Name: James Baker Abram, Jr. Date of Degree: May 26, 1963

Institution: Oklahoma State University

Location: Stillwater, Oklahoma

Title of Study: THE ROLE OF HEREDITY IN INFLUENCING MENTAL DISORDERS AND DEFICIENCIES.

Pages in Study: 50 Candidate for Degree of Master of Science

Major Field: Natural Science

Scope and Method of Study: A survey of mental disorders and deficiencies that are influenced by genetic factors have been undertaken in this report. The materials used in this survey are: (1) textbooks on eugenics and heredity; and (2) periodicals and journals containing recent studies on the mentally defective. It was necessary to include books of psychiatry as well as genetic studies, as the problem is of psychological importance as well as biological. It is the thought that this study will provide a basis of study for the biologist, the psychiatrist, the student, and the interested reader.

Findings and Conclusions: It was emphatically illustrated in the literature that the problem of mental illness lies in the germ plasm of the general population, rather than the mental defectives themselves who make only a negligible contribution to the increase of the mentally deficient. Mental illness, a condition for which people have been tortured in previous years, should be looked upon as a bodily disorder. The ugly stigma that the illness has carried for many years is being gradually erased. Mental illness affects the whole population; by presenting a problem to the families and friends of the afflicted, it also places a financial burden on the nation. New treatments and approaches are being utilized. The "snake pit" era is gone forever. Geneticists and psychiatrists are cognizant of the fact that the problem does not originate from a single source, and are uniting their efforts to combat this problem of long standing.

ADVISER'S APPROVAL James H. Zant

THE ROLE OF HEREDITY IN INFLUENCING MENTAL DISORDERS AND DEFICIENCIES

by

JAMES BAKER ABRAM, JR.

Bachelor of Science

Langston University

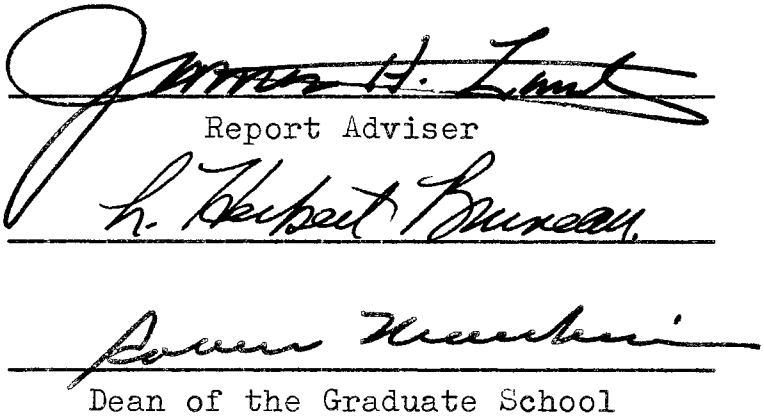
Langston, Oklahoma

1959

Submitted to the faculty of the Graduate School of
the Oklahoma State University
in partial fulfillment of the requirements
for the degree of
MASTER OF SCIENCE
May, 1963

THE ROLE OF HEREDITY IN INFLUENCING
MENTAL DISORDERS AND DEFICIENCIES

Report Approved:



Report Adviser

Dean of the Graduate School

PREFACE

The concept of human heredity is one that greatly influences many facets of behavior. The time-honored arguments of heredity versus environment have persisted. Biologists concentrate their studies on the hereditary aspects of the problem.

The area of human genetics is highly diversified and extensive. It would be utterly impossible to consider a broad perspective of this area. With this in mind, the role of genetics in influencing mental disorders and deficiencies will be considered, including the basic genetic principles involved.

With the alarming high rate of mental illness and diseases of the nervous system in the present population, this study is designed to present a survey on this rapidly developing national problem.

It is impossible to draw any sharp line between mental defects and defects of the nervous system. This distinction is drawn purely for convenience and the evidence justifies the assumption that all mental derangements have at least a partial basis in a defect of the nervous system.

Less emphasis is placed on the mechanics of genetics, but an attempt is made to point out the role that

genetics plays in influencing mental disorders and mental deficiencies.

It is written so that it will provide information for the professional biologist and psychiatrist, and remain comprehensible for high school and college students as well as the interested reader.

Indebtedness is acknowledged to Drs. James H. Zant and L. Herbert Bruneau for critical reading of the study and suggestions, and to my wife, Darlene, for her assistance in typing the manuscript.

TABLE OF CONTENTS

LIST OF TABLES

CHAPTER I

INTRODUCTION

It is thought and believed by many that what a person is born with is somehow related to what he does in later life. There are millions who subscribe to the popular belief that if you are born a member of one "race or ethnic group", you are unlikely to achieve as much or as well as the members of another race or group. Moreover, millions believe that differences in intelligence and mentality is due to heredity, and therefore very little can be done about improving an individual's intelligence, which is set and determined by heredity.

Certain novels and plays further support certain of these concepts. William March's novel, "The Bad Seed" demonstrated how the "bad" heredity of the mother, whose mother in turn was a murderer, shows up in her daughter who inherited her grandmother's disposition to engage in mass murder. Plays like Clemence Dane's "A Bill of Divorcement", seen by millions on the screen, is more directly related to our topic. It's tragic theme being that because a father was insane his daughter could not marry for fear she would pass on his insanity to her children or manifest it herself. Through this and other media, we have quite a sizable body of epidemic confusion about what

heredity is and how it works. It has been suggested that the word "heredity" be dropped altogether because of the long standing abuse of its meaning. On the other hand, when heredity is understood in the light of scientifically discovered facts, it is seen to be a science which yields findings that enable us to improve the welfare and general happiness of mankind. The truths revealed by such systematized knowledge make it possible for us as human beings to apply them to the solution of human problems. It is when we acquaint ourselves with the truth revealed by the science of heredity that we are able to see what needs to be done and what may be done with human beings as biological and social organisms.

