BIOLOGICAL FACTORS AFFECTING FAMILY SIZE

BY

F. A. E. CREW

Latterly much attention has been attracted, and rightly so, to the number, variety, and potency of the socio-economic factors which strongly incline married couples deliberately to restrain their fertility and thereby to limit the size of their families. A tendency to overlook a number of purely biological factors which work in the same direction is to be discerned. These latter are for the most part not under the control of the will of the individual and are as yet far beyond the powers of the medical and social sciences in application. It is not suggested that these factors, as judged by their effects, play as important a role in determining family size as do the socio-economic, or that pronatalist policies are mistaken when based on the demonstrably reasonable assumption that if only the people could be persuaded to unleash their fertility the total size of our population could easily be maintained at its present level or even enlarged. Nevertheless, it is surely desirable when population policies are being fashioned that all and not merely the more potent factors known to affect fertility shall be taken into account.

IMPAIRED FECUNDITY AS A LIMITING FACTOR

As the outcome of the experience of sterility, family planning, and similar clinics and of medical practitioners and specialists, and as the result of a number of specific enquiries, there has accumulated a mass of information of a scientific kind concerning the incidence of childlessness and of small family size (one or two offspring) among long-married couples. There is general agreement concerning the following conclusions (see Lane Roberts and others, 1939; Barton and others, 1943; Engle, 1946; Editorial, *British Medical Journal*, 1946; DeLee and Greenhill, 1947; Whelpton and Kiser, 1948, for example).

Not less than 10 per cent. of all marriages in this country, as also in the United States of America, remain permanently fruitless. Matthews Duncan arrived at the same conclusion in so far as this country was concerned as long ago as 1884. This barrenness cuts right across the social stratification; it is only very exceptionally deliberate and in the overwhelming majority of instances it is due to

persistent infecundity on the part of one or other of the married partners. Those who state so confidently that of the marriages initiated each year one in every ten is destined to remain permanently fruitless do not indicate the route they travelled to reach this conclusion. Nevertheless, there is much which would seem to give it credence. Thus, of all the married women (excluding the widowed and the divorced) whose deaths were registered in Scotland during 1945:

563 (25.1 per cent.)	though married for 10-14 years
	died childless
667 (21.7 per cent.)	though married for 15-19 years
	died childless
1,719 (19.7 per cent.)	though married for 20-29 years
	died childless
2,163 (12.1 per cent.)	though married for 30-39 years
	died childless

But a certain caution must be exercised when labelling a marriage as being permanently barren whilst the partners are still alive and the woman still menstruating, for in Scotland in the same year of the 88,612 women who produced offspring:

32	had their first child after 20+ years of marriage
142	had their first child after 15-19 years of marriage
444	had their first child after 10-14 years of marriage

The same caution is demanded when an unexpected first pregnancy in an ageing woman follows immediately and dramatically upon the desperate trial of some new medicament or therapeutic system or occurs as a startling sequel to the adoption of a child. However, whatever the exact proportion of permanently barren marriages may be, it remains a most disturbing and challenging thought that a very considerable number of such marriages do occur each year and that, furthermore, very large numbers of married couples, having produced one or two children, thereafter remain unable to produce any more, however ardently they may wish to do so. This lowered fecundity on the part of either the male or the female partner is responsible for the relative infertility exhibited by some 60 to 90 per cent. of small-family couples. There is much evidence, which is continually being reinforced, that after the first or second pregnancy the power of married pairs to reproduce shows a definite tendency to fall.

The ascertained and suspected causes of such

infecundity are numerous: for example, in the female, major degrees of uterine hypoplasia, cervical erosion, tuberculous endometritis and salpingitis, uterine retroflexion, occluded tubes, certain of these conditions often being the sequelae of a previous abortion or pregnancy; in the male, ozoospermia, oligozoospermia, necrozoospermia, asthenozoospermia (possibly associated with a deficiency of the enzyme hyaluronidase so that the follicle cells surrounding the ovum are not dispersed), sometimes the sequelae of cryptorchidism, mumps, gonorrhoea, or non-specific epididymitis. Of the male partners examined on account of the infertility of a married pair, at least one in five shows abnormality of the spermatozoa of one kind or another. Infrequent coitus is regarded by some as a cause of infecundity in the male and abnormal utero-tubal irritability as a cause of the same condition in the female. Inability on the part of the spermatozoa to penetrate the interface between semen and cervical mucus has been shown to be causally related to infertility (Barton and Wiesner, 1946, 1948). Hormone imbalance in either sex is commonly blamed (Christie Brown, 1948, for example). It has been suggested that antibodies can be elaborated in the blood of the female partner as a reaction to hyaluronidase and that these antibodies can prevent conception by precluding the action of the enzyme upon the follicle cells (Rothschild, 1947).

