuable one—about other countries than our own. He has reminded us of the immense differential between the so-called developed and the so-called underdeveloped countries, not only in standards of living but, what is in a way much more serious, in rates of increase. We clearly need a discussion some time—not necessarily at this Symposium—of what can be done in the way of aid to these underdeveloped countries to get their rate of population increase down to a manageable level, so that their breakthrough to industrial prosperity is not interfered with, and so that they have sufficient educational and recreational facilities as well as adequate material standards. This raises a very important point, which I hope may come up in the general discussion, namely the duties of scientifically, technically and socially advanced countries to aid the underdeveloped and less advanced countries in this matter of population: by more research, by making the results of research available on demand, by providing advice and trained personnel for setting up and implementing programmes and projects for population regulation and birth control, and by giving contraceptive devices free as part of their aid programmes.

THE COST OF LARGE FAMILIES AND OF THEIR PREVENTION, WITH SPECIAL REFERENCE TO UNDERDEVELOPED COUNTRIES

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FUNDAMENTAL SOCIAL PROBLEMS AND THEIR COST

TO talk about the cost of large families may seem to reverse the title of this symposium—may appear to be discussing not the biological aspects of social problems but the social, or at least the economic aspects, of a biological problem. But if we get down to first principles I do not think I am being quite so perverse as all that. What, fundamentally, are the main social problems facing the world? To be precise, five are usually recognized: poverty, unemployment (including under-employment),* illiteracy and lack of education generally, ill-health and crime. I shall be content with the first three—broadly speaking, poverty, unemployment and illiteracy. They have been attributed to a number of factors, economic, political, psychological and sociological. Here I am bringing forward the factor of the large family—surely a *biological* aspect. Moreover, poverty and unemployment as well as ill-health, illiteracy and crime are biologically a cost to the community as leading either to the waste or misuse of human capacity or, in the attempt (not always successful) to solve or relieve them, leading not just to taxation but to the 'real' costs in human effort and sacrifice of organizing welfare and services.

THE COST OF SETTING STANDARDS AT HOME AND IN UNDERDEVELOPED COUNTRIES

The Economic Research Council is just publishing an Occasional Paper in which I consider the public cost of large

* The problem of lack of employment is not merely the poverty that ensues, but also the frustration and loss of self-respect that is involved. Full employment is normally included as well as higher income per head as a criterion in the plans for development of the underdeveloped countries.

families (meaning by that families of five children or more) in Great Britain to-day and the alternative cost of preventing large families. In that paper I quote Dr Dorothy Morgan's estimate that her domiciliary birth control service to 110 parents saved the City of Southampton £473 a month on Child Care alone. I need, therefore, only allude very broadly to the costs at home. But in more detail I shall try to draw the moral for the world at large, including the underdeveloped countries, most of which are planning to tackle the problems of poverty, unemployment and illiteracy by ambitious educational and welfare services.

The first fact to realize is how costly children are if they are not, when adults, to be a drag on the development of the community. An argument often used against birth control is that the population thereby becomes more aged on average and that the aged are, compared with the young, an economic liability. But this is the reverse of the truth. When we examine our own national accounts, as I do in my Occasional Paper, it will be found that more than double is spent by British national and local authorities on the young, than on the old. Apart from the more obvious items of education and family allowances there are school meals and child services, such as Local Authority ' homes ' to be reckoned with. Moreover it is particularly among large families that the social problems of poverty and crime arise which are so costly to relieve and prevent. Between 1948 and 1950, the Eugenics Society sponsored inquiries in five areas into the ' problem ' families known to the local social agencies and helped by public funds of various sorts.[2] Of these problem families that had any children at home 48·5 per cent were found to have five children or more. The Census of 1951 found, however, that of all families with children in the population at large only 2·65 per cent had five children or more.

As formalized in their four, five or six year plans, most underdeveloped countries aspire to raising ' standards of living ' among their population and, quite logically, to becoming welfare states with schooling for all. I say ' quite logically ' because the standards of living should refer not to an average per head but to a minimum level of income below which no one in a community falls—or hardly anyone. To be precise,

we want to know not the arithmetic mean or even the median but the lowest decile or percentile of a country's income distribution. It is to keep this minimum that the measures of social welfare are required and are paid out of public funds—particularly to the larger families. In the past the economy of most underdeveloped countries was just a primitive agricultural system in which the extended family or community, as a matter of obligation, supplied social security. But to-day most of the surplus population unemployed or under-employed in underdeveloped countries is flocking to the cities, usually settling in ' shanty-towns ' without full-time jobs.

This urbanization, breaking up tribal and extended family organization, will, if the aspiration toward higher standards is to be fulfilled, call for the same sort of welfare measures (e.g. family allowances, child clinics, probation and educational services) as in the developed, industrialized countries. As yet, however, not much industry is there to support them; and of course a much greater distance has to be made up between the current level of abject poverty and standards.

THE FUTURE SIGNIFICANCE OF FAMILY SIZE

Underdeveloped countries at present in dire poverty aspire to higher standards of living, including the welfare services that will prevent any citizen falling below a certain minimum; and families with many children are the most expensive item in this programme. It follows that the costs relative to the national income will largely depend on the ratio of present and future children to the general population. As age pyramids show graphically (that of Pakistan is fairly typical) most underdeveloped countries have a much higher ratio of children to adults than have developed countries.[6, 6*] This ' juvenation '

* In an article in the October 1964 issue of *The Eugenics Review* I compared the age pyramids of four underdeveloped countries with that of Pakistan. To quote: " a short test of . . . similarity is to divide the number of 0–9-year-olds by the number in the ten-year group aged 25–34." This intergeneration ratio was for Pakistan in 1960 2·47 . . . for Mexico in 1960 the ratio was 2·43, for both Thailand and Indonesia in 1961 it was 2·15, and for the Philippines 2·60. For England and Wales in 1871 (when birth rates were at their height) it was only 1·73, in 1961 only 1·22.

These five are the only large underdeveloped countries the age structure of which appears in the U.N. *Demographic Yearbook* for 1962.

of the age structure has been an outstanding feature only since 1950, resulting in a very large bulge indeed in the base of the age pyramids and pointing to a greatly increased number of potential parents from 1965 onwards. Two facts loom on the horizon—one qualitative, the desire for minimum standards of living involving particular costs where children are involved; the other quantitative—the sharply increasing number of

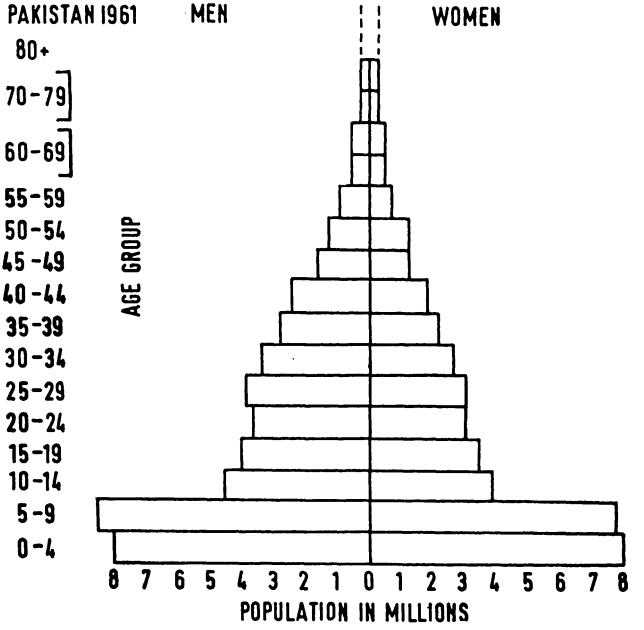

FIGURE 1. Age structure pyramid. Pakistan 1961 census.

potential parents. In view of these two facts, the size of the family (the number of children per parent) obviously becomes crucial. Hence the title of my paper.