With this perspective of heredity, attention is now focused on the mental disorders and deficiencies that are influenced by heredity. The philosophical approach to psychiatry is ably discussed in the literature and it is pointed out that while the relations between the brain and the mind have exercised philosophers for centuries, we do not yet know enough of either to say how they are related. Neurology and psychiatry both have solid achievements to their credit, but there has recently been increasing expression of the view that although it is not at present possible to translate cerebral function into mental happening, yet the mental is capable of interpretation in terms of cerebral physiology, a view which subordinates psychology to neurology. Such interactionism involves more complex relations between brain and mind than

the theory of psychophysical parallelism.

There remain among psychiatrists sharp differences of opinion as to whether mental disorder is ever purely psycogenic. If both mental and physical causes of mental disorder exist, then either may ocassionally produce similiar mental symptoms. An emotional state following a head injury may be psychogenic or physiogenic or both. They are not mutually exclusive and either contributing factor may be large or small in any particular case. Similarly the wave theory and the particle theory of light, which appear mutually incompatible are nevertheless both necessary to an understanding of the action of light in its varying aspects. The correlation between cerebral and mental phenomena continues to be a fruitful field of research, and the unconscious mind is only a contradiction in terms if mind is first arbitrarily identified with consciousness.

CHAPTER II

THE MECHANICS OF GENETICS

In order to consider mental disorders and deficiencies, the basic concepts of genetics must be briefly discussed; this will accomplish a two-fold purpose, (1) to broaden the concept of genetics for the interested observer and (2) review for biologists the principles of genetics, since various portions of this paper will revolve around genetic laws and principles.

Conveniently, this phase of study is divided into three categories: (1) The Laws of Inheritance, (2) Genes, and (3) Cytoplasmic Inheritance.

The laws of inheritance include: (1) Law of Segregation, (2) Law of Independent Assortment, and (3) Law of Dominance and Recessiveness.

The Law of Segregation

The first law to be considered states that in the zygote the paired genes derived from the two parents which influence the development do not blend, but retain their individuality and segregate, unaffected by each other. They pass into different gametes, and are thus able to enter into new combinations when they unite to form a new

zygote. This first law of Mendel is known as the Law of Segregation. It is not convenient to set up experiments to test the Law of Segregation in man, but it is possible to illustrate this phenomena in lower animals.[1]

Speaking now entirely of physical traits or characters, it is known that any trait or character is under the control of pairs of genes, one gene being derived from one parent and the other gene from the other parent. Such corresponding pairs of genes are situated opposite each other in a single chromosome, and each member of such a pair is known as an allele (Greek allelon, of one another). Genes have multiple effects. When each of an allelic pair of genes produces the same effect the genes are said to be homozygous (Greek homo, same + zygos, yolked). When the genes of an allelic pair produce different effects, they are said to be heterozygous (Greek heter, different + zygos, yolked).

We can conclude from these facts, then, that the genes, influencing the development of characteristics, are present in pairs (alleles), and that each allele during meiosis enters a separate germ cell, which is the process of segregation. At fertilization, the separated genes are brought together in a random manner and pairing is by pure chance.[2]

---

[1]Ashley Montagu, Human Heredity (Cleveland, 1959), pp. 342-343.

[2]Ibid., p. 346.

The Law of Independent Assortment

In Mendel's findings of his eight years of experiments on the garden pea, he studied seven pairs of characteristics in the peas. Namely:

(1) Seed form.

(2) Color of seeds with coat.

(3) Color of seeds without coat.

(4) Form of ripe pods.

(5) Color of unripe pods.

(6) Position of flowers.

(7) Length of stem.

Studying F_1 generations of a cross between a pea plant possessing round yellow seeds with one characterized by wrinkled green seeds, he found that the plants of F_1 had all round yellow seeds. The F_1 hybrids were crossed, the F_2 generation showed the two original combinations of characters, round-yellow and wrinkled-green, and also the new combinations, namely, round-green and wrinkled-yellow, in the following proportions: 9/16 round-yellow, 3/16 round-green, 3/16 wrinkled-green, 1/16 wrinkled-yellow.

With this experiment, Mendel discovered the two additional laws of heredity, being "that pairs of alleles conditioning the different pairs of characters in the offspring are distributed independently of each other." In essence, this is the Law of Independent Assortment, or the Law of Free Recombination.

The Law of Dominance and Recessiveness

In the experiment described above, evidently the two color factors differed markedly in their capacity to express themselves. One apparently dominated the other. The same was true of texture factors. Factors that dominate over others are called dominants, and the characters that they prevail over are called recessives. Hence, in the experiments of Gregor Mendel, he found round-yellow to be dominant, and wrinkled-green (which remained unexpressed in the F_1 generation) to be recessive. In essence, this law implies that every character is represented by two genes, one derived from each parent. When these genes are different, one may dominate or cover up the other, which is present but not expressed. This is the Law of Dominance and Recessiveness.

Gene Action

At this point, we shall present supporting proof that the genes are carried in the chromosomes. We find it possible to state a series of principles in logical order. This is refered to as the gene influence.

The most detailed insight into the linear arrangement of specific structures along chromosomes has been gained from studies of the giant chromosomes of the salivary gland of fly larvae. These chromosomes, instead of resembling threads, look like wide cylinders and are marked by cross-bands or disks. Such microscopically visible linear

differentiation of chromosomes is highly suggestive of the linear arrangement representing hereditary properties as postulated by Roux and demonstrated by Morgan. These entities are called genes. Subsequent question to be asked: are the bands the genes themselves or are they, at least associated with them? The answer is: There is much evidence that they are associated; less evidence that they are identical.[3]

The general picture of the role of the gene helps us gain an insight into the interrelation between genes and characters, and particularly in the mental development. A character or trait, by definition, is any observable feature of the developing or the fully developed individual. For the genetic constitution, the term genotype has been coined; for the external appearance, the term phenotype.

The general concept of gene action lead us to expect (1) that no simple connection will exist between most observable characters of the developed human being and the single gene; and (2) that a single gene, by being part of a network of developmental reactions, will often influence more than a single character. We shall, at this point, briefly generalize on each concept.

