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Departmental Report on Sterilisation

Address given by MR. L. G. BROCK, C.B.,
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at a Conference of Mental Health Workers held at Bristol on April 14th, 1934

I have not come down here with any idea of propaganda; this is not the occasion for it, nor is it my task. We were appointed as a Departmental Committee to find the facts and to ascertain how far on scientific grounds there is a case for sterilisation. To the best of our ability we have done so. It rests with the Government to decide what action to take on our recommendations. I have only come here this afternoon because Miss Darwin thought it might be of interest to you if I attempted some explanation of the main points in the report. I thought at one time I might have tried to deal in my address with the criticisms of the report. But although the press has been extraordinarily kind to us, with negligible exceptions, there has been singularly little constructive criticism to which I could attempt any reply this afternoon. Of course, there has been a certain amount of abuse; that must be expected. A critic in one paper, whose name I cannot remember, said that the appendices to the report would have been quite as useful and quite as convincing if the figures

had been thrown together by " orderly monkeys." Although I have a great regard for monkeys, which seem to me extraordinarily intelligent animals, few people I imagine would put them on the same level with Professor Fisher as statisticians. I need not waste time on mere essays in vituperation like this, and I had better come at once to the two main points, closely connected points, on which the whole report turns.

The first question is, what conclusions did the Committee reach as to the inheritance of mental abnormalities, and the second is, how far do their findings of fact establish a case for sterilisation on scientific grounds? If I am to explain the considerations which led the Committee to their conclusions, I shall have to attempt, what I feel is rather a rash task, to give you some kind of explanation of what is meant by the Mendelian theory of inheritance. I will try to put it simply, not because you might fail to understand it, but because if I do not put it simply, I may get lost myself. The Mendelian theory is the most commonly accepted theory of inheritance; in fact it is the only attempt to explain the machinery of inheritance which has behind it a large body of experimental work. Quite briefly, what the Mendelian theory of inheritance amounts to in outline is this. There is in the nucleus of every germ cell a series of bodies, thin thread-like filaments, which can be seen under a powerful microscope at certain stages of cell development. These are called " chromosomes," which means, literally, " coloured bodies." They are called chromosomes not because they have in reality any colour, but because they will take a dye which is necessary to make them visible under a microscope. Chromosomes, although they look like threads, appear to be made up of a large number of smaller units, which are called genes. No one has ever seen a gene: genes are much too small to be visible under any microscope which has yet been invented. Nevertheless, there has been a long series of experiments which demonstrate that the genes are there. Every transmissible character is transmitted through one or more of these genes. A character may depend upon a single gene, or upon a combination of genes, but one way or another every inherited character is determined by these genes. The remarkable thing is that the gene for a given character always comes in the same place in the same chromosome. Each gene keeps its proper place in the series; we may, therefore, think of chromosomes not as single threads, but as strings of infinitesimally small beads arranged in a definite order. Now the fertilised germ cell contains two strings of chromosomes, one derived from the father and one from the mother. The cells increase by division, but each cell always has two sets of chromosomes, one derived from each parent. If the gene which determines a particular characteristic is the same in both chromosomes, that is to say, if you get the same kind of gene from your mother as you do from your father, then the given characteristic which those genes determine is bound to show itself. But supposing you get two genes that don't match, what happens then? Either the result is intermediate, as happens with some physical characteristics, or, as far more generally happens, one gene prevails over the other.

The gene which prevails is called " dominant," and the one that recedes, or remains latent, is called " recessive." But the important point is that though only the dominant gene shows itself by producing the given character in that particular generation, the recessive gene may be passed on to the next generation.

