CONSANGUINITY AND MENTAL DISORDER.*

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ONE difficulty which hinders the advance of psychiatry is that the facts of a mental disorder are not easy to describe and to record accurately. Many of the descriptive words used, such as " depression ", " confusion ", are taken from common speech and carry with them vague meanings. Out of sympathy with the sufferer, we tend to read our own emotions into the clinical picture. Moreover, because a disorder of thought and behaviour is a disorder of adaptation to living, the description of a mental disease involves not only the recording of the symptoms, but also a description of the circumstances and life-setting in which they occur. Another difficulty lies in the great variation in the clinical pictures of mental disorder. These vary so much that a diagnostic label such as affective psychosis or schizophrenia does not convey, and indeed is not intended to convey, an idea of a specific disease. These difficulties are somewhat more marked in the investigation of mental disorder in contrast to the investigation of mental defect. The statement that a patient is a Mongol or a cerebral diplegic conveys the idea of a fairly definite clinical syndrome, whereas a statement that a person suffers from schizophrenia calls up a much less clear mental picture. It seems that the only way out of this difficulty is to give a short statement of the clinical facts about each psychotic whom one discusses.

Genetical research in psychiatry is thus made harder by having to deal not with sharply defined clinical entities whose inheritance can be separately studied, but with disorders which, for the present at any rate, are perhaps best classified into broad reaction types. Moreover, the majority of patients in any large mental hospital fall into one reaction type—that which includes the schizophrenias. On the other hand, from the point of view of research into the inheritance of mental disorder, it is peculiarly fortunate that for the past hundred years many persons suffering from psychoses have been committed to mental hospitals, where statutory records have been kept of their mental state. In this respect, psychiatry has the advantage over all other branches of medicine, for in no other branch of medicine are comparable records available about the disordered relatives of patients.

In recent years evidence has accumulated that dominant Mendelian factors

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are important in the production of the manic-depressive psychosis, and that recessive Mendelian factors may operate in the production of schizophrenia. In the case of rare diseases, an inquiry into parental consanguinity helps in the detection of Mendelian recessive determination. The carrier of a rare Mendelian factor will be more likely to meet another carrier in his own family than among random acquaintances, and thus persons who receive the same recessive genetical factor from both parents, and who therefore suffer from a rare recessively determined disease, will often have parents who are blood relatives. The investigation of parental consanguinity becomes a useful tool for the detection of recessively determined disease. The commoner the disease, the less often will the parents be blood relatives, and the more closely will the incidence of parental consanguinity approach that which occurs in the general population. Thus, the commoner the disease the more essential is it to have accurate knowledge of the incidence of consanguineous marriage in the general population, and to examine large groups of patients in order to eliminate the effects of chance findings.

A relationship between parental consanguinity and disease in the offspring had been noticed long before the growth of genetics from Mendel's work provided the explanation. The growth of biological knowledge last century attracted many to investigate the problem. There was a tendency among these early workers, however, to record those cousin marriages which had been attended by unfortunate results in the offspring and to ignore others. In this way the vague but widespread lay belief in the undesirability of cousin marriage received unwarranted confirmation from the medical profession. In 1847, Prosper Lucas (9) insisted that "experience proves that consanguinity causes the degeneration of the species, the doubling and redoubling of all infirmities, all harmful predispositions, both of body and mind, the stupefying of all the mental faculties, brutishness, insanity". Much of the early literature has been reviewed by Huth (7) and by Desruelles and Gardien (4). Some early investigators attempted to make a complete survey of all the marriages in an isolated district where inbreeding was common, and to compare the incidence of disease among the offspring of the consanguineous and the non-consanguineous marriages in that district. Such a laborious task naturally proved beyond the abilities of any one investigator, except in districts with very small populations. Results therefore were often meagre. But the pre-Mendelian investigations of Voisin (18) in 1865 among a highly inbred population in the isolated commune of Batz, and those of Mitchell (11) in the Highlands and islands of Scotland and on St. Kilda, have been followed by the classical work of Lundborg (10) on myoclonus epilepsy and Sjögren (17) on amaurotic idiocy, which have proved the recessive nature of these two diseases ; and among others, by the work of Brugger (2) in Bavaria and by investigations in Swiss mountain villages. Brugger investigated five districts, and found that more than half of all the schizophrenics and most of the mental defectives came from one district in which there was much

inbreeding. The Swiss workers (5, 6, 12, 16,) found that, while inbreeding was high in all districts, in one there was hardly any hereditary disease, and in another schizophrenia and mental defect were common.