<u>One character from many genes</u>: More single characters depend on many genes, and it is obvious that changes in any one of these genes may result in a change in the

---

[3]Curt Stern, Principles of Human Genetics (San Francisco and London, 1960), p. 25.

character. Thus, for example, hereditary blindness is known to be due to any one of very many different genes.

There are further consequences of the interrelation of gene initiated reactions. One is that individuals carrying identical genetic constitutions may look quite differently. This is due to environmental influence.[4]

Many characters from one gene: Such multiple effects of the above implication is called pleiotropic. An indication of this effect is the rare gene which controls an inherited condition, phenylketonuria.

Persons affected with this rare condition, unlike normal persons who possess the normal allele, excrete in their urine large amounts of phenylpyruvic acid as shown by addition of a few drops of ferric chloride solution resulting in a deep bluish-green color. A second trait in phenylketonuria is mental impairment, usually of a severe type, and a third trait is a slight pigmentary disturbance resulting in light hair.

The sperm and egg contribute equally to the inheritance of genes: Generally, both sperm and egg carry genes. However, there are exceptions as to equal distribution of the sex products. This is the case when either a sperm or egg carry a chromosomal abberation making it different from the other. Both of these exceptions serve only to strengthen the hypothesis that Mendelian genes are carried in the chromosomes, since genetic and cytological parallels

---

[4]Ibid, p. 27.

exist for them.

One more exception must be mentioned. In recent years evidence has been accumulating that the results of reciprocal crosses within a species are not always identical and that there is for some characters a maternal, hence cytoplasmic, transmission.

A striking series of parallels occurs between the behavior of genes and the behavior of chromosomes. The parallels are as follows:

(1) Genes and chromosomes normally occur in pairs in the cells of the individual.

(2) The two genes and chromosomes of each pair segregate in the formation of germ cells.

(3) Certain genes assort at random. Likewise certain parts of the chromatin material assort at random.

(4) Certain genes behave as though only one member of the pair were present in one sex (sex-linked genes). Similarly only one member of one pair of chromosomes is present in the corresponding sex.

(5) Certain genes do not assort at random but occur in paired groups (linkage groups) which tend to be transmitted as units. The chromatin material is also gathered into paired groups (chromosomes) which tend to be transmitted as units.

(6) The members of a linkage group do not stay completely together as a rule, but during the maturation exchange with a definite frequency homologous members of the paired groups (genetic crossing over). The pairs of chromosomes also exchange homologous parts of their lengths during the maturation of the germ cells (cytological crossing over).

(7) In certain genetic cases of crossing over, chiasmata formation is proportionately more frequent in the sex which exhibits more crossing over.

(8) At the time of genetic crossing over, the genes are arranged in a specific linear order. At the time of cytological crossing over, the chromatin material is in an attenuated linear arrangement.

(9) The number of linkage groups is as a rule definite and constant for any species. The number of linkage groups is equal to, or never exceeds, the number of pairs of chromosomes.

(10) Genes occasionally behave in peculiar unexpected ways (abnormal ratios, unusual linkage relationships, genetic deficiency). In these cases the chromosomes are also found to be abberant (non-disjunction, translocation, deletion, ploidy).[5]

Cytoplasmic Transmission

In recent years various observations and experiments have been made which suggest that some genetic variability is the result of self-duplicating, mutable units located not in the chromosomes but in the cytoplasm. Such units are apparently transmitted only by means of the cytoplasm, and therefore only the egg ordinarily contributes cytoplasm to the zygote. The inheritance in higher animals and plants is maternal.

One type of cytoplasmic transmission, plastid inheritance in plants has long been known but it was until recently considered an isolated and unique example. There are now on record a number of clear cases of cytoplasmic transmission in addition to plastid inheritance. These include certain structural characters in mosses, sterility in various plants, size and shape differences in the

---

[5]Laurence H. Snyder, The Principles of Heredity (Boston, 1951), pp. 300-301.

evening primrose, pathogenicity in rusts, color in wasps, sensitivity to carbon dioxide in Drosophilia, and a number of characters in protozoan Paramecium. Some of the examples mentioned are instances of purely maternal inheritance, others are cases where there seems to be an interaction of a Mendelian gene and a unit or units in the cytoplasm, but all involve cytoplasmic transmission.[6]

The researches of Sonneborn and his collaborators with Paramecium are of special importance in understanding cytoplasmic inheritance, and a brief account of some of them will be given here. Although many of the characters of this unicellular organism are controlled by genes carried in the chromosomes of the nucleus, other characters are apparently cytoplasmically determined. Such cytoplasmic determiners are often called plasmagenes. One kind of plasmagene in Paramecium is shown to be self-reproducing and capable of mutation. This kind of plasmagene depends for its maintenance and reproduction of nuclear genes, however, and is therefore not completely autonomous.

A detailed discussion can be carried out concerning cytoplasmic inheritance due to plasmagenes such as Kappa and to viruses such as the milk agent. Yet there is not assurance that all examples of cytoplasmic inheritance have the same physical basis and result from the same mechanism. Sonneborn's investigations already show fundamental differences in the mechanism with Paramecium. The

---

[6]Ibid., p. 305

differences are largely concerned with the extent of the dependence of the physical basis of cytoplasmic inheritance on nuclear genes. Whatever the explanation is, it seems likely that it will throw much light on the problem of how cells that are identical in genes, such as the cells of the various tissues in higher animals, can remain persistently different in structure.

It is not intended that the previous material on laws of inheritance, gene influence, cytoplasmic inheritance give a detailed account of the evidence for the existence of genes and cytoplasm. The phenomenon of inheritance, as such a task, belongs to a general treatise of genetics. Since, however, the concepts underly all discussions of human genetics, it was felt that a brief discussion of these concepts should precede the material of mental defects in human genetics.

CHAPTER III

MENTAL DISORDERS

Medical genetics is that branch of human genetics which is directly concerned with the relationship of heredity to disease. One of the most far reaching aspects of this problem is the study of hereditary mental defects and deficiencies. This problem is rapidly increasing in frequency. The feeble-minded often become juvenile delinquents, problem children, and cases for public charity and relief.