The simplest illustration of this that I can give is eye colour. Eye colour is generally supposed to be the result of a single gene, or what, in the jargon of biology, is called a unit character. Now if you get the gene for brown eyes from both parents, your eyes will be brown; if you get the gene for blue eyes from both parents, your eyes will be blue. But if you get a gene for brown eyes from one parent and for blue eyes from the other, your eyes will be brown, because brown is dominant and blue is recessive. Whatever you may think about blue eyes from the aesthetic point of view, biologically, they are a kind of defect, and defects are generally recessive, though recessive characters are not by any means all defects. As I have said, if you get one gene for blue eyes and one gene for brown eyes, you eyes will be brown, because brown is dominant over blue, but you can pass on a gene for blue eyes to your children. That is a most important point. You cannot tell by any physical examination whether somebody with brown eyes is what is called homozygous, that is, having two genes for eye colour which match, or whether he is heterozygous, having two genes which do not match. I am sorry to have to use these terms, but it is impossible in discussing a scientific question to escape from technical jargon, and though we tried to explain the technical terms in our glossary, I am not sure that the explanation always made them any clearer. But, coming back to our illustration, the essential point is that because the individual has brown eyes, it does not follow that he is, so to speak, pure bred in this respect. In other words, it does not follow that both parents were brown-eyed, or that all his children will be brown-eyed. Two brown-eyed people who are heterozygous, or carriers of a gene for blue eyes, may have blue-eyed children. The point is important because people are so apt to think about inheritance in the terms of the old saying " Like father like son," but that does not describe inheritance as it is now understood, and it does not follow that because the son does not resemble his parents in some particular characteristic, this characteristic has not been inherited. Characteristics in which children differ from their parents may have remained latent and been transmitted through the parents from an earlier generation.

It is one of the great difficulties in investigating inheritance that there is generally nothing to distinguish the heterozygote, or carrier, from the homozygote, or pure bred type. In the case of some physical characteristics, the heterozygote may exhibit some kind of intermediate type, but in mental characteristics, so far as we know, there is nothing to distinguish the heterozygous person from the homozygous. It follows that dissimilarity in type between parent and child is in no way a disproof of inheritance. The qualities which the child shows may have been recessive and therefore latent in the

parents. Most defects are recessive; if this was not so, defects would be far commoner than they are. Normality is generally dominant, abnormality recessive. In the case of the rarer recessives, a defect may be passed on from generation to generation without coming to light until two persons each carrying the recessive gene happen to mate. I do not know whether any of you keep sheep, though I hardly expect that many do. I don't keep sheep myself; what with my Commissioners and my family I have got my hands full already. But you know the old saying that " there is a black sheep in every flock." That is broadly true, and what it means is that blackness in sheep is a rare recessive quality. Nobody wants black sheep; nobody breeds from black sheep; nevertheless, in spite of all efforts to reject the black and to breed it out, from time to time black sheep continue to appear. In other words, in every flock there is a certain number of sheep carrying the gene for this defect, and, sooner or later, they will mate with a ram which is also a carrier, and when this happens, a certain number of the offspring will be black.

There could be no better illustration of the way in which the Mendelian theory works than the piece of research mentioned in our report which was undertaken in Sweden by Dr. Sjögren. He was extraordinarily fortunate, because he found just the ideal conditions, which all investigators want and so few find. He worked in a small village community in a remote part of Sweden, shut in by high mountains. The village had little communication with the outside world, and, consequently, almost no opportunity of marriage outside its own population. The people of the village inter-married because they had no one else to marry. Now some generations ago, I think more than a hundred years back, three defectives appeared in that village community. I ought to have explained that the Swedish parish registers are much fuller than ours, and they report the physical characteristics of the inhabitants, and, fortunately, this particular type of mental defect was marked by unmistakable clinical signs. How the defect first appeared in the village I cannot say, but the important point is that for several generations afterwards it seemed to have disappeared. Then, in comparatively recent times, and this was what attracted Dr. Sjögren to undertake this investigation, a number of fresh cases showed themselves, but the striking feature was that in none of these cases did any of the parents or grandparents exhibit the defect. So you see, what happened was that these three defectives appeared in the village, they married normal unaffected persons, and their children became carriers of the defect. They did not show it themselves, nor did the next generation of carriers, and it was only in the third generation that the carriers became sufficiently numerous to marry each other. Up to that point they had been too nearly related to intermarry. Directly the heterozygotes, or carriers, began to intermarry, the defect appeared again, and an examination of pedigrees showed that every single case could be traced back to one or other of the three original defectives of three or four generations earlier. The isolation of the village and the well marked characteristics of that particular type of defect created ideal conditions for research,

and so afforded a classic demonstration of a mental defect inherited as a simple Mendelian recessive.