Though some gynaecologists of wide experience present the view that the artificial deferment of pregnancy leads to infertility (see Siegler, 1945, for example), the great majority of studies of this matter (see Barton and others, 1943; Whelpton and Kiser, 1948; Dickinson and Morris, 1941, for example) would seem to show quite clearly that the use of contraceptive practices (other than those involving intrauterine appliances) in the earlier years of marriage has no discernible effect upon the fecundity and fertility of couples who later decide to have children.

It seems to be agreed that in a very large proportion of barren or small-family marriages no physical cause for the infertility is to be identified. It is not surprising, therefore, that attention, somewhat timid in nature, is now being paid by gynaecologists to the possibility that psychological factors are operating to a degree previously unsuspected. Thus Stallworthy (1948), having called attention to the possible significance of functional or intrinsic dysmenorrhoea, vaginal spasm, vaginismus, functional bleeding and amenorrhoea, plucks up his courage and with the air of one expecting professional ostracism as a reward for daring asks whether

it is not possible that a woman may deprive herself of the conception she desires or fears because of the very strength of her desire or of her fear. Walser (1948) goes further. He records that some 30 per cent. of those who attend the sterility clinics with which he is associated show no demonstrable cause for their condition and boldly expresses the firm opinion, based on his clinical judgment, that some of these are undoubtedly childless because they are afraid.

If it is assumed that emotional disturbances can possibly find their expression in some form of functional abnormality which renders the fruitful meeting of ovum and spermatozoon unlikely, a more satisfying explanation of a number of somewhat puzzling observations can be reached. For example, Sydenham (1946) records that 53.67 per cent. of the 655 women between the ages of 15 and 45 interned by the Japanese in Hong Kong in 1942 suffered from amenorrhoea of more than three months' duration and dating from the time of the capitulation. She does not doubt that the initial cause was emotional shock. An intriguing question at once presents itself. Is such amenorrhoea associated with non-ovulation? The temperature test for ovulation applied to a series of cases of amenorrhoea caused by emotional disturbance might, as is suggested by the experience of Tomkins (1946), yield information, of great value. Bearing on this question also is the authentic account of the reproductive history of the Dutch women in 1944-45, the starvation year in the Netherlands. Clement Smith (1947) reports that during that year the conception rate underwent a sharp fall, that half the female population suffered from amenorrhoea, and that 50 per cent. of the other half menstruated very irregularly. He quite reasonably presents the view that the cause was malnutrition. It is of interest to note, however, that the stillbirth rate, the neonatal mortality rate among infants born in hospital, and the ability to breast-feed were not significantly affected. At this time Holland was an occupied country, the very atmosphere of which was rank with hatred and fear. With the rising tempo of the war the hope of liberation, long deferred, seemed almost within reach so that tortured hope battled with black despair. Surely if psychological factors can affect ovulation, the passage of the ovum onwards toward the uterus, conception, or implantation, their effects would be revealed under such circumstances. And if it be granted that psychological factors can so affect the female it becomes possible to accept the psychiatrist's teaching that the same factors may be related causally to many of the

varieties of abnormality of the male contribution to conception.

At present it would seem that general medical therapeutics can repair infertility in about one-third of the cases that present themselves for advice and treatment. It is to be expected that developments in reproductive physiology and gynaecology will soon yield added power to repair much of the infertility that is based upon developmental imperfection and to prevent much of the infertility that derives from obstetrical mischance or mismanagement. But if, as seems probable, much of the existing infertility is the expression of the action of psychological factors yielding fear or profound anxiety, the therapy demanded must be of quite another kind and must concern itself with the education of the citizen and with the cultivation of new aspirations and new loyalties. Moreover, it will become urgently necessary to enquire whether or not this impaired fertility is a phenomenon that is commonly associated with, caused by, the disharmonies which manifestly exist between the biological nature and needs of mankind and the peculiar social organizations and economic systems that civilized man has evolved; whether it is in large measure a reaction on the part of individuals and groups to conditions and circumstances in the external world that are not conducive to the maintenance of reproductive efficiency.

GENES AS LIMITING FACTORS

In forms other than man it is firmly established that fecundity rests, partly at least, on a genetic basis—there are "fertility" genes. Studies of human pedigrees yield evidence of a sort which suggests that this is also the case in the human subject (see Penrose, 1936; Gates, 1946, for example).