By breeding early they increase their kind. The end result is a rapidly mounting expense for their care and for dealing with their crimes. Of perhaps less alarming nature is the mental pathological traits exhibited by these cases.

Due to the broad scope of the topic, neurological disorders will not be considered. Among the hereditary ills with which human beings are affected, those affecting the development of the mind could be rated first in order of importance. These mental ills are of many varieties, but they will be considered or grouped in two fundamental classes:

(1) Mental deficiency--the lack of normal mental growth.

(2) Mental disorder----abnormal mental development.

Mental deficiencies, termed amentia, may be either due to hereditary causes (primary amentia), or to environmental factors (secondary amentia). Occasionally a childhood disease, such as meningitis or encephalitis, leaves a previously normal child with defective intelligence. Congenital syphillis is responsible sometimes for mental defects as well as nervous disorders of children.[1]

A small amount of mental deficiency is due to thyroid deficiency, inherited or acquired. Head injuries at birth and during development are probably responsible for a large number. The recent obstetrical practice of dosing the pregnant mother with barbiturates and similar drugs prior to delivery may so overload the fetal blood stream as to produce asphyxation in the fetus at birth, with either permanent brain damage or subtle damage that could lead to mental impairment. Fortunately, the trend today is away from heavy sedation.[2]

Our first consideration shall be concerning mental disorders. Geneticists postulate that mental phenomenon have a physical basis, and they follow with high expectations the work of physiologists and biochemists who attempt to find physical or chemical properties which characterize patients with specific mental diseases.

---

[1]S. J. Holmes, Human Genetics and its Social Import, (New York and London), p. 126.

[2]Montagu, p. 98.

Such findings it is felt, reveal the underlying action of genotypes which physiologically cause abnormal states of the brain and express themselves also in abnormal mentality.

Certain aspects of the interrelationship between bodily and mental states are demonstrated by the use of certain compounds which when taken by normal persons induce symptoms similiar to those found in persons with psychoses and by other compounds which when taken by the mentally ill persons change them temporarily toward normality.

Theoretically, the joint efforts of geneticists and psychiatrists could yield the conclusion that (1) mental disease is invariably fated by specific genotypes; or (2) that it is equally likely in all known genotypes; or finally (3) that nongenic influences lead to illness in some, but not all, genotypes. The first alternative is easily seen to be false, since identical twins are not always concordant with respect to mental illness. It is more difficult to decide between alternatives (2) and (3), complete versus partial nongenetic determination.[3]

Although there has been no definite investigation, there are general facts which point to genetic factors in mental illness or, to express the same idea differently, to genetic factors providing resistance to mental illness. Kallman stresses specificity. The two most frequent types

---

[3]Stern, p. 579.

of mental disorders are schizophrenia and manic-depressive psychosis.

Schizophrenia

It has been found that among the relatives of schizophrenia propositi the frequency of schizophrenia is higher than in the general population, but that the frequency of manic-depressive psychosis is not increased.

As further supporting evidence which implies schizophrenic disposition is in the case of identical twin sisters who were separated from one another at an early age and who both became mentally ill. The sisters hardly had any contact during the first ten years, and little contact later. At the age of 15, one, a factory worker, gave birth to an illegimitate child, while the other lived as a domestic servant in the sheltered home of a private family. Nevertheless, both became schizophrenic- the first shortly after the birth of her child, the second about one and a half years later.[4]

This and other concordances are suggestive of a constitutional background of the disease, but they are still compatible with purely environmental interpretations. It lies in the nature of twin studies that the existence of nongenetic factors in mental disease, as in other traits, is proven beyond doubt whenever discordant identical pairs

---

[4]Ibid., pp. 580-581.

are encountered. In the absence of any proof to the contrary, however it will be assumed here that greater discordance of nonidentical than of identical twins and greater incidence of affected relatives of affected propositi, in mental disease as well as in most other traits, is in part due to genetic causes.

The incidence of schizophrenia in the general population has been estimated as approximately 1 percent. This disease, which frequently begins during the third decade of life and thus has been called dementia praecox or early insanity is characterized by a cleavage in personality that, in extreme cases, may necessitate a permanent institutional confinement for the patient.[5]

Moreover, schizophrenia is responsible for 20 percent of first admissions to state hospitals and 29 percent of the readmissions. The category schizophrenia recognizes four types of behavior; (1) apathy and carelessness, (2) stillness and bizzare ideas, (3) negativism and mutism, and (4) paranoia, which displays delusions of persecution and grandeur.[6]

The children of one schizophrenic patient have a probability of developing the disease 19 times that of the general population. The grandchildren, nieces, and nephews are about 5 times more likely to show a recurrence

---

[5]Ibid., p. 582

[6]Frederick Osborn, Preface to Eugenics, (New York), pp. 39-40.

of schizophrenia than is the average person. The schizophrenic rate of the parents approximates 10 percent. The expectancy for brothers and sisters as well as fraternal twins partness is somewhere around 14 percent. The highest morbidity rates are found among the children of two schizophrenic parents, who have about 80 times the average expectancy, and among the identical twin's partners of schizophrenic index cases who show an expectancy rate of 85 percent.

Rosanoff and his workers in this country have made use of twin material to throw light on the genetic factors underlying schizophrenia. They found, for instance, that where one identical twin was a schizophrenic, the twin mate was also affected in 68 percent of the cases. Where one fraternal twin was affected his mate (of the same sex) was affected in only 15 percent of the cases.[7]

Is the high concordance of schizophrenia in identical twins due to the mental shock which the onset of the disease in one twin causes in the other? The data on non-identicals show that this shock, in most cases, is not sufficient to cause the disease if the genotypes of the twins are different from each other. It is, of course true that the mental reaction of an identical twin to the illness of his twin partner is probably different from that of a nonidentical twin, who knows his fate is dissimilar in many other ways. It seems unlikely, however, that this difference in psychological attitude can account

---

[7]Ibid.

for the observed difference in concordance.[8]

Do all schizophrenics possess the same abnormal genotype? The evidence is not clear, as is so often for a trait whose presumed genetic basis is not fully penetrant. Phenotypically schizophrenia appears in strikingly different forms, and some indications have been found of a tendency for similar types of the illness to occur in the relatives of an affected propositus. While administering batteries of psychological tests to a number of mentally retarded patients of the familial type who had been referred because of recent schizophrenic episodes, some observations were quite revealing.