Clearly if families remain at their present size the population explosion of to-day will appear quite 'pre-atomic' compared with that to be let loose in ten years time or so. The present explosion is due to a fall in the death rate not (taking countries as a whole) to any increase in the birth rate. But soon, if families remain the same size, the increase in

potential parents will push up national crude birth rates. This, combined with death rates continually falling, will produce an explosion of atomic bomb dimension.

To an audience numerate as well as literate, I must be precise on the size at which families should aim to avoid too great a cost to the community. We must take account of the biological (or is it psychological?) fact that the great majority of women—and of men too—want children. If the atomic population bomb is to be banned this must be brought about, not by depriving some couples of children altogether, but by laying down an average size of family to apply to all. In fact the average is not just an abstract academic expression but becomes of real import to policy. In a closely reasoned paper at Hamilton College (March 1964) Professor Henry Villard took account of all the world's land resources. He was even prepared to concede that desalinization of sea-water and cheap nuclear or solar power can be used to make garden spots of the Sahara and of the interior of Australia, though doubtful what could be done with northern Canada, Greenland, Siberia or Antarctica. In the result, he considered that, for a minimum of food, world population must be absolutely stabilized in a hundred to two hundred years from now. Stabilization implies an average family of only a little over two. Obviously birth rates cannot be brought down suddenly to produce this average size; but we must start, right now, the process of bringing down the birth rate to stabilization level. How can families be reduced in size really extensively and quickly?

THE COSTS OF MASSIVE CONTRACEPTION

To an economist like myself it seems necessary to add a third criterion in judging any birth control method to set beside the criterion of efficiency in controlling conception and social acceptability—namely its economy. The peoples of the underdeveloped countries are poor—to us unimaginably poor—and whether their governments help in organizing and financing birth control or not, they must seek the cheapest method for the *massive* cutting down of birth rates that is

necessary *immediately* if their standard of living is ever to rise appreciably. To be sure, there is aid from foreign countries; but whether from government or private sources (as in the recent Freedom From Hunger Campaign) this has not so far affected standards of living appreciably except in a few specially favoured countries (favoured usually for military reasons) or in the still fewer countries, like Japan, that have taken birth control seriously. Indeed if attention is concentrated solely on increasing production to the neglect of decreasing reproduction as the hunger campaign has unfortunately done, I fear foreign aid may well have made the long-term position worse.

I conclude then, that the biological aspect of the problem of poverty, unemployment and illiteracy in underdeveloped countries (or in classical economic terms, the problem of the means of subsistence not outrunning population as it should) can be met only by including birth control in the programme and by using the cheapest method of birth control.

Unlike the death control which has recently become so successful, birth control cannot be administered by a few experts. The cost of birth control which will be extensive enough to affect a country's population explosion, must include the cost of bringing knowledge to the masses.

If recent sociological *research* rather than sociological *theory* is to be credited,[1, *] a heavy cost is not, in most countries, required to overcome definite motivation toward large families. Motives, in fact, if involved at all, point the other way.† Queen Victoria wrote to her uncle of the great inconvenience a large family would be.‡ In the event, she had nine children. Similarly Birmingham immigrant mothers are 'moved' to have a much smaller family as an ideal than, in the event, they actually produce.[8] No religion except the Roman

* I refer particularly to the field work of Stycos and Bogue and to Berelson and Freedman's account of the Tai-wan experiment.

† See Florence, *Economics and Sociology of Industry*, pp. 195 ff. Large families in Great Britain and one suspects elsewhere are largely due to (1) ignorance of effective birth control methods, or (2) fecklessness even if informed of such methods. In neither case do 'motives' play a part, unless motives to control fertility are thought of as forces strong enough (or too weak) to overcome fecklessness.

‡ Letters of Queen Victoria. See also *Dearest Child*, edited by Fulford, 1964.

Catholic seems positively to condemn birth control, though many religions prevent higher standards of living by reducing productivity and the more equal distribution of wealth. Mohammedan Ramadan and the Hindu sacred cow and caste system need only be mentioned.

In short, except in Roman Catholic countries, propaganda or counter-propaganda to make converts to birth control is not required to any great extent, particularly after two children have been born. The costs of introducing birth control can generally be confined to discovering the most acceptable form of contraceptive and then mass communication to have it as widely accepted as possible.

THE COSTS OF DIFFERENT CONTRACEPTIVE METHODS

Dr Peberdy contributes to this symposium a most important paper on her experience in Newcastle, in applying different methods of birth control among problem families which were, as usual, mainly large families or likely to become so. I need therefore say no more in comparing different methods at home. Abroad, in a still fairly underdeveloped country, a most hopeful experiment in the comparative success of different methods of spreading information on different contraceptive methods has only recently been described and must be reported. The country is Tai-wan.[1] The objective was to learn how much family planning could be initiated at how much cost in money. Throughout the City of Taichung posters were displayed pointing out the advantages of birth control, and meetings held with community leaders to get their co-operation. But in some areas of the city additional methods of communication were applied; (*a*) letters to newly-wed couples or parents with two or more children; (*b*) in the ' everything treatment ' personal visits also, by specially trained nurse-midwives to every wife twenty to thirty-nine years old, and in half the cases their husbands, too. These methods were applied according to sector in three degrees of intensity: ' light ' to every fifth neighbourhood of the sector; ' medium ' to every third; ' heavy ' to every second.

The results were quite encouraging. Forty per cent of the

'eligible' * women took up contraception in the first thirteen months.

TABLE I

COMPARISON OF THE DIFFERENT BIRTH CONTROL METHODS

	CAP	WASHABLE SHEATH	PILL	PLASTIC COIL
A. ACCEPTABILITY—				
	Distasteful to many women *		Requires sustained habit but regularity makes easier	No need for sustained effort
	In Tai-wan only 15 per cent accepted without need of home visit. Preferred to other methods by 20 per cent		In Tai-wan only 2 per cent prefer to other methods	In Tai-wan 75 per cent accepted without need of home visit. Preferred to other method by 78 per cent
B. EFFECTIVENESS				
	High if instructions carried out		100 per cent	In Tai-wan 20 per cent expel or remove after six months
C. ECONOMY				
Materials	A cap once a a year (8s.) + chemicals	2 a year approx. 2s. 3d. each	6d. each, 300 a year £7. 10s.	2d. each not more than one a year
Labour	Doctor for prescribing; nurse for teaching	None	Qualified doctor to supervise	Qualified doctor for insertion
Special Premises	A clinic; one or two visits a year	None	None	A clinic visit once a year

* See Lella S. Florence, 1956 *Progress Report on Birth Control*, pp. 129–30.

A tentative conclusion was that the maximum return for minimum expenditure can be obtained with something less than the heavy-sector degree of intensity and that the added

* Excluding women not already practising contraception, sterile or sterilized, or already pregnant.

effect of visiting husbands as well as wives was not worth the expense. The average cost of getting acceptance was $4 to $8; but a 'take-off' effect is produced after a certain concentration. Information gets round by word of mouth without further organization or cost.

After the costs of introduction come the costs of operation. The items involved in its economic operation can be analyzed like manufacturing costs into:

i. The costs of materials;
ii. The cost of labour: professional, skilled or unskilled;
iii. The cost of premises, fixed equipment and tools.

For various contraceptive methods, a table can then be drawn up, as a final offering, comparing their acceptability, effectiveness and the economy of operation.

It looks, in short, as though the plastic coil were the most acceptable, effective and economic answer for 80 per cent of women at least in the underdeveloped countries.

REFERENCES

1. BERELSON, B. and FREEDMAN, R. 1964. A Study in Fertility Control, *Sci. Amer.* May 1964.
2. BLACKER, C. P. (Ed.). 1952. *Problem Families; Five Inquiries.* London. The Eugenics Society.
3. FLORENCE, L. S. 1956. *Progress Report on Birth Control.* London. Heinemann.
4. FLORENCE, P. S. 1964. *Economics and Sociology of Industry.* London. Watts.
5. FLORENCE, P. S. 1964. The Public Cost of Large Families. Occasional Paper, Economic Research Council. (10 Upper Berkeley Street, London, W. 1.)
6. FLORENCE, P. S. 1964. A Note on Recent Age-Pyramids in Underdeveloped Countries. *Eugen. Rev.* **56**, 143.
7. VILLARD, H. 1964. *The Economics of Population Growth.* New York. Hamilton College.
8. WATERHOUSE, J. A. H. and BRABBAN, DIANA H. 1964. Inquiry into Fertility of Immigrants. *Eugen. Rev.* **56**, 7.