Reproduction implies much more than the production of a newborn child. This child must live to reproduce in its turn. There are genes that slay the individuals possessing them before the reproductive phase of life is reached or finished. The genes which are the causes of Morquio's osseous dystrophy, xeroderma pigmentosum, epidermolysis bullosa, brachyphalangy, telangiectasis, thalassemia, haemophilia, acholuric jaundice, Cooley's anaemia, Werdnig-Hoffman's progressive spinal muscular atrophy of infants, subcortical encephalopathy of Globus and Strauss, encephalitis periaxialis diffusa, Tay-Sachs' amaurotic idiocy, and glioma retinae are examples of these. By their action they tend to limit the size of a sibship. Thus haemophilia, caused by a sex-linked recessive gene, extinguishes the life of the homozygous female *in utero* and

greatly shortens the postnatal life of the affected male. He rarely reaches the age of twenty-five, and the few who do and marry have but scant time to reproduce. On the average, haemophiliacs produce only about a quarter as many children as do normals. It is established (see Cappell, 1946; Race, 1948, for example) that in erythroblastosis foetalis, another disease genetically determined, the production of the maternal antibodies in response to the foetal antigens is so slow and gradual that whilst the earlier members of a sibship remain unaffected the later ones are destroyed (in the absence of appropriate therapy). Mongolism is a disease the causation of which is the interaction of the peculiar genotype of the foetus and certain peculiar conditions of its environment, the maternal organism. It is a disease which is generally exhibited by the later members of sibships and, though not in itself lethal, so handicaps its exhibitors that reproduction is impossible and life expectancy greatly reduced.

The existence of these genes is clearly demonstrated in the records of stillbirth and infant and child morbidity and mortality. From what is known concerning the prevalence and action of similar lethal and sub-lethal genes that operate during the earlier stages of embryonic life in experimental animals and plants it is safe to postulate that in man also such genes are responsible for much embryonic and early foetal death and thus for much abortion and apparent infertility. For example, Waterhouse and Hogben (1947) have offered convincing evidence that A—B—O iso-immunization is responsible for the loss of about 25 per cent. of the A children expected from marriages A × O, or about 3 per cent. of all conceptions. The action of such genes is evidenced in the non-appearance or numerical deficiency of certain expected phenotypes in sibships in pedigrees in which the abnormality figures, such non-appearance or deficiency yielding a disturbed secondary sex-ratio when a differential sex lethality is involved, as is frequently the case.

THE BIOLOGICAL NATURE OF THE HUMAN FEMALE AS A LIMITING FACTOR

The number of offspring produced at one and the same time by the female of a species must necessarily affect the total number of offspring produced by her during the whole of her life. A survey of the different species in their order of zoological classification strongly suggests that between the place of a species in the zoological scale and the number of offspring produced by the female of that species at one and the same time there exists a direct

relationship—the “higher” the species the fewer the offspring. This observation, to which there are, of course, many exceptions, has provoked much speculation. Spencer (1899), for example, made the reasonable suggestion that between the maintenance needs of the individual's own life (individuation) and the energy requirements of reproduction (genesis) there is a conflict and that the two are inversely proportional, the long-lived species being remarkable for the smallness of their litters. Wood Jones (1915) presented the argument that offspring can under certain circumstances constitute a definite handicap to their parents, interfering more or less seriously with their usual behaviour patterns. Necessarily there must be some connexion between the conditions of the habitat and the behaviour of the parents on the one hand and the reproductive behaviour of the species on the other. Only in those in which the pregnant and nursing female is not too greatly disadvantaged by carrying or rearing a large number of offspring are large litters possible, and only in those species in which the parents provide a “nursery”—a nest, a burrow—in which the young can find shelter and protection are large litters to be encountered. Marshall (1922) hazarded the suggestion that the average number of young produced at one time by the female and the average size of the individuals of the species are inversely proportional, the larger the animal the smaller the litter. Fisher (1938) introduced the notion of reproductive economy. If a species is to flourish, then, assuming that the conditions of the habitat do not change too drastically, what is required of the individuals of one generation is that they shall produce a succeeding generation at least numerically equal to their own. The well-being of a species or population can be measured by the movement of the net reproduction rate. Population size is maintained when every fecund female in one generation is represented by at least one fecund female in the next.

In general there are two ways in which population size can be maintained or increased, one extravagant, the other economical. In the first, far more than enough offspring are produced, the excess being removed by gross mortality. Thousands of ova are made available by each female for fertilization at the same time, but by no means all are fertilized. In such forms external fertilization is the rule and there is a complete disregard on the part of the parents for their offspring. The newly born young are more or less well equipped for the satisfying of their own immediate biological needs, but being unprotected they suffer great loss from mishap and they form the food supply of their own and other

species. In the second, the number of ova produced at any one time by the female is reduced to the absolute minimum, fertilization is internal so that conception is made certain, and to the immature young, made precious by their very paucity, are given by their own parents great care and protection.