Mitchell's investigation in Scotland is the first careful work on this subject in this country. Besides other investigations, he made a survey of two small fishing villages in Easter Ross, with a population of 1,500 and a rather high incidence of idiocy. He found that nearly a quarter of all marriages were consanguineous, but that idiocy was rather more frequent among the offspring of unrelated than of related parents. Mitchell inquired into the parentage of all the mental defectives he visited throughout Scotland. He found that of 711 idiots and imbeciles, at least 13·6% had blood-related parents, a percentage which he thought was about ten times greater than that in the general population. He found also that in families where more than one idiot occurred among brothers and sisters, parental consanguinity rose to 44%. Estimations of the frequency of parental consanguinity have been made in series of cases of various other diseases. The following percentages of consanguineous marriages have been found.

TABLE I.

	Percentage parents consanguineous.
Myoclonus epilepsy (Lundborg 10) . . .	35
Amaurotic idiocy (Sjögren, 17)	26
Cerebral diplegia (Penrose, 15)	9

In 1875 George Darwin (3) published the results of a wide survey of hospitals for the insane and for the defective. By an ingenious method he estimated that the rate of first-cousin marriage in the general population lay between 1½% and 2¼%. He found the following results in mental hospitals (Table II). As the amount of consanguineous parentage in mental hospitals was little greater than that in the general population, Darwin rather naturally concluded that "as far as insanity and idiocy go, no evil has been shown to accrue from consanguineous marriages". It must be remembered that he was writing thirty years before Mendel's discovery became known.

Recently I have completed a similar inquiry into the incidence of consanguineous parentage among the mental hospital populations of seven English counties and of two cities, Cardiff and Edinburgh. The inquiry also included the examination of 152 patients with related parents, and the comparison of them and of their families with a similar group of patients with unrelated parents selected at random from the mental hospital populations. The investigation involved the recording of some 17,000 persons, and took about three years to do. The detailed results will soon be published.

Of 4,200 patients surveyed, 2·4% had parents who were blood relatives. As might be expected, the rate of parental consanguinity was rather higher in

TABLE II.—*Inquiries in Asylums by G. H. Darwin in* 1875.

Asylum.	Doctor.	Number of patients surveyed.	Number derived from first cousins.	Percentage first cousin parentage.
Wakefield*	Dr. Crichton Browne	655	31	4·7
Hatton, Warwick*	Dr. Woods	258	8 or 9	3·1 to 3·5
Crichton, Dumfries*	Dr. Gilchrist	251	12	4·8
Caterham†	Dr. Adam	560	20	3·5
Earlswood†	Dr. Grabham	1,388	53	3·8
Other asylums	..	1,560	45 to 50	2·9 to 3·2
All asylums	..	4,672	169 to 175	3·6 to 3·8

Inquiry by Dr. Shuttleworth, 1886.

	Number of patients surveyed.	Percentage consanguineous parentage.	Percentage first cousin parentage.
Royal Albert Asylum, Lancaster	About 900	5·1	2·9

* Chiefly psychotics, but many defectives.
† Mental defective patients only.

hospitals serving rural than in those serving urban populations—rural 3·8%, urban 1·9%. The rate of consanguineous marriage in the general population is not accurately known. The families of patients with unrelated parents are possibly in respect of consanguineous marriage not an abnormal sample of the general population. Among about 2,400 married relatives in such families, 0·8% had married blood relatives. In a recent survey by the Human Genetics Committee of the Medical Research Council (1), a similar figure of 0·8% consanguineous parentage was found among about 3,400 patients in urban general hospitals. The interpretation of the higher rate of parental consanguinity among mental hospital patients is complicated by the fact that the two surveys do not refer to the same areas, and the methods of ascertainment were not the same. We require knowledge about the local variations of cousin marriage in the general population. I have some figures which refer to Cardiff alone (Table III). In Cardiff parental consanguinity is higher among the mental hospital patients than among the general hospital patients.