THE RELATIVE ACCEPTABILITY OF CONTRACEPTIVE METHODS AMONG IMMIGRANTS

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FROM the variety of devices now available for the purpose of voluntary limitation of family size, the choice, for peoples of underdeveloped but overpopulated countries, is likely to be made not on grounds of efficacy but of acceptability. The urgency of the need cannot wait for the slow advance of literacy, nor in so meagre an existence can any realistic programme expect to rely upon voluntary purchases in competition with food and the necessities of life. Though cost may be the ultimate arbiter of method, compatibility with religious tenet or culture pattern can be of the utmost importance in acceptance or rejection on a mass scale.

Before embarking on any large-scale programme, a pilot investigation, small in size but intensive in scope, is necessary to discover the comparative suitability of different contraceptive methods to peoples of varied cultural origins. Equally important may be information obtained on the unsuitability of a method, or of a manner of presentation, so that costly mistakes may be avoided. In the critical stages of a nation-wide campaign, a mistake of this kind could, by engendering a population resistance, jeopardize the entire programme.

In common with many other large cities of this country, Birmingham has acquired within the last ten years a large immigrant population. Recently Miss Brabban and I published the preliminary results of a sociological survey we conducted in Sparkbrook, which, of all the wards of Birmingham, possesses the highest proportion of immigrants.[2] We had obtained details of family size and structure as part of our inquiry, and had also asked about awareness of birth control and the methods used. We reported that " one of the important consequences of this investigation has been to reveal the

demand, and the need, for a family planning service in this district ". We went on to say that " our experience suggests that given suitable initial publicity, family planning . . . would be readily accepted and well patronised ". Such findings reinforced our belief that an experimental study of family planning methods and their differential acceptability could usefully be undertaken here in Sparkbrook.

The principal immigrant groups represented are from Ireland, India, Pakistan and the West Indies. In our earlier paper Indians and Pakistanis were combined because their numbers were too small to justify separation, and a fourth

TABLE I

STRUCTURE OF SAMPLE

	NUMBERS		PERCENTAGE	
	(*a*)	(*b*)	(*a*)	(*b*)
English	54	74	21·8	29·5
Irish	69	69	27·8	27·5
West Indian	56	52	22·6	20·7
Indian/Pakistani	69	56	27·8	22·3
	248	251		

Note: (*a*) Groups defined by nationality of male consort.
(*b*) Groups defined by nationality of female.

group, the indigenous English population, were included as a control. In that paper, although in the main it was female-orientated, the groups were defined by the nationality of the male. When the data are re-examined, according to the nationality of the female, the relative composition of the sample is altered, as is shown in Table I, chiefly by increasing the size of the English group at the expense of the Indian-Pakistani group. Table II, showing the nationality of the consorts of women in each group, demonstrates the differences more clearly, showing that the consorts, both of West Indian and Indian/Pakistani women, are from the woman's own group, whereas the two white groups include all four categories.

Table III summarizes the pregnancy experience of the four groups, classified by nationality of mother. Again it appears

that those of the Indian/Pakistani group have the fewest miscarriages, but as we pointed out before, we consider this to be more probably attributable to under-representation than to a genuine difference. A more informative picture of the pattern of fertility by age for each group is shown in the graph of cumulative fertility (Fig. 1). Based here on the nationality of the

TABLE II

Nationality of Woman	Nationality of Consort					No Consort
	English	Irish	West Indian	Indian/ Pakistani	Other	
English	65·5	12·1	5·2	10·3	6·9	21·6
Irish	6·8	81·3	1·7	10·2	...	14·5
West Indian	...	...	100	...	...	19·2
Indian/Pakistani	...	...	...	100	...	1·8

mother, it shows little overall difference from our previous graph. The Indian/Pakistani and Irish groups run closely parallel, as before, the English shows the lowest fertility by age, and the West Indian is intermediate.

Though the Indian/Pakistani group does in fact exhibit the highest fertility rate with age, and proceeds to the largest

TABLE III

Summary of Pregnancy Experience

	Average number of Pregnancies	Average number of Live Births	Women having one or more Miscarriages
English	2·9	2·4	25·7
Irish	3·8	3·0	33·3
West Indian	3·3	2·7	26·9
Indian/Pakistani	3·9	3·6	12·5

overall total of children, this appears not to be in accord with their aspirations. In response to enquiry, we learned that their ideal size of family averaged 3·3 (Table IV), spaced at intervals of three years. Taking this in conjunction with the information on family planning, for which, although only two out of three were aware of birth control, a larger fraction of this group

than of any other group used family planning methods, we gathered that the incentive to limitation of family size is not lacking among Indian/Pakistani women. Irish women, on the other hand, prefer a family of four, with a spacing interval of $2\frac{1}{2}$ years. Their Church permits them to use only abstention, or the 'safe period'; although nearly all were aware of the existence of birth control methods, only about half of them made use of them. If one of the results of the deliberations of

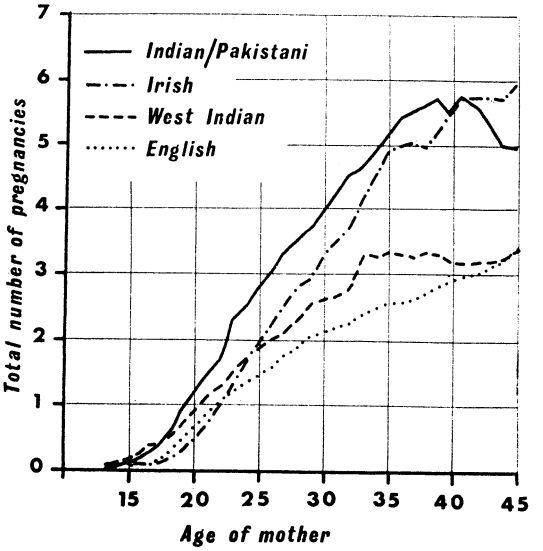

FIGURE 1.

the present Vatican Council should be to lessen the opposition of the Roman Catholic Church to other methods of birth control, it is almost certain to be followed by a stream of Irish candidates for family planning, and to be followed also by a reduction in the number of miscarriages in this group. It is impossible to estimate from our data the number of procured abortions—although we asked the question, we were informed of none—but it is our firm impression that the number of these also would diminish.

Apart possibly from the good Catholics of the Irish group,

who observed the teachings of their Church in the practise of birth control, our findings parallel those of Berelson and Freedman [1] in Tai-wan, ". . . that these women as a group wanted to have a moderate number of children, were having more children than they wanted, approved of the idea of family limitation . . ." The preferred family size for married women of the city of Taichung they found to be four—the same as that of our Irish group.

Berelson and Freedman went on to assess several different methods of ' putting over ' a family planning programme to the eligible population of the city, and in some respects the

TABLE IV

FAMILY PLANNING

	Ideal size of family	Ideal space between children (years)	Not wanting more children * per cent	Knowledge of F.P. per cent	Users of † F.P. per cent
English	3·1	2·8	24·7	93·2	57·4
Irish	3·9	2·5	34·8	95·7	48·5
West Indian	3·2	2·8	42·3	86·5	46·7
Indian/Pakistani	3·3	3·0	45·5	67·1	64·9

* Of those mothers < 45 years old. † Of those with knowledge of F.P.

results we are obtaining from the extension of our inquiry may be comparable with theirs. We are contrasting, however, the relative acceptability of oral contraceptive tablets and intra-uterine devices. Advice, examination and all services (including the supply of contraceptives) are without charge. In exchange we ask for the help and co-operation of each woman —and, whenever possible, of men too—in supplying us with information relevant to the investigation. We endeavour to maintain the groups as near equality in size as is feasible. We cannot expect to fulfil the requirements of a properly-designed clinical trial in a survey of this kind; but we hope to compensate for some of the uncontrolled variation by transferring as many women as possible from one method to the other after an interval of from six to twelve months. To keep the records as complete and up-to-date as possible our

social worker follows up each family by regular visiting. Repeat visits to the clinic are welcomed, and there are also domiciliary visits by midwife, doctor or health visitor.