It is in the amalgam of all these various hypotheses that the correct explanation of all the different observations is most likely to be found. In general the larger and more complicated the creature is, the longer is the period of time demanded for its growth and differentiation. The longer this period is, the longer is the phase of immaturity and dependency of offspring upon adults of its own kind. The greater and the longer this dependency is, the fewer the offspring that can be given parental care at any one time. Parental care of the immature young can best be provided in an extra-corporeal uterus (a micro-environment equipped with facilities for protection and food supply): a nest, burrow, nursery. The fewer the offspring born at one time the fewer the ova that need to be shed. The fewer the ova, the more certain must fertilization become. For the accommodation of one fertilized ovum a non-cornuate uterus suffices; for the reception of many a bicornuate uterus is required (see Wood Jones, 1945). Where many offspring are born at a time to a mammalian female she must offer a number of mammary glands at least equal to the number of offspring. When only one is produced one mammary gland is required (but the rule of symmetry operates to yield a pair). In those mammalian forms which are monotocous there is a direct relation between the anatomy, habitat, and habits of the species and the particular pair of mammary glands that is retained.

The human female is characteristically monotocous and her anatomy is in accord with her reproductive habits. She extrudes as a rule only one ovum at each ovulation during the period puberty-menopause. Exceptionally she reveals her phylogenetic relationship by shedding more than one ovum (polyovular multiples) or, like the armadillo, produces an ovum which after fertilization divides to give rise to several embryos (monovular multiples). Fertilization is internal and conception certain if intercourse occurs shortly before or after ovulation (and there are no pathological barriers). Her uterus is non-cornuate, suitable for the accommodation of a singleton. She has but one pair of mammary glands, but, unlike the marsupial which often produces young in numbers greater than the number of her teats so that some must necessarily die, the human female can attach the surplus to a bottle. The human young is so immature at birth

and demands so long a period of time to achieve self-sufficiency (and for initiation into society) that in man parental care reaches its highest degree of expression. So it is that in man the family grouping reaches its maximum development and in a human society social agencies share, reinforce, and extend parental responsibilities by means of crèches, nurseries, schools, universities and the like, and by the provision of foster parents: nurses, teachers, policemen, and others. By these and similar means any conflict between individuation and genesis can be resolved and the reproductive habits of our species are no longer necessarily limited by the circumstances of our external world, since these latter can be changed by man.

Parental care reinforced by communal care does much to reduce mortality among the young. The application of science by means of sanitary laws and habits, raised standards of living and of education, advances in medical knowledge, have played their parts in reducing infant and child mortality and in extending life expectancy. Since such reduction has outpaced the fall in the birthrate it is eminently possible for a population such as our own to maintain or even enlarge its total size with comparative ease if it so desires. Monotocia suffices.

THE HAZARDS OF POLYTOCIA AS LIMITING FACTORS

It would seem to be generally agreed that not more than 20 to 50 per cent. of all the twins that are conceived are alive at the end of the period of pregnancy. Twins are commonly reduced by foetal death to a singleton, triplets to twins or a singleton. The observed facts concerning stillbirth, infant mortality, and maternal mortality in relation to multiple births support the view that the human female is not equipped for the production of more than one young at a time.

Stillbirth is several times as common among twins as among singletons. Munnell and Taylor (1946) found that the gross foetal mortality (28.1 per cent.) among multiples was four times greater than the gross foetal mortality rate (7.0 per cent.) for all deliveries, and that the corrected foetal mortality for twins (9.1 per cent.) was almost twice as great as the ten-years' average for all deliveries (5.5 per cent.). According to Hirst (1940) stillbirths are five times as common among multiples as among singletons. Burns' (1942) figures show that it was two to three times as common. The figures for Scotland for 1945 were 68 per 1,000 for twins and 33 per 1,000 for singletons.

Authorities are agreed that dystocia and birth injury are far more common among multiples for

the reason that their mutual interference during parturition tends to lead to faulty presentation. As a result of birth injury mental defect has a higher incidence among multiples than among singletons.

Prematurity is commoner among twins than among singletons and is universal among triplets and higher. Kerr and others (1944) state that premature labour occurs in about 70 per cent. of all cases of multiple births. It is the outstanding cause of the greatly increased mortality among them. Baird (1945) records that multiple pregnancy was associated with 12.2 per cent. of premature births, whilst Crosse (1945) puts the figure as high as 16.6.