Many social, economic and personal factors influence the frequency of cousin marriage. It is higher in rural than in urban areas; it is influenced by the recent increase in road travel and by the decreasing size of the human family. In my material, first-cousin marriages make up about two-thirds of all consanguineous marriages. Men more often married their maternal first cousins than their paternal first cousins, possibly because the prejudice against cousin marriage made them avoid people of the same surname. It seems that cousin

TABLE III.—*Parental Consanguinity Rate among Patients in Cardiff Mental and General Hospitals.*

	Number of patients.	Percentage parents consanguineous.	Percentage parents first cousins.
Cardiff City Mental Hospital:			
Present patients . . .	466	2·58	1·72
Former patients . . .	1,226	2·04	..
Cardiff Royal Infirmary . .	1,723	0·81	0·46

marriages tend to run in some families. This is noticeable in the Cambridge Fens, in Cornwall, and to a lesser extent in Devon. Cousin marriages are no less fertile than others. Mental disease or mental abnormality is no more frequent among people who marry their cousins than among people who marry outside their own family. It was, of course, difficult to discover the personal reasons which had influenced cousins in marrying each other; it seemed that love and propinquity over years were often the main reasons. In some instances there were other considerations; some men considered that women of families other than their own were not good enough to marry, or the cousins had had an illegitimate child, or the woman had for long been housekeeper to her cousin, or the marriage had been arranged by parents to keep money in the family, or there were no other suitable acquaintances.

Perhaps the most striking fact which appears on analysis of the families of patients is that there exists a definite and specific familial incidence of mental disease. The relatives of patients with affective psychosis, when they are psychotic, tend to suffer from affective psychoses, and the relatives of schizophrenics from schizophrenia. This specific familial incidence is true not only of the broad groups of reaction type, but also for each clinical type within the group, with the exception of the organic psychoses of exogenous origin. The more strict the criterion of manic-depressive disease in the patient, the more typically manic-depressive are the disordered relatives, and the more definitely dominant is the inheritance. Familial incidence holds for the schizophrenias, though it is most true of paranoid schizophrenia, and least true of catatonic schizophrenia. The incidence of mental disorder among relatives decreases in an orderly way with decreasing degree of relationship to the patient.

Among the patients with blood-related parents were several instances of strange and atypical psychoses. In some of these I found quite good evidence of Mendelian recessive determination (8, 13). Patients with schizophrenia occurred in the consanguineous group rather more frequently than might occur by average chance. The parents of schizophrenics were nearly always themselves unaffected, but there were other cases of schizophrenia among the brothers and sisters. Such facts point to the operation of Mendelian recessive factors in schizophrenia.

It is well known that the near relatives of schizophrenics often show marked personality abnormalities. In my material such abnormalities are particularly common in the families of schizophrenics where the parents are consanguineous. The abnormalities occur chiefly among the parents and brothers and sisters, and consist of paranoid and eccentric personalities, hypochondriasis and chronic alcoholism. The frequency of such abnormalities among the relatives of schizophrenics suggests a genetical relationship, and their special frequency among these near relatives when the parents are consanguineous suggests that the psychopathic parents and brothers and sisters carry a recessive genetical factor which is present in full recessive form in the schizophrenic patient. Such a factor in the psychopathic relatives would be, of course, incompletely recessive, and could be considered as a partial dominant. The important point is not so much the question of dominance or of recessivity, but whether the genetical factor is present in single or in double form. Penrose (14), in discussing the manifestation of dominant factors in double (homozygous) form, has indicated that the double form may be lethal, and he has also drawn attention to the presence of cases of affective psychosis among the relatives of microcephalic idiots. In my material there is evidence that incomplete dominant factors determine manic-depressive psychosis. I have some instances of children born to first-cousin parents, one or both of whom suffers from manic-depressive psychosis. I have been impressed by the frequency with which such parents produce miscarriages or children who are stillborn or die in infancy Several of the children who have died have been in some way grossly disordered. It is possible that they have received the dominant factor for manic-depressive disease from both parents, and in consequence manifest a gross disorder fatal in infancy or earlier.

Such findings as these indicate that by the application of genetics to psychiatry we may hope to understand some of the relations of mental disorder to mental defect. The combination of a clinical with a genetical approach to mental disease should also help in the isolation of clinical syndromes from the heterogeneous material with which we have to deal.

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Discussion.

Dr. J. A. FRAZER ROBERTS said that he entirely agreed with Dr. Penrose that the whole subject of heredity and mental deficiency was an extremely complicated one, but he thought it could be said that there had been advances during the last few years. Quite a number of these advances were owing to Dr. Penrose himself, and they had had that day from him a very comprehensive review of the whole subject. He felt quite sure that the advances which would take place during the next few years would be along the lines Dr. Penrose had been laying down during the past five years or so. They would also agree that the essential criteria of mental deficiency included a great variety of conditions. Some years ago Dr. Lewis drew the very important distinction, that one could divide mental deficiency roughly into two groups—the sub-cultural group which could be regarded as part of the normal curve of distribution of intelligence, and a pathological group which could not be so regarded. He thought the line which divided those groups could be fixed at the I.Q. level on the Binet scale of about 45; this was approximately the level which separated the feeble-minded from the idiot and the imbecile. Idiots and imbeciles were in Dr. Lewis's pathological group and the feeble-minded were in the sub-cultural group.