Because no charge is made for the contraceptives themselves—nor indeed for any clinical service—we have virtually eliminated the cost differential between pill and intra-uterine device, which seems to account in large measure for the preference for devices to pills among the women of Taichung. Our aim is certainly to present each method without bias, and in fact to ask that each be tried in succession. Several regimens are available for the pill, which, within wide limits, are open to free choice. We are also using several different designs of intra-uterine device, to determine the extent of variation in their use and acceptance.

When first invited to participate in this Symposium, I had hoped to be able to present some of our early experience with the experimental study of relative acceptability. In the event this proved to be impossible because of the delay in the start of the trial. Consequently I have been able to describe here only the foundations and groundwork of the investigation, and must leave to a later date any attempt to assess its results.

REFERENCES

1. BERELSON, B. and FREEDMAN, R. 1964. A Study in Fertility Control. *Sci. Amer.* **210**.
2. WATERHOUSE, J. A. H. and BRABBAN, DIANA H. 1964. Inquiry into Fertility of Immigrants. *Eugen. Rev.* **56**, 7.

FERTILITY CONTROL FOR PROBLEM PARENTS: A FIVE-YEAR EXPERIMENT IN NEWCASTLE UPON TYNE

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THIS survey into the use of contraception amongst problem families, conducted in Newcastle upon Tyne, was prompted by the observation that the normal birth control services available to the public are infrequently utilized by that section of the community which would appear to have the greatest need for them. Parents of large families in the lowest income groups rarely arrive at ordinary family planning clinics of their own volition, and even after attendance are unlikely to continue to make use of the service offered to them. It must be remembered that contraceptive advice is not part of the National Health Service and patients usually have to pay, at least for their supplies or, alternatively, to plead poverty.

In January 1959 a pilot scheme was opened for the contraceptive care of such families. It aimed to study the relative acceptability and efficiency of contraceptive methods in this particular group of our society.

Family doctors, hospital specialists, health visitors and social workers were invited to refer those patients who had medico-social problems, and who were willing to be given aid in contraceptive methods, but who were nevertheless unlikely to benefit from the usual facilities. The criteria of eligibility were twofold: first, that the husband be in semi-skilled or in labouring work, or unemployed, and second, that his wife had had at least four pregnancies in the preceding eight years; 150 couples were accepted.

SOCIAL CHARACTERISTICS

The average age of the husband was thirty-four years; that of the wife thirty years. The average number of pregnancies for married life was 6·3. Chronic physical illness was

present in 40 per cent of the women, and in 31 per cent of the men. Mental illness or marked mental instability was present at the rate of 23 per cent for both sexes. Alcohol was consumed regularly by 48 per cent, usually to excess.

The marriage relationship appeared stable in 56 per cent, somewhat unstable in 16 per cent and grossly unstable in 28 per cent. Coitus interruptus had almost invariably been the only method of contraception practised during their married lives.

The majority of the men came from casual labouring workers. There was a high rate of unemployment which appeared to increase during the five years of the survey.

TABLE I

Average age of wife: 30 years	Mental instability—women: 23 per cent
Average age of husband: 34 years	Mental instability—men: 23 per cent
	Alcohol: 48 per cent
Average number of pregnancies: 6·3	
	Marriage stability:
Physical illness—women: 40 per cent	Stable, 56 per cent
Physical illness—men: 31 per cent	Moderately stable, 16 per cent
	Unstable, 28 per cent
	National Assistance, 80 per cent

Over 80 per cent received National Assistance either just before the survey or during the five years under review. The housing standards were very varied. A quarter of the families were re-housed during the survey. The standard of home care in many instances was bad in spite of reasonable housing. Many of the families were well known to the various social and welfare agencies in the city. Children in care and court attendances were common occurrences.

ADMINISTRATION

A weekly clinic session was opened in a maternity and child welfare centre in the poorest area of the city. Patients were encouraged to attend this, but their failure to arrive was invariably followed up by a visit to the patient's home. Initially, one-third were first seen at the clinic but two-thirds requested a visit at home in the first instance. Only about

one-sixth of the patients could reasonably be relied upon to attend the clinic for examination and supplies. It is therefore true to say that the greater part of the clinical work was undertaken in the patient's own home.

The work was conducted by a team working for one or two sessions per week. As the doctor, I was assisted by a nurse skilled in family planning methods, two qualified social workers (a man and a woman), and a secretary.

Wherever possible, both partners of the marriage were interviewed and their attitude to the sexual side of their marriage—with particular reference to contraception—assessed. All the wives were seen by the doctor and the woman social worker, but rather less than two-thirds of the husbands agreed to discuss such matters with our male social worker.

Following the preliminary interview with the wife, who was without exception the first partner to be seen, contraceptive methods were offered. Throughout, we endeavoured to let the patient have as much freedom of choice as possible. Except where oral contraception was medically contraindicated, patients were allowed to transfer from one method to another at their own wish. No charge was ever made for services or supplies. Where necessary, free transport to and from the clinic was also given.

METHODS OF CONTRACEPTION

During 1959 and 1960 the sheath, and cap with cream methods were those generally offered. Oral contraception only became available in January 1961 and was in use for the latter three years.

Sheaths in the form of disposable condoms were offered to all couples. Just over half accepted and used them at some time during the survey. They were given liberally at monthly intervals and, to couples established on this method, distributed by post.

The cap with cream method was also offered to all the 150 women. Two-thirds accepted the fitting of a cap; eighty-three either came or were brought to the clinic for this; fourteen were fitted at home. Spermicidal cream was taken to

the patient at regular intervals, and, as with the sheaths, liberal supplies were given to ensure that failure did not occur by the patient running short of the necessary supplies.

Oral contraception was accepted by just under half of the patients. Twenty-five patients had already withdrawn from the survey before it became available and fifty-five patients were already established on the other methods and did not wish to change. It was prescribed following a pelvic examination with a routine cervical smear. This was usually

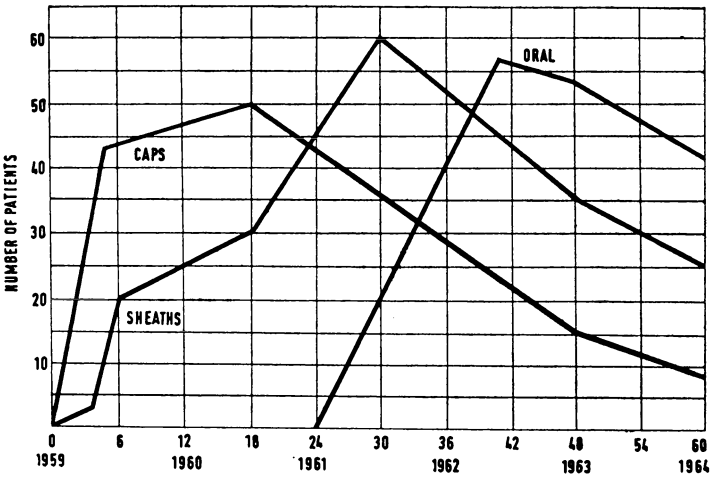

FIGURE 1. Contraceptive methods used in survey.

done at the clinic but occasionally where it was found impossible to persuade the patient to attend, it was done in her own home. The courses of pills were given to the patient at the clinic or taken to her by the doctor or nurse. In many patients this meant monthly visits and, in particularly irresponsible women, even weekly visits. To begin with, oral contraception was offered only to patients who had previously failed on either of the conventional methods during the survey, but as we proceeded it was offered as an alternative method to all patients remaining on the survey.