Neonatal death is twice as common among multiples as among singletons, as would be expected since so many of the causes of stillbirth continue to operate during the neonatal period. The toxæmias of pregnancy, polyhydramnios, uterine inertia, prolapse of the cord, and post-partum haemorrhage are all much more frequently encountered in multiple pregnancy (see Munnell and Taylor, 1946). Hirst (1940) states that neonatal deaths are five times as frequent among multiples as among singletons. Burns (1942) found that the neonatal rate for multiples was about six times that for the whole group, including both multiples and singletons, studied. The postnatal rate for multiples in Burns' (1942) survey was twice that of the total population at risk.

Maternal mortality is higher in multiple pregnancy. Hirst (1940) found it to be, as also did Burns (1942), twice that of singleton pregnancies on account of the greater incidence of haemorrhage and toxæmias.

So far medicine has not gained the power to overcome these hazards of polytocia.

THE RATE OF REPRODUCTION AS A LIMITING FACTOR

After the first pregnancy the rate of reproduction would seem to have a direct influence upon maternal and infant mortality and stillbirth. The advocacy of "spacing" on the part of family planning clinics would seem to be more than justified. The state of health of the mother influences in a general way the state of health of the unborn child. Child-bearing, and especially long-continued lactation, is a strain from the effects of which the mother recovers, though perhaps never completely in a great number of instances, in time. Exposure to the same strain before recovery increases the hazards of pregnancy for both mother and infant. Such mothers commonly produce weakly babies among whom the neonatal death rate is high (Burns, 1942). Birth intervals of less than two years predispose to prematurity (Joint Committee, 1948).

THE HAZARDS OF PARITY AS LIMITING FACTORS

Stillbirth, infant mortality, and maternal mortality are all related to parity. It would seem that either the strain of reproduction is cumulative or else the reproductive efficiency of the human female deteriorates with an astonishing rapidity. It is to be stressed, however, that it is not always a simple matter to disentangle the effects of maternal age and of socio-economic circumstance from those of parity itself.

The probability of a stillbirth is least in the case of a second pregnancy and thereafter rises with increasing parity. The probability of a stillbirth in the case of a first child is greater than in the case of a fourth but less than in the case of a fifth. The stillbirth rate is higher at all ages of the mother for primiparae than for multiparae of the same age, the rates for both increasing after the age of 25 (Sutherland, 1946), and of the two factors, parity and maternal age, the former would appear to be the more important.

Prematurity is least among mothers aged 25 to 35. First babies are more often premature (8.0 per cent.) than those resulting from subsequent births (5.3 per cent.). For all birth orders premature births are more frequent among the poor, the cause for this being multiple: maternal age, parity, inadequacy of antenatal care, nutrition (Joint Committee, 1948).

Neonatal mortality, like stillbirth, decreases with increasing parity up to a point for the reason that first births carry an extra risk. But Burns (1942) found that among seventh and later children neonatal deaths (including stillbirths) were greater than the total deaths (including stillbirths) up to the age of five in the case of the first, second, and third children, and presents the view that this early death among later sibs would not be seriously affected by any system of family allowances or such like.

Postnatal mortality cannot so easily be related directly to parity. It increases with family size for the reason that large families involve an extra risk of infection of the most recent addition by its older sibs. In this country, too, there is a direct relation between family size and the socio-economic circumstances of the home: larger families are found for the most part among the poorer strata in which the women seem incapable of planning their lives (*Lancet*, 1945). Thus it is that large family size is so commonly associated with low income, thriftlessness, poor educational standards, low standards of living, insanitation in the home, and avoidance on the part of the mother of the clinics which offer help in the matter of contraception and maternal and child welfare. No wonder, then, that Yudkin (1944) found that children from small

families were bigger and heavier and had a higher haemoglobin level than children from large families and that these differences were greater in the case of children from the poorer areas, or that Burns (1942) found in her classical survey of infant and child mortality in Durham that families with the lowest death rate were the one-child families, and that the late children of large families had a high death rate at all stages. Bearing on this question also are the observations and tentative conclusions of Fraser Roberts (1939), Burt (1946), Thomson (1947), and Himmelweit (1948), which suggest that there is a negative correlation between intelligence and size of family. These most important and disturbing conclusions have been subjected to criticism by Woolf (1947) and await final proof.