To take the sub-cultural group first, these formed part of the normal curve, or at least the great majority of them did. This meant that for every person falling into the group of feeble-minded, there was a corresponding person who exceeded the average for the whole general population by an equivalent amount. For every person with an I.Q. of 50 in the general population there was a corresponding person with an I.Q. of 150. Of course, the tests would have to be made sufficiently early, simply because the Binet children did not go high enough—not much above the age of 10. Still, if the age of 10 were taken, it would be found that there were as many with an I.Q. of 150 as with an I.Q. of 50. If the sub-cultural or feeble-minded group was part of the normal curve, they would expect to find that the feeble-minded person differed from his normal or merely backward brothers and sisters, not in respect of one factor, but in respect of several or many or perhaps very many. On the other hand, if the pathological group were taken, they were no longer dealing with the normal curve. Idiots and imbeciles were far more numerous than one would expect to find on the basis of the normal curve. Let it be supposed that the persons who exceeded the average of intelligence by the amount that the imbeciles fell short of it would be only $\frac{1}{15}$ or $\frac{1}{10}$ as common as the imbeciles, while the persons who exceeded the average by the amount the subculturals fell short of it probably corresponded to the persons in number. Recently a colleague of his was testing a number of 10-year children, and found a boy of the mental age of 17 years and the I.Q. was just over 170. In parenthesis he was not sure but that the child with an I.Q. of 170 was not as abnormal socially as any imbecile. This boy's interests were entirely at the adult level, his remarks were extremely wise and pedantic, and he was a great thorn in the side of his teacher and his companions.

With regard to the pathological group, here it might be expected that the factor of inheritance, in so far as inheritance was involved at all, was relatively simpler. The idiot or imbecile would frequently differ from his normal brothers and sisters by one factor, or one factor conditioned by environment—in any case a relatively simple situation as compared with what was found in the feeble-minded. That was no doubt the reason why people had supposed in the past that inheritance was more important in the case of the feeble-minded than in the case of the idiot or imbecile. There was the general resemblance of all brothers and sisters in a family to one, so that it was fairly obvious to the lay mind that there was this general family resemblance, whereas the picture of the segregated individual was not the same at all. But he agreed with Dr. Penrose that when more was known about it, it would probably be discovered that there was very little difference between heredity and environment in the two groups.

Dr. Penrose seemed to suggest that quite frequently people in the category of the

feeble-minded were related to idiots and imbeciles, and that their feeble-mindedness might be due to the same factor, possibly in a different " dosage ". If the curve was normal approximately down to an I.Q. of 45, one would not expect that very many of the feeble-minded group would actually show such relationship, but it was known that of the people sufficiently retarded intellectually to require certification or to be possibly certifiable, only a minority were in fact certified or held to require a certificate. Did Dr. Penrose think it possible in an institutional population, that is, including the feeble-minded people, that such feeble-minded people would be a fair sample of that degree of intellectual retardation in such a community or was there some other factor that should be taken into account ? In other words, if one took the feeble-minded people in institutions, would one find that they had more idiot or imbecile relations than a group of people of the same I.Q. who had not been certified and did not need certification ?

He added one word on Dr. Munro's paper. They had all been impressed by his examples, and it was clear that the work he had done had led to very big advances in their knowledge of another very obscure if related subject.

Dr. PENROSE, in reply to Dr. Roberts, said that he did not consider the institutional population a thoroughly representative one, and he thought there was a sociological difficulty in working from institutional cases of defect.

Dr. E. O. LEWIS desired to make a few general remarks as one in very close touch with the research work on this subject. He thought it was a matter for pride that we had in this country two centres at which research was organized and carried on in a systematic way, namely, the Royal Eastern Counties Institution at Colchester under Dr. Penrose, and the Stoke Park Colony, under the direction of Prof. Berry. At Colchester the fact that Dr. Munro also was carrying out researches into these problems would add to the value of the work Dr. Penrose himself was doing.

Before passing on to Dr. Penrose's paper, he desired to ask one question of Dr. Munro. Dr. Munro had stated that schizophrenia had a fairly large incidence in the group of consanguineous families. He would probably have some evidence on the point that Kraepelin had mentioned. Kraepelin had stated that schizophrenia was the forerunner of mental defect. He even called certain types of mental defect congenital dementia præcox. He would have thought that if there was any foundation for Kraepelin's view, the study of consanguineous families which had given rise to cases of schizophrenia would afford evidence for or against it.