Figure 1 shows the number of patients using the various

methods as the survey progressed. New couples were accepted during the first two years. It is noticeable, however, that at the end of two years the number of patients using the cap is beginning to decrease, while the number using the sheath is on the increase. The rapid acceptability of the oral method during the first eighteen months of its use is also to be noted.

RESULTS

The rate of conception in the years of marriage up to the time of entering the survey was estimated at 130·2 per 100

TABLE II

PREGNANCY RATE PER 100 WOMAN YEARS

Prior to survey	130·2
During survey	23·7

FAILURE RATE PER 100 WOMAN YEARS

Cap	39·3
Sheath	27·7
Oral	9·0

woman years of exposure. Years of exposure were calculated by assessing the years of married life and deducting ten months for each full-time pregnancy and four months for each miscarriage.

During the five years of the survey the conception rate dropped to 23·7 per 100 woman years of exposure, so that we can reasonably assume that by providing this service the number of pregnancies to these familes was reduced to one-fifth of what might have been expected.

Failures of contraceptive methods can be divided into two categories: firstly, *method failures*, where conception occurs even though the method has been used at every coitus; secondly, *patient failures* where the patient fails to use the method on one or more occasions in the cycle, even though this prescribed method has not been abandoned by the couple as their birth control method.

We found that the number of method failures was so small as to be discounted and therefore the failure rates are in great part also a measure of acceptibility.

The strikingly low failure rate of the oral method as compared with the mechanical methods is significant. There is little doubt from our observations that many patients very frequently failed to adhere to regular tablet-taking, but unless this occurred for sustained periods it did not result in pregnancy. All the failures on this method in this survey were due to omissions of tablet-taking in excess of a week.

Pregnancies occurring while either the cap or sheath were in current use could often be related to an omission to use on a single intercourse.

It is well known that the oral method has a method failure of almost nil and from our observations in this particular group it also has a low patient failure. Seven patients, however, withdrew from this method on account of side effects, a few of which were entirely physical—although some could be attributed to adverse publicity at the time.

Although on the number of woman-months available, the difference between the cap method and the sheath method is not statistically significant, the difference between the failure rates, 39·3 for cap and 27·7 for sheath, is strongly suggestive that of these two methods the sheath is a more acceptable and hence a safer method in this class of our society.

Although the survey was exclusively conducted on social grades IV and V, during its progress each individual was given a personal grading of A, B or C. This in the male was based on work record and the degree to which he undertook his social and domestic responsibilities. In the woman it was estimated on her level of home and child care.

A correlation was calculated to determine the relationship between the failure rate and the combined social grading of the couple. There was found to be a statistically significant relationship between social grading and failure rate, a low social grading being associated with a high failure rate. The correlation coefficient was 0·27. It was also found that when the failure rate of each contraceptive method was rated against the social grading of the partner responsible for that method—in the case of the oral and the cap methods the woman, and in the case of of the sheath, the man—an even closer relationship was found to exist, that is 0·40.

This would suggest that in prescribing contraceptive methods for couples in grades IV and V it is important to take into consideration the separate degree of responsibility of each partner.

TABLE III

150 PATIENTS AT END OF SURVEY

Hysterectomy	11	
Tubal tie	18	
Sheath	26	
Cap	9	
Oral	41	
	—	105
Moved out of area	3	
Lost contact	14	
Deceased	2	
Separated	13	
Pregnant/puerperal or awaiting further advice	12	
IUCD.	1	
	—	45
TOTAL		150

CONCLUSION

At the end of the five years, out of the 150 couples, seventy-six were satisfactorily established on contraceptive methods, forty-one on oral, twenty-six on sheaths and nine on the cap.

Eighteen had been sterilized by tubal tie. With the exception of two cases this was performed immediately following confinement, the pregnancy occurring as a result of a failure of contraception on the survey.

Eleven were sterilized by hysterectomy, performed for gynaecological reasons. It is of interest that seven of these were undertaken for confirmed or strongly suspected malignancy revealed as a result of the cervical smears, which were interpreted on 108 of the women. The incidence of this condition is approximately ten times greater than one would expect to find in the population at large. It is well known that carcinoma of the cervix has a high incidence amongst parous women of these lower social grades.

Three couples moved away from the area. Fourteen couples for various reasons were lost or lapsed in contact. Two patients died during the five years. Thirteen couples permanently separated. Twelve cases at the end of the survey were either pregnant, puerperal or awaiting further action. In one case an intra-uterine device had been inserted.

Since the survey ended last December the seventy-six patients established on contraceptive methods have been taken into the care of the Newcastle upon Tyne Family Planning Association, aided financially by the local authority. These patients continue to have free supplies and home visiting when necessary.

Since then the polythene coil has been accepted as an intra-uterine contraceptive device. Some of the patients referred to in this survey have already been transferred to this method. It is as yet too early to draw any conclusions, but it would seem that a form of contraception which would induce temporary sterility free from patient responsibility would have considerable potentialities in the case of highly fertile women in this section of the community.

THE ACCEPTANCE BY PROBLEM PARENTS IN SOUTHAMPTON OF A DOMICILIARY BIRTH CONTROL SERVICE

DOROTHY MORGAN
Central Health Clinic, Southampton

TO-DAY in our affluent society the existence of ' problem parents ' is much more evident that in previous generations.

A proportion of the population of our towns and cities have failed to adapt themselves to the social and moral establishment, and have become an increasing burden and responsibility to the Statutory and Local Authorities.

These so-called ' problem parents ' were in need of a definite form of help, and one of their biggest problems was family limitation. These families are such that regular attendance at a Family Planning Clinic was beyond their capabilities, and the answer was to provide a Domiciliary Birth Control Service. With this aim I applied for, and received, a generous grant from the funds of the Marie Stopes Memorial Foundation. With the co-operation of the Medical Officer of Health, the welfare workers, social workers and general practitioners of Southampton were informed that the Service was available, and our work started in June 1961, with a team consisting of a nurse, secretary, two male social workers and myself.

At the present time (August 1964) we have 150 families who have accepted some form of birth control.

ACCEPTANCE OF A DOMICILIARY SERVICE

All these families live either in Rehabilitation Centres (flats where families are sent following eviction from Council or private property) or in sub-standard accommodation, mainly blocks of tenement flats, and on my first visit their reception of me was cool. These families are by their very nature ' anti-officialdom ', and the stranger is suspect, whether male or female, for she may be the rent collector, debt collector

or some official asking awkward questions such as why Mr A. has failed to go to work or report to the Labour Exchange; or why Mrs A. has failed to keep a hospital or clinic appointment.

Having gained a foot in the door one is still suspect; for these homes in the vast majority of cases are untidy and ill-kept, and the children inadequately clothed and shod; and care has to be taken not to project on to them the anxieties one has for them, and in particular for their children—this I found most difficult during the severe winter of 1963, when the sparseness of their homes seemed so much worse. If one is accepted, this acceptance is complete and to some extent reciprocal—one is not surprised when asked, for example, " How many flick-knives does your boy possess? "

These families live on their doorsteps and their bush telegraph is second to none, so once you have been accepted in one home entry into the adjoining homes is easier.

BIRTH CONTROL

The main discussion on birth control is reserved for the joint interview with the couple—this is to try and increase the *rapport* with the patient, to see the family as a whole, and to exclude birth control as a cause of marital disharmony; for it is not surprising that in these families, many of whom are co-habiting, there is often friction between the two partners, and the joint interview often helps to sort out sexual problems. Should the husband be awaiting discharge from prison, his written consent is obtained before giving his wife birth control advice. Where the man decides to use the contraceptives the subsequent visits are undertaken by the male social workers.

The oral contraceptive has been available to these families since January 1963, and sixty-eight patients are now on the ' Pill ', which is by far the most acceptable form of birth control because it calls for the least effort.

For the first eighteen months of the Service only the mechanical forms of contraception could be offered, and the occlusive cap with contraceptive cream was preferred to the sheaths. I am afraid the psychologists among you will shudder

when I tell you that I taught my patients of very low intelligence—some registered mental defectives—to use the cap just as you would teach a child to brush his teeth; in fact you can say that something has been achieved when these families have accepted the habit of using birth control.