There is a gradual increase in maternal mortality with increasing age of mother, the rate becoming sharply accelerated after the age 30 to 34. Furthermore, when marriage or the first pregnancy is postponed to an age around 30 then, as Burns (1942) has shown, not only is the first birth relatively dangerous but all subsequent births are associated with a maternal death rate which is relatively high for that stage of the family. This in part explains the lack of improvement in maternal mortality in the better-off classes who, on the whole, marry late. It is exceedingly difficult to separate the effects of maternal age and parity. It can safely be said, however, that if all families consisted of three or four children born to mothers under 30 the maternal mortality rate would be halved. Primiparae have a higher death rate than 2-, 3- and 4-parae. Mortality is lowest in 2-parae and is under average in 1-, 3-, and 4-parae. In more than 4-parae it is in excess, this excess increasing steadily with each additional pregnancy (see Report of Scottish Departmental Committee, 1944).

Although the production of large families is associated with a high maternal mortality the decrease in the average size of family has not been attended by a corresponding fall in the maternal mortality rate, for the reason that a decrease in average family size means an increase in the proportion of first births, these being associated with increased hazards, and that first births now occur more frequently at older maternal ages than used to be the case so that greater risks must be encountered. Moreover, when the first birth is postponed all subsequent births must occur at ages which are associated with maternal risks relatively high for the birth rank of the child.

It is exceedingly difficult, if not impossible, to advocate early marriage and early childbearing on purely biological grounds. Now that life expectancy

has become so greatly extended it is but reasonable to spend more time in apprenticeship. This is the real argument in favour of the raising of the school leaving age. If the average woman at birth can expect to live to 60 she can afford to spend her first 25 years in equipping herself with the skills and the arts of living. Marriage is a social institution and those who engage in this contract can reasonably be expected to be socially as well as biologically mature.

There can be no doubt that if the present social gradient in respect of stillbirth, infant mortality, and maternal mortality could be obliterated by adequate social action these rates would greatly fall. At the present time the poorer among us tend to marry and reproduce earlier than do the relatively well-to-do. There is thus an association of early reproduction and poor environmental circumstances on the one hand and later reproduction and good environmental circumstances on the other. The present distinction between the socio-economic classes in respect of infant and maternal mortality suggests that in so far as these hazards are concerned environmental circumstance is more important than parity and maternal age (within limits). But even though the obliteration of this gradient would undoubtedly yield an equalization, and the stillbirth rate and the infant and maternal mortality rates generally would then be those which now are characteristic of Class I of the Registrar-General, it is quite certain that the effects of parity and of maternal age would still be easily discernible.

Two notions emerge from a consideration of these observations. The first is that since the first pregnancy is so much more hazardous than the second (the least hazardous of all) for both mother and child it would seem that in the human subject, as in the laboratory animal (see Asdell, Bogart, and Sperling, 1941), pregnancy has a maturing effect

which yields a greater efficiency in reproduction. If this is so, then investigation should be planned to discover a method whereby the O-para could be prepared for pregnancy (endocrinological or nutritional priming, application of physical medicine ?) so that the first pregnancy could, as it were, become the second. If this were possible much life could be saved. Another matter that deserves investigation is that of reducing to a minimum the drain of lactation upon the mother and at the same time safeguarding the interests of her infant. Maternal age is, of course, a complicating factor. The potential reproductive efficiency of the human female is at its peak in the woman under 25, appearing to begin to decline after the age of 20 or so. The observation of Matthews Duncan (1866) that the initial fecundity of women gradually waxes to a climax, probably about the age of 25, and then gradually wanes still commands the support of the great majority of obstetricians and is in line with the evidence derived from animal experimentation. It follows, therefore, that what is required is the maintenance of youthfulness of the individual as a whole and the maturation of her reproductive system.

The second notion, due allowance having been made for the effects of maternal age and unpropitious environmental circumstance, is that the human female is not built for the efficient production of more than four children in all, or, as Kerr and others remark (1944): "These facts might very naturally be advanced as an argument in favour of limitation of conception after the fourth or fifth pregnancy." In connexion with this second notion it is of peculiar interest to find that there is a very widespread lay opinion that the optimum family size is three to four. The British Institute of Public Opinion recently issued the figures given in the table (*News Chronicle*, May 30, 1948), whilst Baird

TABLE
BRITISH INSTITUTE OF PUBLIC OPINION SURVEY OF PREFERENCES IN FAMILY SIZE
(From the *News Chronicle*, May 30, 1948)

Number of children thought best	Great Britain %	France %	Canada %	Australia %	Holland %
1	4	5	1	1	1
2	39	31	21	14	22
3	27	38	22	21	29
4	20	17	28	46	24
4+	4	5	26	16	22
No opinion	6	4	2	2	2
	100	100	100	100	100

(1946) found on enquiry that most of the poorer mothers with ten children, as also most of the well-to-do with one or two, admitted that they would have been happier with three or four. These expressions of opinion can surely be accepted as being representative of the peoples of present-day communities with a social structure and philosophy more or less similar to our own and with adherents to the different churches present in the population in the same kind of proportions. They are supported by the observations of Titmuss and Grundy (1946) to the effect that in Luton there had occurred during a period of three generations a large increase in the proportion of one- and two-child families and a dramatic fall in the proportion of families of five or more.