He was sure that many of those present had had the privilege of reading Dr. Penrose's recent report published by the Medical Research Council, and would agree with him that Dr. Penrose had increased their confidence considerably in the genetical approach to the problem. What he liked especially about Dr. Penrose's work was the soundness and reliability of his clinical data. He wished the same could be said of the work of many statisticians in mental science. He could not help contrasting some of the genealogical tables shown by Dr. Penrose with those drawn up some years ago by Dr. Karl Pearson and by Goddard. The younger generation seemed to have very meagre data as compared with the old, though he was not sure that that made their task easier. But the meagreness of the data was a merit. It showed that they were far more critical of their data than were Karl Pearson or Goddard, and he thought that Dr. Penrose and others were extremely critical, and this would increase the confidence of them all in their conclusions.

After reading Dr. Penrose's report he had the feeling that none of them who were not experts should ever use the word " heredity ". That word had better be left to the geneticists in the future, and most of them could not claim an expert knowledge of genetics. Instead of " heredity " he would suggest some such term as " familial concentration ". Dr. Penrose in his criticism had implied that he (Dr. Lewis) had been guilty of using the term " heredity " rather rashly ; but he was sure that there were few members present who had not been guilty of the same offence. The term had certain mathematical and scientific implications that it would be well for the average person to avoid. He believed that Dr. Penrose would agree that the

"familial concentration" of feeble-minded was considerably greater than that of idiocy. That was a question which should be carefully investigated.

He felt that Dr. Penrose's work was giving a new orientation in these researches into mental deficiency. Until recently most of them had thought that it was amongst the feeble-minded that they should look for genetical factors, and that most of the lower-grade cases were pathological. The definite evidence they had now of inheritance working in its own infallible way with a small group of lower-grade cases, mostly idiots, pointed in another direction, and he thought that for the next decade or so, in view of the results of Dr. Penrose's researches, they had better confine themselves to looking for genetically typical groups amongst the lower-grade cases. He hoped the medical superintendents would take the trouble to make a special note of all such instances, because the information would certainly be required in the near future for following up the study of genetic groups.

Dr. B. M. LOTHIAN said that he had been taught that mongolism was entirely due to environment, and it was not until he began to interview parents of mongols that he suspected that this might not be true. He had seen in a number of parents one or two mongoloid signs—the fissured tongue or the slant eye or something of that sort. Had Dr. Penrose any observations of that kind ?

Dr. PENROSE said he thought it was true that there were classical mongol signs in near relatives of mongols. He had seen them in the single line on the little finger or the transverse line on the palm, but he had not known of a fissured tongue in the parent.

Dr. DOUGLAS TURNER said he hoped that next year the session for discussing the subject of mental deficiency would be arranged earlier in the week. He believed it would have been earlier on this occasion but for certain difficulties. He wished to offer his congratulations to Dr. Penrose and his staff on the big work they had done during the past six years in this research. No one who had not seen them at work and the enormous amount of detail with which every single case had been investigated could appreciate what it had meant. Genetic problems were difficult for the ordinary man to understand, because the whole question of inheritance was so complicated and so inextricably mixed up with environment. Hooton in his recent book on *Apes, Men and Morons*, said, "The future of mankind does not depend on political or economic theory nor yet on measures of social amelioration, but on the production of better minds in sounder bodies". Dr. Penrose's work had that aim in view. Better minds could not be forthcoming until it was known what were the causes of defective and abnormal minds. Dr. Penrose seemed to have disproved the extravagant claims of some eugenists in the past who had asserted that their remedy, had it been applied, would have reduced mental defect by 50% in three generations. The present speaker's own opinion, which had no scientific value, was that the higher-grade cases which became certifiable were a small proportion of those of below-average intelligence. The majority carried on in the world satisfactorily. Many feeble-minded persons with a mental age of eight, nine, ten or eleven years, if they had been brought up in a more favourable environment, would never have been certified. Others failed no doubt because of instability added to their below-average intelligence, but those two things were different, and it was the adding of one to the other which made care necessary. Others had a certain amount of instability which might be due entirely to a different hereditary factor. Whatever they might do in the way of selection in genetical treatment, they were not going to alter matters very much. Supposing it were possible by any genetical means to raise the average I.Q. from say, 80–120 to 120–160, there would still be many who would fail to carry on in the more difficult environment in which they would then be living. He could see no escape from the fact that there would always be many people with a below-average intelligence, whatever the average might be.

Dr. FITZGERALD added his congratulations, and said that the Brock report now needed to be re-written.