This in itself is not sufficient. I firmly believe that to teach these families birth control *per se* is not enough, but that the *rapport* between doctor and patient must be such that you give the patients a confidence in their capabilities, however limited, which they have lost. My dream is that this could be used as a boost to improve the living conditions of themselves and of

TABLE I

CONTRACEPTIVE METHODS BEING USED AUGUST 1964

150 FAMILIES

Male contraceptives	17	Left area	11
Caps and Cream	31	Separated or divorced	6
Pill	68	Pregnant	6
Sterilized	3	Discontinued method	8

their children, and the service we give be a corner stone in the rehabilitation of these families.

What do these figures mean? Of the 150 families, 116, i.e. 76 per cent, are using birth control at the present time. What of the other 24 per cent? Six patients, i.e. 4 per cent, are pregnant, or query pregnant; this is a high figure for any clinic, but perhaps some allowance will be made when I tell you that the reason for one of these six being pregnant is because her kleptomaniac aunt helped herself to my patient's contraceptive!

Compared with figures from Family Planning Clinics, the fecundity rates of these patients is high—sexual intercourse four to five times per week, and in some cases this is due in part at least to their insecurity. Not one of my ' problem fathers ' is in a skilled occupation; all work, when employed, as casual or unskilled labourers, and I am sure that many, because of their mental limitations, are the butt of their workmates—tales are heard of them being sent to buy glass nails and rubber hammers.

The mothers, too, are often being reminded of their

inadequacies; so it is hardly surprising that they frequently participate in an act at which they do not feel inadequate.

Further, the urge to procreate is strong in all mankind and where no economic or social considerations deter, this force is proportionately greater.

Eleven families have left the area—the nomadic instinct is noticeable in these families; they move about, believing that the distant fields are greener, and to keep track of them is often impossible. This is not without its tragic side; for example, one family referred to me last year had six children either left with relatives, or in care with Local Authorities from Tyneside to Portsmouth.

Eight patients have discontinued any method of birth control; here we can only say that the service has not been accepted.

ACCEPTANCE BY LOCAL AUTHORITY

As from April 1964 this Service has been taken over and we are now completely financed by the Southampton Local Authority. Table II shows some of the results, which I believe persuaded the Finance and General Purposes Committee to take this step and be the first Local Authority in the country to accept this responsibility.

What has been achieved in three years? Unfortunately I cannot speak of spectacular rehabilitation, but in many homes I can see some improvement, the most obvious being the beginnings of pride taken in the home—wallpaper is replacing peeling distemper and chipped paint.

Tuberculosis, malnutrition and a high infant mortality rate no longer act as the population control factors of previous generations. Something must replace them, particularly in those families who, due to their own ineptitude, are less able to help themselves.

The domiciliary birth control service has been accepted by the far-sighted Local Authority of Southampton because of its benefit to the ratepayers financially, and its benefit in solving some of the problems of the ' problem parents ' not only for our decade, but, we hope, for decades to come.

TABLE II

ESTIMATED SAVING TO NATIONAL REVENUE

	Babies Born	£	s.	d.
June 1959 to June 1961	142			
June 1961 to June 1963	32			
Fewer babies born during domiciliary service	110			
Maternity Services				
Maternity grant per baby, £16 0 0; hence Estimated saving on 110 babies not born, £1760, per annum		880	0	0
Home confinement additional benefit, £6 Assuming 50 per cent would have home confinement, Estimated additional saving, £330, per annum		165	0	0
Child Allowances				
As 110 families would have been eligible for the family allowance of 10s. per week, hence Estimated saving, per annum		1430	0	0
Children's Department				
One child in five from families in the Service was taken into care before or during the Service.				
Cost per child per week in older age group:				
Reception Centre	£13 13 0			
Children's Home	8 5 0			
Foster Homes	2 10 0			
Weighted average cost per child per month, £21 0 0 Estimated cost for 22 children per month, in each year		462	0	0
		£2937	0	0
Estimated total saving on 110 children not born during domiciliary service		£5874	0	0

DISCUSSION

Replying to a question as to whether there had been religious or other opposition to the domiciliary birth control service, Dr MORGAN said she would tell a little story:

" I met the prison padre at Winchester quite early on in the scheme and he said ' Well, you know there are so many of your dads with me at the moment that I think you ought to come and talk to them.' So I was invited to go and speak to the men in Winchester Prison as part of a rehabilitation

course for prisoners before discharge. The morning I was due to speak the Roman Catholic padre happened to look at the speaker for the evening and asked, ' Isn't that the family planning doctor? ' and everybody said yes. He said, ' Well, I'm quite willing for her to come here and speak, but I put her on her honour not to speak on birth control '; which really took the ground from under my feet. The padre said, ' Will you still come, and what do we do? ' I said, ' I think you must tell the men, because he has put me on my honour and if I am asked a question, I am going to be sorely tried not to do a little propaganda '. So it was agreed that they were not to ask me any questions on birth control. At the end of the meeting, as the prisoners were filing out, the padre said, ' It would be very nice, if you do not mind, shaking hands with them.' I of course agreed and shook hands with the first three; and then one prisoner made a gesture with his thumb, and I was absolutely terrified; soon it was happening about every fifth or sixth man. I thought it was some sort of frightful sign that when they got out they were going to beat me up! As soon as the last prisoner had gone I turned to the padre and said, ' What on earth have I done for them to do this to me? ' He said, ' Not at all, those are all your problem fathers you fitted up with birth control; I told them they were not to say anything, so they decided they'd give you the sign.'

I cannot really say I have had any religious opposition."

THE FUTURE OF FERTILITY CONTROL

A. S. PARKES
Physiological Laboratory, Cambridge

MY THEME is the future of fertility control. On the purely technical side this would take us very quickly into the realms of science fiction, and I want to attack the subject in a more general way. I want to try to answer two questions: (*a*) Will fertility control be necessary in future? (*b*) Will it be generally acceptable?

I. WILL FERTILITY CONTROL BE NECESSARY?

The limiting factors of nature

In nature, the first job of a species is to survive and to this end reproductive capacity is such that any animal able to multiply without hindrance to the limit of its potential would soon swamp every part of the world in which it could live. In practice, of course, this does not happen. All species are kept in check by limiting factors, of which the critical ones are food supply, disease and enemies. This principle applies equally to the comparatively slow breeding mammals and to man himself; in human terms, the limiting factors are famine, pestilence and war, the traditional scourges of man. The rigour of these limiting factors needs no emphasis; there is no doubt that during the vastly greater part of human history a high birth rate was almost neutralized by a high death rate, so that increase in numbers was very slow.

The population explosion and its causes

The situation now is very different, as described in Dr Gille's paper. I will mention only one statistic, that in the last thirty years as many people have been added to the human race as were to be found after the first 2000 centuries or so of man's existence in 1830. We are indeed in the early stages of a population explosion, which is likely to double the numbers

of human beings to between 6000 and 7000 million by the end of the century, a mere thirty-five years away.

This unprecedented growth of population raises two very important questions: (*a*) What happened in the last hundred years to bring it about? (*b*) What is going to happen now?

First, taking the world as a whole, the enormous increase in population is not due to increased overt fertility—it has been caused very largely by advances in the medical and related sciences, which have resulted in a comparatively sudden and dramatic fall in the death rate in areas where the birth rate remains high. In such circumstances there are many more young people than old people to receive the benefits of medical science; the immediate consequence is a tremendous excess of young people. We heard about this also from Dr Gille, and it remains for me only to emphasize again the tremendous reproductive potential arising from an age distribution of this kind.

What next?

In nature, the relaxation of one limiting factor allows a population to increase until it bumps up against the same factor again, perhaps in a modified form, or against one of the other two. In the case of man, medical science has greatly relaxed the limiting factor of infective disease, and under natural conditions the present population explosion would be halted by the reappearance of control by disease (using the word in its widest sense), by famine or by war. It remains for man to break the grip of these harsh limiting factors of nature. How is this to be done?