THE BEARING OF THESE OBSERVATIONS AND SUGGESTIONS UPON POPULATION POLICY

The industrial revolution can rightly be blamed for much human misery and for much misdirection in our development as a society. Nevertheless, it is to this industrialization and urbanization and to the reactions of human beings to the circumstances so created that indirectly a significant reduction in family size must be ascribed. Economy in children made it possible for families to survive and to share in the abundant material wealth that was then created and thus in the rising standards of living. It is to be noted that this reduction in family size was not followed by a reduction in population size. In fact the population increased at an unprecedented rate, for the reason that though fewer children were being produced far fewer died than was formerly the case. In this decline in mortality the reduction in the average size of the family was by no means the least important factor, for between the size of the family and the chances of survival on the part of the members thereof there was and still is a direct relationship. It is established that large average family size is not a prerequisite for population maintenance or increase.

In a recent P.E.P. publication (1948) it is stated that "the objective to be achieved (by a population policy) is stable fertility at a level involving an average number of about 2.5 children in each family, and this average will mean a considerable number of families of four or five children." This statement, which will command the support of almost everybody and every organization that is concerned with the maintenance of our population size, is in remarkable accord with the facts and opinions presented above. If it is true that there is a strong biological urge toward parenthood in

most individuals, then this urge in the great majority of instances is satisfied with a family of two, three, or four. The individuals in the opinion survey cited were relating family size to their own circumstances and were not interested in demographic considerations. Yet their opinions concerning the optimum size of their own families coincide exactly with the conclusions reached by scientists in their calculations concerning population growth and with the observed facts relating to the connexion between mortality and family size. Manifestly if the average family size is to be 2.5 and the maximum 4, then in the absence of families of 4+ there will have to be far more families of 3 and 4. How many more it is difficult to determine using such figures concerning family size as are available, but if the Scottish figures are taken and if the present distribution of family size in the population is accepted as the basis for argument and if illegitimacy is disregarded, then it would appear that certainly not less than about 50 per cent. of the parents with no, one, two, and three children would be required to produce an additional child. The ideal policy would seem to be the encouragement of the majority of parents to produce a family of three whilst the mother is still young, and in the case of the families of proven biological worthiness, as estimated by the low incidence of stillbirth, infant and maternal mortality and morbidity, to encourage the production of additional offspring. Should this ever become the objective, then the impaired fertility of the childless and small family couples desiring children or more children would become a problem of even greater importance. (In the rat (see Asdell and others) the productive ability of the female throughout life is indicated by her initial performance. This is probably true also in the case of the human female, but the yardsticks by which performance is measured must be different.)

SUMMARY

1. The phenomena of childlessness and small family size are common and are important limiting factors. The part played by psychological factors in the causation of low fecundity is considered.
2. Lethal and sub-lethal genes play their part, a by no means unimportant one.
3. It is argued that the human female is characteristically monotocous and shown that polytocia greatly augments the hazards of childbearing and that monotocia suffices for the maintenance or increase of population size.
4. It is shown that the rate of reproduction, parity, and maternal age all operate to limit family

size—a rapid rate, increasing parity, and increasing age all being attended by increasing infant and maternal mortality.

5. Since the first pregnancy is peculiarly hazardous and the second the least so, it is suggested that investigations should be undertaken to discover the means whereby the first could be ridded of its dangers; could be, as it were, transformed into the second.

6. Evidence is displayed which points to the conclusion that the maximum family size should be four for the majority of married couples.

7. The evidence derived from opinion surveys shows that the "best" size of family is three to four.

8. Demographers are agreed that for the maintenance of our population size the average size of a family should be 2.5, which means that many must produce three, four, and five.

9. All things considered it would seem that the majority of married couples should be encouraged to produce three children whilst the mother is still young, and that those who together with their early offspring display an extraordinary healthiness should be encouraged to produce additional children. If families of more than four are not to be encouraged then it would appear that not less than 50 per cent. of parents with one, two, or three children would have to produce an additional child if the total population size is to be maintained.