That helps to explain what has puzzled a great many people when they come to look for the causes of mental defect, that so surprisingly few defectives can be shown to have had defective fathers or mothers. But, you may say, are we to assume that all defect is transmitted according to simple Mendelian rules? Is mental defect a simple Mendelian recessive? That is not an easy question to answer. There are certain types of low grade defect which have been definitely proved to be simple recessives. Both types of amaurotic idiocy are simple recessives, and so is the type of low grade defect which Sjögren found. But most types of mental defect are certainly not simple recessives. Some of them may be, but I am inclined to think many are, in fact, multifactorial recessives, that is, characters which require a combination of genes in order to produce them.

Another difficulty in any enquiry into the causation of mental defect is that the more you come to study it, the more I think you will be convinced that it is not a single character, and that the difference between one defective and another is not merely a difference in degree but in kind. Of course, it has always been recognised that certain types of defect are clearly distinguishable from other types. The simplest and most obvious illustration of this is mongolism, a type of low grade defect which is associated with clearly marked and unmistakable physical peculiarities. You cannot mistake the mongol, he is the most clearly recognisable of all defectives. But while the existence of separate clinical types has long been recognised, there has been a tendency to treat feeble-mindedness as a single type. The conclusion to which the Committee came is that there are probably many more distinct types of defect than have yet been identified. In the case of the high grades, mental defect is not necessarily associated with any physical peculiarities, but we believe that even in the higher grades of defectives different types of defect exist, though no one has yet devised any criterion by which to distinguish them. If this belief in a variety of types is well founded, it helps to explain what would otherwise on Mendelian lines be difficult to explain, how two defectives can mate and yet produce normal children. If it is assumed that defect is a Mendelian recessive, then we must regard the defective parents as homozygous. If two people who are homozygous for the same character mate, their children must also be homozygous in respect of that character. The fact that you can find two defectives mating and producing apparently normal children does, I think, go to reinforce the view that there are far more different types of defect than it has yet been found possible to identify.

Of course, the crux of the problem is always going to be feeble-mindedness. When you get to the higher grades, mental defect is not necessarily associated with the physical abnormalities which are almost always exhibited in one form or another by the lower grades, and there are people who take the view that a good deal of higher grade defect is really only a section of what

they call the general curve of intelligence. In other words, the people who hold this view believe that defect is only an extreme form of dulness, and that the point at which you draw the line between the child who is dull and the child who is defective is really a shifting point which depends upon the standard of intelligence in the community. There is a good deal to be said for that view, but what I personally feel about it is that if you believe that high-grade defect is only an extreme end of the curve of the distribution of intelligence, it is extraordinarily difficult to explain why defectives exhibit not only educational failure, but emotional failure and peculiarities of behaviour. For myself, though I am very reluctant to formulate any theory at all, I think there is much to be said for the view that, while dulness is unquestionably very closely associated with defect, the merely dull child is different from the defective, but that dulness is favourable soil in which any inherited tendency to defect is likely to become much more marked than in persons endowed with a more active intelligence. That intelligence is transmitted seems to me beyond doubt. I know some people sometimes question this, and point triumphantly to instances of distinguished and able men who have had undistinguished and stupid sons. But, after all, we have all of us had two parents, and able men do not always marry able women, just as clever women sometimes marry very stupid men. It may be that the dull son of the distinguished father inherited his dulness from the mother's side, and equally a brilliant son of a dull father may have inherited his brilliance from his mother's side. But the question of the inheritance of intelligence has really been settled, and, in my view, put beyond all reasonable doubt, not by arguments from isolated individual cases, but by mass tests. If one were disposed to argue from individual cases, you could not have a better instance of transmitted intelligence than the distinguished family to which your Chairman belongs; but I would much rather rest the case for the transmission of intelligence, not on exceptional families like the Darwins or the Balfours, but on the mass tests which have shown that the children of the more intelligent parents do definitely beat the children of the less capable classes even where, as in the United States, all are educated in the same schools. Of course, the superior children enjoy a more favourable environment, but, making allowance for this, I think no one would now seriously question that intelligence is transmitted, though there is the widest difference of opinion as to the manner of its transmission.