It has often been said that it should be possible to increase food supplies and other resources as fast as the population increases. Let us examine this thesis. The present rate of increase in world population is about 2 per cent per annum. This does not sound very alarming, but it is fatally easy to overlook the explosive force of geometric progression. According to a recent report by the U.S. National Academy of Sciences, if this rate of 2 per cent had existed throughout the Christian era—covering possibly one-hundredth of human history—the increase in population would have been 7×10^{16}, giving

20 million people in place of each one now alive, or 100 people per square foot of the earth's surface. The Academy report goes on to extrapolate into the future:

> If the present world population should continue to increase at its present rate of 2 per cent per annum then within two centuries there will be more than 150 billion people.

That is 50 times as many people as we have to-day, to be fed, housed, educated, exercised, transported and given the amenities of life, and all this in a habitat which in many places is already being destroyed by man's activities. The American report concludes:

> There can be no doubt concerning the long-term prognosis. *Either the birth-rate of the world must come down or the death-rate must go back up.*

This quotation shows the futility of arguments based on the indefinite increase of resources. Even if technically possible to produce, adequate resources would be little consolation to people for whom there was not even standing room.

Pathological togetherness and the peck order

Nor it seems would adequate resources stave off the biological effects of overcrowding. I said a few minutes ago that I was using the word ' disease ' in its widest sense. And here I want to quote from a recent paper by Hudson Hoagland on *The Cybernetics of Population Control*, in which he speaks about the rabbits of Minnesota as follows:

> These populations rise and fall through cycles of several years' duration. There is a build-up followed by a dying-off. Why do these marked oscillations in numbers of rabbits occur? It was observed that when the animals died off there was usually plenty of food—they didn't starve. There was no evidence of an excessive number of predators. Furthermore, the bodies showed no sign of any specific epidemic that killed them.

Here then is a new aspect of population control by disease—the pathology of overpopulation, of which the symptoms are highly suggestive of the stress syndrome characterized by hyperactivity of the pituitary-adrenal axis. And here we run into another important biological principle—the peck order. If half a dozen hens are put into a pen they will soon sort

themselves into a social hierarchy—the peck order—in which No. 1 hen pecks all of the other five, No. 2 pecks Nos. 3–6, but is, herself, pecked by No. 1 and so on to No. 6 who is pecked by all the others and herself pecks no one. This phenomenon of the peck order, in various forms, is of course seen abundantly in mammals and in man himself. The establishment and maintenance of the peck order is necessarily a stressful process, the extent of the stress depending on the frequency of disturbance in the hierarchy—always a painful process. In such disturbance the density of the animals is a major factor because increase of density means increased social contact and hence increased competition.

Hoagland cites many examples of the rise and fall of natural populations where the fall cannot be attributed to lack of food, enemies or infective disease. I want in particular, however, to quote from his account of experiments by Calhoun in which rats were kept at merely double the density compatible with a healthy population.

> The females that lived in the densely populated pens became progressively less adapted to building adequate nests, and eventually stopped building them at all.
>
> Among the sub-dominant males there was much abnormal behaviour. For instance, there was a group of homosexuals. They were really pan-sexual animals, and apparently could not discriminate between sex partners.
>
> Another type of males emerged in the crowded pens. This was essentially a very passive type that moved a good deal like a somnambulist.
>
> They appeared to be healthy, attractive and sleek but were simply zombies in their conduct as far as the other rats were concerned, never engaging in fights or showing sexual interest in the other animals.

Most pathetic of all, he records that the subordinate males, in order to be able to eat and drink in peace, had to adopt the habit of early rising.

Unmaternal mothers, pan-sexuals, somnambulists, zombies and early risers—the horrors of overcrowding!

So much, and you may say quite enough, for the pathology of over-population. For those who dislike the expression, I would recommend the one coined by Calhoun ' pathological togetherness '.

The idea that resources might be increased indefinitely, in keeping with population growth, is thus both unreal and

unhelpful. What then remains? There is no evidence known to me that, whatever may have been happening to demographic fertility, the biological fertility of the human race is decreasing.

The human and humane factor

Some positive human limiting factor is therefore necessary if we are to avoid the harsh limiting factors of nature. This fourth, human and humane, factor can only be the possession of the will and the means for the conscious control of human fertility. The fact is that medical science has put the human race in a position where man's urge to mate can be reconciled with the bearing capacity of our planet only by conscious limitation of the results of mating. Surely, few would wish to achieve this aim by abortion, infanticide or an increase in child mortality.

There remains the control of conception, about which we have heard much in the last few years, and about which I do not propose to say more than a word about the latest manifestation in this field. I refer to the so-called IUCD, intra-uterine contraceptive device, a modern version of the old and cumbersome Gräfenberg ring. The appearance of this device has depended not on increase of biological or medical knowledge, but simply on the development of plastics technology. The advantages of this method of preventing conception are obvious and for the immediate future it may well be the answer to population control in less sophisticated countries. Unfortunately, efforts are being made to discredit the device on the grounds that it is abortifacient. This is surely wrong. It is true that it possibly interferes with implantation rather than fertilization, but this is not abortion. I have repeatedly stated my view as a biologist that conception means implantation of the fertilized egg, not fertilization itself; and you cannot cause abortion before conception has taken place. Biologically, therefore, the IUCD is not an abortifacient; it is a legitimate contraceptive device. On a more philosophical plane, with the use of the IUCD it is not known in any particular cycle whether an egg has been fertilized or not, because the menstrual rhythm is not disturbed, and I do not think you can abort a hypothetical embryo.

I have stressed this matter because the IUCD has emphasized the need for clear and accepted definitions of conception and of abortion, a need which has been evident for a long time, but which is now most urgent. Every effort should be made to prevent the abortifacient smear from hindering development of the IUCDs, and I am prepared to argue the point with biologists, bureaucrats, bishops or any others who wish to take issue on the matter.

II. WILL FERTILITY CONTROL BE GENERALLY ACCEPTABLE?

Motivation

We have seen that there will be an urgent and continuing need for fertility control and we know that methods are already effective and will certainly be greatly improved. There remains the problem of motivation. And here we must consider two quite different aspects of the problem, motivation towards family planning and motivation towards population control. Family planning implies the limitation of the number of children and spacing of births in the best interests of mother and child and the rest of the family. Motivation towards this ideal is now spreading widely, but its progress has been obstructed to an extraordinary extent by ignorance, taboo and dogma.

Fortunately, things are now changing very rapidly. Ignorance can hardly survive indefinitely the impact of modern methods of mass communication, education must surely put taboo in its proper ethnological setting, and whatever may be happening in theory, Catholic dogma on birth control is crumbling in practice. A young woman, a devout Catholic, married to a doctor working in one of the poorer parts of London wrote to me recently saying " as a result of what my husband sees here, I can no longer accept the Church's teaching on birth control ". A formula to reconcile Catholic doctrine with the circumstances of the modern world cannot be far off. In the meantime, the vast majority of the human race, which holds different views, should refuse, and in fact is refusing, to be intimidated by a small minority which in any

case will have to change its mind sooner or later. There are also significant stirrings within the World Health Organization, which recently published the report of a scientific group convened to advise the Organization on the present state and future needs of the study of human reproduction.

Family size and population growth

Family planning, then, is well on the way, but good family planning must often be compatible with a family size of four or even more. How does this fit in with population control, which I take to mean limiting the rate of population growth to an acceptable level? First, what would such a level be? We have seen that an annual increase of 2 per cent cannot be maintained indefinitely. Let us say, for the sake of argument, that a rate of increase of 0·5 per cent per annum would be acceptable. What average size of family would this rate of growth imply? And here I reproduce a table, from a recent issue of *The Eugenics Review* (**55**, 250).

Average Number of Children per Married Couple in Completed Family	Rate of Growth of Population Per cent per annum
2	...
2½	¾
3	1½
3½	2¼
4	3

There is here an acute problem of social, as opposed to personal, motivation. Will people want to limit their families to a size which in the light of current social and medical trends is compatible with an acceptable rate of population growth—that is, according to these figures, to an average family size of less than 2½?