REFERENCES

- Asdell, S. A., Bogart, R., and Sperling, G. (1941). *Cornell Univ. Agric. Exper.*, Memoir 238.
- Baird, D. (1945). *J. Obstet. Gynaec. Brit. Emp.*, **52**, 217 and 339.
- (1946). *Lancet*, **2**, 41.
- Barton, M., and others (1943). *Brit. med. J.*, **2**, 493.
- Barton, M., and Wiesner, B. P. (1946). *Ibid.*, **2**, 606.
- (1948). *Ibid.*, **2**, 847.
- Brown, R. Christie (1948). *Ibid.*, **2**, 851.
- Burns, C. M. (1942). "Infant and Maternal Mortality in relation to Size of Family and Rate of Breeding." Univ. of Durham.
- Burt, C. (1946). "Intelligence and Fertility." Hamish Hamilton: London.
- Cappell, D. F. (1946). *Brit. med. J.*, **2**, 641.
- Crosse, V. M. (1945). "The Premature Baby." Churchill: London.
- DeLee, J. B., and Greenhill, J. P. (1947). "Principles and Practice of Obstetrics." Ninth edit. Saunders: Philadelphia.
- Dickinson, R. L., and Morris, M. W. (1941). "Techniques of Conception Control." Williams Wilkins Co.: Baltimore.
- Duncan, J. Matthews (1884). "Sterility in Women." London.
- (1866). "Fecundity, Fertility, Sterility, and Allied Topics." Edinburgh.
- Editorial (1946). *Brit. med. J.*, **2**, 618.
- (1946). *Lancet*, **2**, 53.
- Engle, E. T. (1946). "Proceedings of Conference on Diagnosis in Sterility." C. C. Thomas: Springfield, Ohio.
- Fisher, R. A. (1930). "The Genetical Theory of Natural Selection." Oxford.
- Gates, R. R. (1946). "Human Genetics." Macmillan: New York.
- Himmelweit, H. T. (1948). *Eugen. Rev.*, **40**, 77.
- Hirst, J. C. (1940). *Penn. med. J.*, **43**, 1553.
- Jeffcoate, T. N. A. (1946). *Brit. med. J.*, **2**, 185.
- Joint Committee of the R.C.O.G. and Population Investigation Committee (1948). "Maternity in Great Britain." Oxford University Press: London.
- Kerr, J. M., and others (1944). "Combined Textbook of Obstetrics and Gynaecology." 4th edit. Livingstone: Edinburgh.
- Lane Roberts, C., and others (1939). "Sterility and Impaired Fertility." Hamish Hamilton: London.
- Marshall, F. H. A. (1922). "The Physiology of Reproduction." Second edit. Longmans Green: London.
- Mengert, W. F. (1948). *Amer. J. Obstet. Gynec.*, **55**, 660.
- Munnell, E. W., and Taylor, H. C. (1946). *Ibid.*, **52**, 588.
- Penrose, L. S. (1936). *Ann. Eugen.*, **7**, 1.
- P.E.P. (1948). "Population Policy in Great Britain." P.E.P.: London.
- Race, R. R., and others (1948). *Blood*, **3**, 689.
- Report of Scottish Departmental Committee on Puerperal Morbidity and Mortality (1944). H.M.S.O.
- Roberts, J. A. Fraser (1939). *Eugen. Rev.*, **30**, 237.
- Rothschild, Lord (1947). *Brit. med. J.*, **2**, 239.
- Siegler, S. L. (1945). "Fertility in Women." Heine-mann: London.
- Smith, C. A. (1947). *J. Paediatr.*, **30**, 229.
- Spencer, H. (1889). "Principles of Biology." Revised edition. Vol. 2. New York.
- Stallworthy, J. (1948). *J. Obstet. Gynaec. Brit. Emp.*, **55**, 171.
- Sutherland, I. (1946). *Lancet*, **2**, 953.
- Sydenham, Annie (1946). *Brit. med. J.*, **2**, 159.
- Thomson, G. (1947). "The Trend of National Intelligence." Hamish Hamilton: London.
- Titmuss, R. M., and Grundy, F. (1946). *Lancet*, **2**, 687.
- Tompkins, P. (1946). (See Engle.)
- Walser, H. C. (1948). *Amer. J. Obstet. Gynec.*, **55**, 799.
- Waterhouse, J. A., and Hogben, L. T. (1947). *Brit. J. soc. Med.*, **1**, 1.
- Whelpton, P. K., and Kiser, C. V. (1948). *Millbank Memorial Fund Quarterly*, **26**, 182.
- Woolf, B. (1947). *Brit. med. Bull.*, **5**, 226.
- Wood Jones, F. (1915). *Lancet*, **1**, 1113.
- (1945). *J. Obstet. Gynaec. Brit. Emp.*, **52**, 55.
- Yudkin, J. (1944). *Lancet*, **2**, 384.