The examiner found a peculiar ease in which he could communicate with the retarded schizophrenic. The question arose as to why this was possible with these patients, and not with more intelligent schizophrenics.

Meager and qualitative observations indicate that perhaps mental deficiency prevents an individual from developing a very idiosyncratic and high level symbolic world of his own, forcing him to draw from limited intellectual and informational resources.

After extensive clinical observations, three main hypothesis were offered:

(1) Low capacity for symbol formation makes it more difficult for a mentally retarded person to flee into an inaccessible world of his own phantasy.

---

[8]A. J. Rosanoff et al., "The etiology of so-called schizophrenic psychoses, with special reference to their occurrence in twins," American Journal of Psychiatry, XC (1931), p. 247.

(2) The mentally retarded schizophrenic may be intrinsically easier to treat than the bright schizophrenic.

(3) Delusions or even delusional systems in retarded people are often due to forced attempts to make sense of events they cannot otherwise explain, rather than being secondary symptoms of a schizophrenic process. Also inability to validate constructs perseveration of a false conclusion.

In spite of the suggestions of genetic diversity, it is possible to assume that a single gene is the necessary prerequisite for schizophrenia, with minor genes in the genetic background accounting for the specific form of the illness. Actually, it cannot yet be decided whether a single factor difference or perhaps polygenic differences account for the genetic basis of schizophrenia.[9]

Manic-Depressive Psychoses

Manic depressive insanity is marked by alternating periods of excited elation and abysmal depression. Some affected individuals are violent and destructive. Mild degrees of the condition, however are compatable with satisfactory social adjustment, and borderline cases of good mentality are in some instances highly creative persons.[10]

Manic-depressive illness has an incidence in the general population of somewhat less than ½ of 1 percent.

---

[9]Wolf Wolfensberger, "Schizophrenia in Mental Retardates." American Journal of Mental Deficiencies, LXIV (1960), p. 704.

[10]Stern, p. 583.

Empirical data on its incidence in relatives suggest inheritance with dominance involved, and further evidences imply that the role of heredity in manic-depressive psychosis is as great or greater than in schizophrenia.

Rosanoff and his co-workers, studying identical and fraternal twins as to occurrence for one or both members of the twin pair, found that, where one identical twin was affected, the other was affected in 70 percent of the cases. Among the fraternal twins of the same sex, when one twin was affected, the other twin was affected in only 16 percent of the cases.

According to Kallman, among couples where one parent is a manic depressive, from 30 to 33 percent of the children will be afflicted. In addition, there will be a number of individuals who have manic-depressive tendencies to a lesser degree, yet not enough to merit a complete diagnosis.[11]

Epilepsy

Hippocrates expressed the opinion that epilepsy is a familial disease and the question of its inheritance has been a subject of debate ever since. In recent years new methods of attack have aided in separating the various types of inherited and non-inherited epilepsy.

Many of the diseases for which it seems a carrier state can be recognized are rare. One of the problems

---

[11] F. J. Kallman, "The Genetics of Schizophrenia: A Study of Heredity and Reproduction in the Families of 1,087 schizophrenics." J. J. Austin, Inc., (1938), p. 164.

with which medical genetics is concerned is the detection of the characters present in the carriers of inherited diseases. Epilepsy provides an example of a poorly defined carrier state. Spontaneously developing epilepsy is at least in part, genetically determined. The brain waves of epileptics are abnormal in 84 percent of affected persons.[12]

Epilepsy, like insanity and feeblemindedness, has long been observed to run in families to a certain extent, although it may appear sporadically for reasons not understood. There are many kinds of epilepsy, those causing spasms or unconsciousness, which may be caused by injury or disease. However, this discussion is limited to the inherited cases.

Inherited epilepsy usually behaves as a recessive or partly recessive trait, although Dordan has described a few families in which it seems to be an irregular dominant. In one family in which both parents were epileptic, one daughter was epileptic and the other was not. The epileptic daughter had four children of whom two had the malady. One of these produced two sons, both epileptics while the other epileptic individual had a daughter so affected. One normal daughter married and produced three epileptic children. The daughter of the original pair that did not develop epilepsy married and had two children, one normal and one epileptic. The epileptic offspring

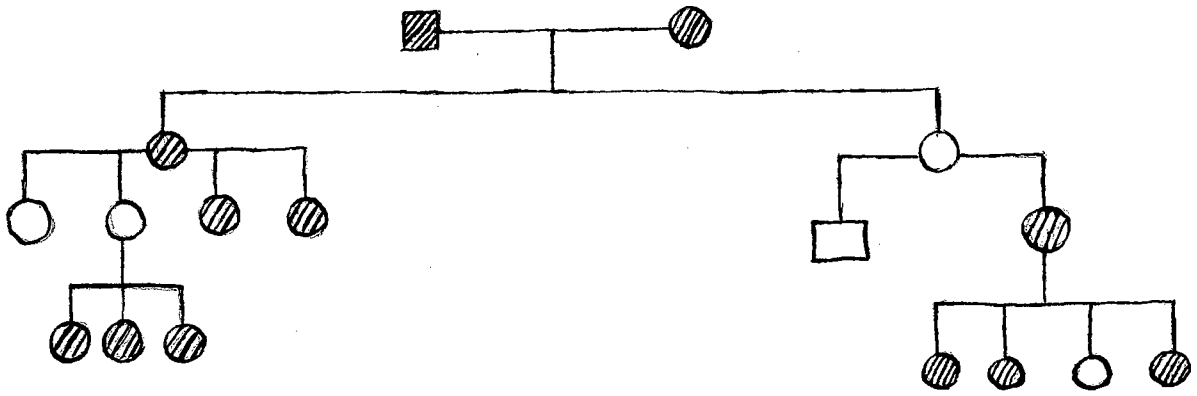
---

[12]Holmes, p. 143-144.

married and produced four children, three of whom became afflicted with the same disorder.[13]

Such a clustering of cases within a family can hardly be due to chance. It is likely that the different kinds of epilepsy are inherited in different ways.