Research into the causation of defect may go either up stream or down. Hitherto all enquiries, both in this country and on the Continent, have gone up stream. The investigators have studied the families from which the defectives came. We were peculiarly fortunate in having the co-operation of the local authorities, and we were able to do what no investigators had done before, to study the characteristics of the descendants. With the help of the local authorities, and I am glad to take this opportunity of thanking you for the part which many of you played in this enquiry, we were able to obtain particulars of some thousands of children of known defectives. That enquiry

produced very startling results. The children were divided into age groups, and we found that with the higher ages and the consequent greater certainty of diagnosis, the proportion of defective children showed a marked increase. In the group of age 13 and over, out of more than 1,800 children 32% were definitely defective, and 13% more were seriously retarded, and by retarded I mean at least two years behind the average. In other words, if we take the children of known defectives who are old enough for a definite diagnosis of their mental condition to be made, we find that 45% exhibit marked mental abnormality. That is a most astonishing result and one which has not yet received the attention it deserves.

The enquiry would have been still more valuable if we had been able to get more information as to the mental condition of the other parents. Probably in the majority of cases the other parent, if not actually defective, was not far off it. As some of you know better than I do, defectives tend to sink to the bottom, their economic and social inefficiency drives them down, and they form a kind of social sediment. More than this, like marries like, and it is therefore not improbable that in many instances the defectives were mated to carriers of defect. But, even allowing for this factor, the proportion of defective children is so large that it suggests that possibly some forms of defect may be really not recessives but partial dominants. However, that is merely speculation.

There is one other feature of the inheritance of mental abnormality which still further complicates the problem. There are many families in which you find more than one kind of mental abnormality. You find defectives coming from families which in other generations exhibit mental disorder, or allied conditions such as epilepsy. This is what biologists call in their dreadful jargon, " Polymorphism "; and some authorities, including Dr. Tredgold, have put forward the theory that this occurrence of different types of mental abnormality in the same family is inherited, but is not transmitted on Mendelian lines. Dr. Tredgold, and those who share his view, believe that there is some impairment of the germ plasm which may show itself in a variety of forms. It is admitted that the germ plasm may be affected by toxins, for example, so that the offspring may be seriously affected. The doubt is whether this germinal impairment can be transmitted to succeeding generations. The theory seems to me to involve serious difficulties, because it cuts across the Mendelian hypothesis which has such a mass of experimental work to support it. It is possible, however, that the clue to the problem is to be found in the influence of environment.

You are all of you familiar with the distinction between heredity and environment, nature versus nurture. It is a beautifully clear cut logical distinction; the trouble is that it leads us to assume that any particular character must be due to one cause or the other. But heredity and environment are not mutually exclusive causes. Nobody lives, as it were, in a vacuum; everyone is influenced in a greater or less degree by his environment. While experi-