The right to reproduce

This raises in acute form the question posed by A. V. Hill in his Presidential address to the British Association in 1952, the question of whether the rights of man, about which much is heard, can under modern conditions be held to include the

right to unlimited reproduction. Under present and foreseeable future conditions the answer must obviously be "no"; but the task of persuading people, even in the West, that the average size of family should be nearer two than three is not going to be easy. Evolution has endowed man not only with an urge to mate, but also with a parental urge at a level appropriate to a low survival rate. Medical science has altered the one, but not the other. Our present dilemma is indeed a striking example of the effects of unilaterally interfering with nature. What must be done to redress the balance? We cannot put back the clock of medical progress and we cannot cope on the planet Earth with unlimited numbers of people. Emigration to other planets is not likely to be a practical solution within the visible future. Mass reduction of fertility, as for instance, by the medication of a staple food, would be possible only under a rigid dictatorship. Possibly economic pressures and inducements will have to be employed; they are apparently already being used in some parts of the world. Certainly, by some means, the parental urge must be further diverted from quantity to quality, with all that quality implies. I do not pretend to know the answers to these problems, but of one thing I am sure—that the acquirement of the means and the will to adjust its fertility to the capacity of its environment, and so to avoid the harsh limiting factors of nature, will be a critically important step in the continued evolution of the human race.

CONCLUSION

SIR JULIAN HUXLEY

May I make one or two comments on Professor Parkes's paper? I think he might have drawn attention to the fact brought out by Sir Alexander Carr-Saunders many years ago in his great book on population (you will remember that Carr-Saunders was trained as a biologist) that, so far as can be discovered, all so-called primitive peoples employ some method for checking excessive population increase. We were given examples of the influences of stress; when Charles Elton began his notable study of periodic fluctuations in mammals, he first thought that the sudden ' crashes ' in population were due to a bacterial or viral disease; now they have been shown to depend primarily on psycho-physiological stress arising from overcrowding. This is of immense importance for man too, because in him stress will manifest itself in mental instability and all kinds of social and political disturbance.

Professor Parkes mentioned the absurd idea of providing a home for our surplus population on other planets. I was lecturing about this in America recently and I brought home the difficulty of doing so by explaining that the net surplus of human population is the equivalent of eleven baseball teams, complete with coach, every minute of every day. That is quite a tall order to export to the nearest habitable planet, provided there is one!

I was very glad he mentioned the Report of the National Academy of Sciences in America; it seems to me quite admirable. I hope that the Royal Society here may do some similar work applicable to this country, where conditions are very different. In any case, we here are already overcrowded. Here is a nice statistic: if every man, woman and child in this island were to go to the seaside on the same day, they would have just over one linear yard each, which, considering our highly indented coastline, is rather surprising.

Then there was his point about resources and habitat. I have come across numerous cases where animals are destroying their own habitat owing to man's interference with their conditions of existence—for instance the hippos in north-eastern Uganda and the elephants in Tsavo National Park.

If man interferes by killing off predators because the farmers complain they kill their domestic stock, the wild prey may be adversely affected. Thus, when the pumas on the north side of the Grand Canyon were exterminated, the deer first of all increased enormously, then ate themselves literally out of house and home until they were almost extinct. There are cases where cats have been introduced to oceanic islands and have simply eaten the whole supply of prey, to die out finally themselves.

This destruction of natural and favourable habitats has been going on since the dawn of so-called civilization. The whole of the northern Mediterranean and the Middle East was once heavily afforested. Plato himself drew attention to the amount of deforestation that had taken place in Greece; he thought it was a matter for pride, showing how efficient the Greek farmers were, but he would not say so now. Tunisia was once the granary of Rome; now it is semi-desert.

Professor Parkes showed a graph which, if population increase continued at its present rate, would go into the room over our heads. Well, according to certain religions a very large number of people will go prematurely into the celestial room above this earth if we go on exploding at this rate. However, perhaps a new Catholic doctrine is emerging, founded on a new Rock!

I just want to add another point to my comments on Professor Parkes's paper. I was very glad that he drew the distinction between family planning and population control, the one being essentially individual with slight social repercussions locally, the other being essentially national and, I hope, soon a matter of national policy. A questioner has suggested that not sufficient emphasis has been given to the part played by war in controlling populations; quantitatively war has made very little difference. I remember a remarkable graph that Raymond Pearl produced after the First World War: it showed the steady increase of world population up to 1914; then there was a four-year dent; then it went up again and resumed its old course on the same old curve.

I entirely agree with the participant who urges a mass handout of contraceptives along with food. I look forward—going for a moment outside the biological aspects, but everything

ties up—within five or at the most ten years to a total remodelling of all our so-called aid and assistance, whether unilateral, bilateral, regional or international, to underdeveloped countries. I believe that we shall come to the conclusion that what we are all engaged on is a co-operative attempt at world development and that when, for instance, a country asks for aid it will be scrutinized for what I yesterday called 'demographic credit-worthiness', and that this will be taken into account. Then, if it is decided that a particular aid which is asked for—food, shall we say, or help in building industrial plant—is in the long run actually going to do harm demographically and socially, the aid given will make it a condition that the receiving country shall accept part of the aid in the form of advice and medical help in a policy of population control. Everything has got to be integrated. At the moment it is completely chaotic. We have not got an international ecology going; that is our next job.

To the questioner who deplores the discrediting of IUCDs as so-called abortifacients and wants to make the word 'abortion' respectable by legal reform, I would say that certainly we must reform the abortion law, which is at the moment absolutely monstrous; but it will be very difficult to take away the stigma. This may largely be a semantic matter, but every problem in this world has its semantic aspect, and we must try to use terms with less provocative semantic connotations.

As a biologist, I agree with Professor Parkes that you really cannot talk of a new conception until after implantation. What you have before that is merely a mass of cells which will not develop further unless implanted; it is not in any sense a proper conception of a human being.

Professor Meade has told us that in the Freedom from Hunger Campaign the undergraduates at Cambridge have for the last two years carried out the policy of collecting money both to support projects of economic development and to support the Family Planning Association in Hong Kong, in spite of great pressure not to include the F.P.A.* This is another example of what I was pleading for, the integration

* I would like to make the further suggestion that Oxfam and the F.P.A. should march under a common banner entitled *Oxfamplan*.

of all projects and ideas concerned with the proper development of the world as a whole.

Sometimes ideas get wrongly integrated. About ten years ago in India, as you all know, the rhythm method was tried as a pilot project, and the women were given necklaces with beads for ' baby days ' and ' safe days '; it did not work very well. I am told that you can now see ladies who (in spite of what Dr Tanner and others have said about the mounting age of the menopause) are clearly well beyond it, wearing them as a status symbol, guaranteeing their youth and full femininity!

Mr Cadbury has told us that in July this year, after a very long battle, the Economic and Social Council of the United Nations recognized the International Planned Parenthood Federation as entitled to consultative status. I have had similar difficulties in getting even old-established bodies recognized in UNESCO, still more with forming and getting recognition for new ones; but each of these acts of official recognition is a step forward towards this integrated planning that we need. One encouraging fact is that the British Medical Association next year is going to hold a plenary session on population. This is the first time that this highly organized and important professional ' trade union ' has dared to handle this thorny problem. It will be an important occasion for this country.

I think we all agree that these two days have been most rich and fruitful. We have heard a great many encouraging things. The papers about Newcastle and Southampton were truly inspiring, showing what can be done by devotion and patience backed by knowledge; and we have had Professor Parkes, one of the world's great authorities on reproduction, to give us a fascinating account of progress and prospects in the subject.

I think we have all come to the conclusion that the population explosion is part of a great world crisis. It is a world crisis in itself, but it is part of a still greater one where we have suddenly realized that our own evolutionary advance (which goes on primarily by cultural means) is reaching what may be a point of no return, owing to our abusing the world's resources and destroying our own habitat. I believe that the realization of this fact is going to mark the next decade, and I am sure that this meeting has made an important contribution to that realization.