TABLE I
THE INHERITANCE OF EPILEPSY THROUGH FOUR GENERATIONS



Owing to the fact that epilepsy is usually a recessive or partially recessive character of relatively rare occurrence, many cases of inherited epilepsy doubtless arise in families in which no other instances of its appearance have been observed. This circumstance has lead many writers to minimize the role of heredity in the production of this disorder.

In four pairs of presumable monozygotic twins reported by Humm, epilepsy appeared in both members in three cases and in only one member in one case. In all six pairs of dizygotic twins with epilepsy, the malady was confined to one

---

[13]Ibid.

member of each pair. These twin studied are exceptions to the probable genetic factors that are so pronounced in schizophrenics and manic-psychoses patients.

A description was given of a pair of identical twin boys in which both were affected with a peculiar type of epilepsy beginning at an early age. Dr. J. Sanders of Holland studied a pair of twins, Mien and Nel, who were said to be monozygotic by the physician who attended their birth. Both girls had their first attack of epilepsy in their fourth year within a few days of each other, and the later course of the seizures was much the same in both. In another pair of identical twins, Jan and Cor, epileptic attacks occurred in both boys before the end of their second year.

The general character of the attacks, their premonitory symptoms, and after-effects were similar in both boys. The father was also subject to epileptic seizures from his twelfth to his thirtieth year. In the third pair of twins both members had their first epileptic seizure on the same day, but one boy commonly had more severe symptoms than the other; and while both appeared to be about equally bright, the twin who was afflicted to the greater degree suffered more mental deterioration than the other, so that there came to be a considerable disparity in their general intelligence.[14]

---

[14]Ibid., p. 145.

Professor Abadie contended that epilepsy is a constitutional disorder in which heredity plays a very minor part. Instances of epilepsy in twins are not very numerous, but the evidence thus far accumulated points to the importance of genetic factors in causing this disorder, although they may require the cooperation of some unfavorable environmental influence to enable them to become manifest.

Epilepsy sometimes classified as a neurological disorder, was considered in this section because it ranks next to schizophrenia and manic-depressive insanity in the extent of the suffering it causes.

In essence, a study of electrocephalograms of members of a family give evidence that the condition is strongly inheritable. The expectancy of genuine epilepsy is about 2.9 per 1000.[15]

---

[15] Osborn, p. 44.

CHAPTER IV

MENTAL DEFICIENCIES

Oligophrenia, or mental deficiency, persists in those who are sufficiently subnormal and require more or less supervision. This large group probably includes a large number of different genic types, many of which are simple recessives. The condition can be accidentally produced, but Mapother concluded after an extensive experience that almost all oligophrenics who remain at large until the reproductive age are of the inherited type.

A highly debatable issue in our society has been concerning the worth of the mentally defective and their place in society. In the scale of humanity every human being has a value, whatever his qualities may be, and unless he is a complete idiot, even if he is feeble-minded, as long as he is able to work he can be a useful member of society. As the great geneticist J. B. S. Haldane has said: "I am of the opinion that a man who can look after pigs or do any other steady work has a value to society, and we have no right whatever to prevent him from reproducing his like."

Clearly, mental deficiency is not so much a biological

as a social problem, for in a society in which occupational status would be measured by ability, places could be found for persons of very limited ability. It is estimated that well over 2,000,000,000 mental defectives are in the United States presently. There are also at the present time some twenty-eight states scattered throughout the country that laws prevail concerning the sterilization of mental defectives. Over 60,000 have been sterilized since the first of these laws was passed in 1907.

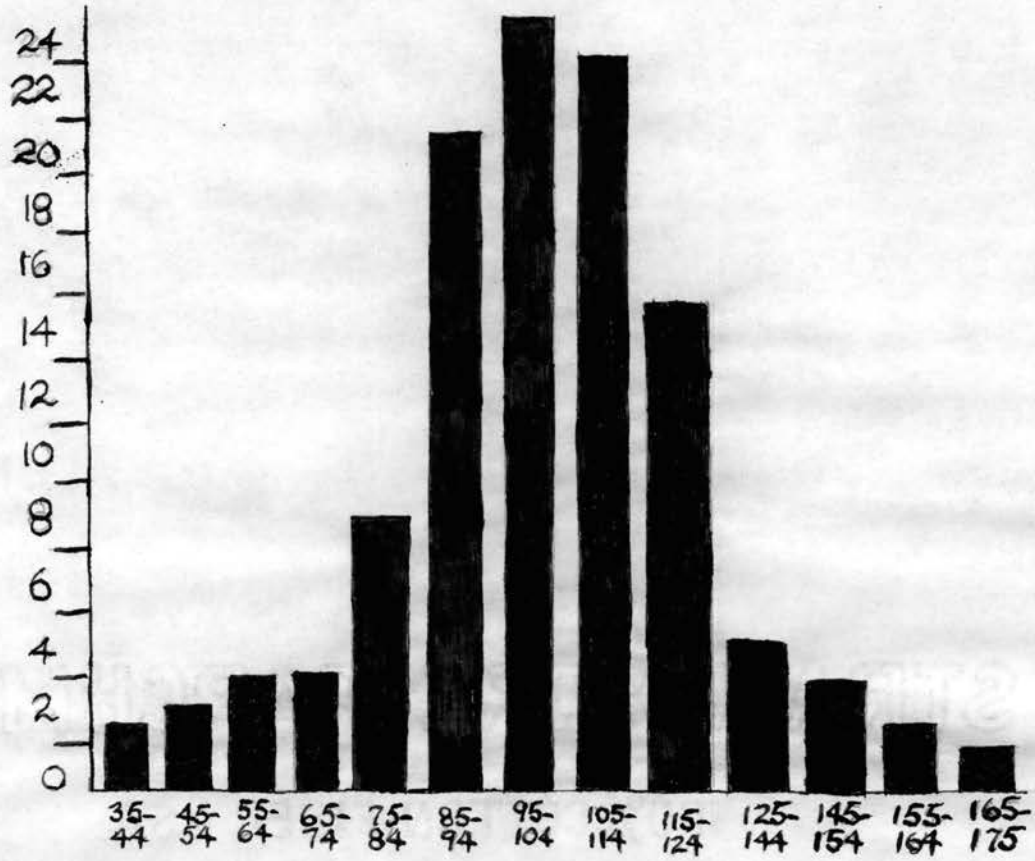
TABLE II. PARENTAGE OF MENTAL DEFECTIVES