ment has shown that some genes will produce a given character in any environment in which the individual can live, others will only produce it in a favourable environment. It is extraordinarily difficult, and indeed, often impossible, to disentangle the effects of heredity and environment. Incidentally, I would remind you that a high familial incidence of disease is not necessarily proof of inheritance, because, for one thing, members of the same family generally, though not always, live in the same environment, and if you find members of the same family exhibiting the same peculiarity, this is not conclusive proof that the peculiarity is transmitted. Let me take a rather absurd illustration, because absurd illustrations are easier to remember. Suppose a particular kind of drinking water had the effect of turning hair green, and there was a village partly supplied by a stream of ordinary water, while the other inhabitants got their supplies from a deep well which had this peculiar contamination. You would find, in our imaginary village, members of families in which the children and the parents both had green hair, but the greenness would not be an inherited quality, it would be simply due to the environment. Take another and more complicated case, the effect on children of having neurotic parents. It is possible that the parents may transmit to their children their own nervous irritability, but it is also highly probable that the way in which they treat their children will produce in them the same kind of nervous symptoms which the parents themselves exhibit. Who is to say how far the neurotic tendencies of the children are due to inheritance, and how far to the abnormal environment in which they have been brought up. The point is of special importance in relation to what has been called the " social problem group." As I have already said, the defectives tend to form the sediment of society, and Dr. Lewis has well called the stratum at the bottom the " social problem group." There you have a group who combine the worst possible inheritance with the worst possible environment, an environment from which they have no chance of escape, because it is of their own creation.

What are the conclusions which the Committee base on these findings of fact? The importance of the hereditary factor is beyond question, but, in the present state of knowledge, we cannot predict with certainty the results of any individual union. We might predict with a reasonable degree of probability the average results of a large number of unions, but it cannot be said with certainty that the children of any two people must inevitably be defective. If this conclusion is accepted, it seemed to the Committee to be fatal to compulsion. We did not believe that the legislature would feel justified in compelling any person to submit to sterilisation unless it could be said with certainty that he or she was incapable of producing normal children. On scientific grounds the case for compulsion is not established, but, even if it were, there are grave practical objections to compulsion. In practice, it does not work, as the experience of the United States has shown. Judged by the number of operations, sterilisation has been most successful in those States where it is worked on a voluntary basis. But there is the further objection that compulsion will in-

crease the difficulties of ascertainment by "driving defect underground." Already there is evidence that one result of compulsory sterilisation in Germany is that many doctors are refusing to give certificates in terms which would bring the patient within the scope of the sterilisation law.

There are, of course, objections to a voluntary scheme; in this world there are objections to most proposals. The objections to voluntary sterilisation fall under four main heads. In the first place, it is said that a voluntary scheme will fail because no one will volunteer. In the second place, it is argued that consent is meaningless in relation to defectives. Thirdly, it is urged that a voluntary scheme is a sham and will end in veiled compulsion. Fourthly, it is said that defectives are so suggestible that they will volunteer wholesale and repent afterwards. All these objections contain a certain measure of truth, though to some extent they cancel out. As I have already said, United States' experience indicates that a voluntary system is far more effective than a compulsory one. As to the validity of consent, it is not true that consent in the case of the higher grades is meaningless. High grade defectives may not understand all the implications of sterilisation, but those who come in contact with them agree that they are quite capable of understanding what it means and what its results are. Indeed, one witness who argued most strongly against the validity of consent, admitted that his patients freely discussed the whole question. Even now requests for sterilisation from defectives are by no means infrequent. I agree that veiled compulsion would be deplorable, but we have suggested every possible safeguard to prevent this. As for the suggestibility of defectives, they are suggestible, but so are many other people. Provided that those who object are free to refuse, does it greatly matter if patients accept the advice of those to whom they naturally look for guidance.

There is nothing sensational or drastic in the report, but it is at least an attempt to apply the principles of biology to a practical problem of everyday life. But there is one caveat which I am bound to add. Sterilisation can never be a substitute for the provision of institutional beds. It may, I hope that it will, tend to check the increase in the incidence of defect, but it will not enable local authorities to do without colonies and certified institutions. We have said this in the strongest terms, and the witnesses with practical knowledge of mental defectives and their needs were emphatic in confirming this view. In our view, sterilisation is neither a penalty nor a panacea, and we regret that in some foreign laws, such as that of Denmark, it has been given a penal character. The Committee regarded sterilisation as an act of social justice, a right which society ought not to refuse, least of all to the carrier of defect. Let us have every possible safeguard against abuse, but do not let fear of abuse condemn people to a choice between unnatural abstinence and the risk of bringing into the world children who can never have a chance, who will be a misery to themselves and a burden to society.