Matings of Parents	Character of offspring		
	Normal	Dull	Mentally Deficient.
Superior X Superior	126	13	49
Superior X Dull	36	20	24
Superior X Defective	11	14	20
Dull X Dull	8	12	17
Dull X Defective	0	1	12
Defective X Defective	1	3	18

Mental defects do not reproduce themselves efficiently. The idiots, those who belong to the lowest grade of feeble-minded, and are unable to do anything for themselves, have a mortality rate which is more than five times greater than that of the general population. In the younger ages, their death rates are nearly ten times greater than that of the

general population. The imbeciles, who can learn simple manual tasks, have a great death rate that is about twice as high as that of the general population. Among morons, who have a mental capacity reaching to the borderline of low normal intelligence, death rates are also higher at every age than for the normal population. The greater the degree of feeblemindedness among the mentally defective, the lower is their reproductivity, so that a natural limiting factor is operative upon their multiplication.[1]

TABLE III. DISTRIBUTION OF INTELLIGENCE LEVELS THROUGHOUT THE GENERAL POPULATION



[1]Montagu, p. 295.

The great majority of mental defectives are born to perfectly normal parents. Hence, the sterilization of the mentally defective would scarcely result in a significant reduction in the number of mental defects born. The difficult part of the process of eliminating feeblemindedness would be to eliminate feeblemindedness from the germ plasm of the general population.

TABLE IV. FREQUENCIES AND COMMONLY APPLIED I. Q. LEVELS: DESIGNATIONS

Frequency (%)	I. Q.	Designation	
4.7	140 or over	"near genius, genius"	
	130-139	"very superior"	
21.2	111-129	"superior"	
48.6	90-110	"normal"	
24.2	80-89	"dull"	
	70-79	"borderline"	
1.3	50-69	"moron"	mentally deficient
	25-49	"imbecile"	
	0-24	"idiot"	

Classification of the Mentally Deficient

Idiots are persons who have failed to develop mentally beyond the level usually attained by a child of about two years of age. Like most two-year olds, they are unable to guard themselves against common physical dangers, and can feed and dress themselves, but are incapable of earning their own living.

Morons are defined as persons who are capable of managing themselves under conditions requiring or involving much competition, but who, under supervision, may become at least partially self-supporting; they have also been defined as those who cannot receive proper benefit from ordinary elementary schools but who may be benefited in special classes. Adults whose mental ages are between eight and ten are generally classified as morons, but it should be obvious that diagnosis of mental deficiency at this level must be based primarily on social competence. We can be fairly sure that anyone with an I. Q. below 60 will require supervision, and therefore be classed as a moron. But, depending in part on their temperaments and in part on the environment in which they may find themselves, some individuals with I. Q.'s over 70 may have to be considered in the moron category, while others with I. Q.'s of 65 or even lower may be sufficiently self reliant to escape this classification.[2]

Mongolian Idiocy

Mongolism (Mongoloid or Mongolian idiocy) also obscure ethiologically and easy to identify clinically, is a comparatively common type of mental defect, making up from 5 to 10 percent of all defects. It is detected by a peculiar assortment of physical traits such as

---

[2]Snyder, pp. 443-444

stunted growth, slanting palpebral fissures with epicanthic folds, short nose, enlarged and fissured tongue, and flat facial features vaguely resembling those members of the Mongol race. Most of the symptoms are recognizable at birth and are not progressive. The intelligence defect is usually severe (I. Q. range is 15-40), and early mortality from intercurrent infection is high.[3]

There is evidence that morons and imbeciles tend to come from families with high fertility. While the idiots are themselves infertile, the other categories of mentally deficients are not. Some writers think that when mental deficiency runs in families it may be partly for cultural reasons such as poverty and lack of mental stimulus or opportunity. Every normal individual sooner or later selects his own mental and physical environment, even though obstructed by poverty. Mental defect is often recognizable in infants in arms. It is also pointed out that feeblemindedness and dullness are familiar to a much greater extent than the low grade defectives.

In Mongolian idiocy, sexual development is incomplete. Practically all Mongolian idiots are sterile. Therefore, the genetically determined origin, if any, has been assumed by mutation. Evidence supporting a genetic etiology has been chiefly derived from twin data, with invariable concordance in one-egg pairs and very

---

[3]Kallman, pp. 220-221.

infrequent concordance in two-egg pairs. Additional factors in favor of genetic mechanisms are the following:

(1) The extraordinary physical likeness of Mongoloid defectives does not seem consistent with the action of variable exogenous influences.

(2) There is a moderately increased incidence of the disease in siblings resulting in the following empirical risk figures: The frequency of Mongolism in the general population is somewhat between 1:500 and 1:1500, but any woman who becomes pregnant after age 40 runs a statistical chance of about 1 to 6 percent of having a Mongoloid child; the chances are 4 percent for any woman who has borne a Mongoloid child to give birth to another Mongoloid child, a risk 40 times greater than the average of all ages.

(3) The increase in abortion frequency in mothers of Mongoloids indicates, if some of these aborted foetuses are assumed to be Mongoloid, that familial occurrence of the disease may be higher than usually recorded.

In 1946, D. K. Kenyon began the excavation of a burial site at Breedon-on-the-Hill in Leicestershire. It is fairly certain that this is a late Saxon burial ground, probably associated with a monastery of the 8th and 9th century.

Evidence lies in the cranial material, and includes all but twenty specimens. Unfortunately Mongoloid skeletal material is not easily available for examination, and only four skulls of other types of mental defectives were available. Other evidence is provided by publications and a study of Mongol children at Harperbury Hospital.

The cranial capacity of the skull, is estimated to be 835 cc., whereas a small crevice of normal skulls gave

the same range between 1130-1290 cc. Considering its small size in terms of the length, a feature which is typical of Mongoloids, is the most outstanding feature. The breadth is next most affected and the height is the least. This order of reduction is the reverse of normal skull growth.

The vault, unlike that in true microcephaly, is very globular in form. More emphasis has been placed on hyperbrachycephaly as one of the diagnostic characteristics of Mongolism. However, although this feature is important in diagnosing Mongolism, it is by no means infallible, and it must be remembered that extreme degrees of brachycephaly can be associated with other forms of idiocy. In fact, one skull of an 18-year old epileptic idiot collected by Greig has a cephalic index of 98. Some Mongoloids are relatively longheaded.

Briefly then, the anatomical features which strongly suggest Mongolism in the Saxon child are:

(1) Microcephaly, as shown particularly by vault length, breadth, and cranial capacity.

(2) Hyperbrachycephaly, the index being abnormally high for Anglo-Saxons (whether children or adults).

(3) Small sphenoid body and high angle of the basioccipital.

(4) Thinness of the cranial bones.

(5) Small maxilla but fairly normal mandible size.

(6) Minor anomalies of the dentition.

Other minor features also suggest Mongolism in the Saxons, but will not be discussed.[4]

When a pair of twins boys with Mongolism was admitted to the children's unit of the mental hospital "Voorburg" it was decided to make a study of the chromosomes for two reasons: (1) chromosomes in Mongoloids might tend to elucidate monozygosity and (2) providing material on the chromosomal influence.

The pair was born as the eleventh pregnancy of a 43-year old mother. They presented definite physical resemblances to Mongolism. Blood groups of parents and children gave a chance for monozygosity of 99.5 percent.

Both children had Mongoloid traits with brachycephaly, epicanthus, fissured tongue, short thick fingers, Mongoloid palm prints, and hypermobility of the joints. Child C has a cardiac anomaly. Meanwhile, W died revealing an open ductus Botalli and a septum anomaly by autopsy.

Bone marrow puncture was made at seven months. A short term culture of bone-marrow cells was produced and good metaphases were scanned in aceto-orcein squashes.

For both parents, the modal chromosome is numbered at 48. The cell from C with greater than 50 chromosomes had exactly the double modal number, i. e. 96 chromosomes. From W, eight cells with 48 chromosomes were analyzed and from C six cells, all revealing a Y-chromosome. Sixteen

---

[4]D. R. Brothrell, "A Possible Case of Mongolism in a Saxon Population." Annals of Human Genetics. XXIV (1960), pp. 141-149.

chromosomes in groups 6-12 and an extra chromosome number of 21.

It was not possible to obtain a clear result, but the two X-chromosomes are present because a skin biopsy conducted later revealed the presence of Barr-bodies in the interphase nucleus. Most probably both parents are of the chromosomal constitution $AA+A_{21}XXY$.

The findings were of interest in several respects:

(1) This is the first time that it was possible to conclude monozygosity in a twin pair on karyograms. There is a very low chance that two individuals had the same double chromosome abberation through unrelated events and obviously this twin arose from one abberant zygote.

(2) The anomalous modal chromosome complement proved to be stable during cleavage, foetal and early postnatal life. However the zygote originated from a non-disjunction mechanism.

(3) The children are too young for Klinefelter's Syndrome but the chromosome complement gives an early diagnosis.

(4) This is the fifth case known in which Mongolism is associated with 48 chromosomes and with an $AA+A_{21}XXY$ idiogram. The number of the regular type with 47 chromosomes from primary non-disjunction and with trisomy for A_{21} is not represented as most cases studied are not yet published. The translocation type with forty-six chromosomes seem to be rather frequent as already several independent families have been found. Selection for mothers' age and familial Mongolism makes it rather easy to find such cases. The Klinefelter type with 48 chromosomes from a double non-disjunction was found in most cases by selection through clinical signs or Barr-bodies. It would seem that there is more frequent association of Mongolism with Klinefelter's syndrome than the combined frequency of Mongolism (about 1 in 500) and males having positive sex chromatin would suggest.

(5) A stickiness has something to do with the anomalies in chromosome distributing mechanisms. Harden observed that satellite chromosomes show this phenomenon which suggests a lag during the interphase. Nondisjunction during meiosis probably is due to the same lagging in separation from heterochromatic substances that are fused during the interphase stage of the mitotic division.

The next description will point out carefully the physical and developmental differences between cytologically confirmed Mongoloids and our reported case which must be put back into the great pool of mental deficiencies with unknown etiology.[5]

A 7½ year old boy is the child of healthy parents. His father had eight normal children in a previous marriage. The patient is the only child of his mother. No other cases of mental retardation are known among relatives. The mother was 40, the father was 42 years old when the boy was born. The mother was totally unaware of anything amiss with the little boy until the age of 2½ years, when following a routine physical examination, the attending physician told her the boy was a "borderline child." At the age of four, the boy was examined at a medical school and the diagnosis of Mongolism was established.

The overall picture is certainly that of Mongolism, although there are some important inconsistent findings which are probably the exceptions which prove the rule. For this reason he is considered a borderline case. For

---

[5]T. W. Hustinx, P. Eberle, and S. J. Geerts. "Mongoloid Twins with 48 Chromosomes (AA+A21XXY)," Annals of Human Genetics. L (1961), pp. 111-114.

example, this boy is advanced in longitudinal growth which is very unusual. His head is not as small as the usual case of Mongolism---and he does not appear so severely retarded as many. His personality differs from the usual case. On the other hand he possesses a short, broad neck, his hands and fingers are short, and he has a wide gap between first and second toes.

White blood cells separated from 10 cc of the patient's blood were grown in tissue culture for four days. Phytohemagglutinin was added as an agent to induce mitotic activity. Thirty-one good mitotic figures were selected for chromosomal counts. Twenty-one of these showed 46 chromosomes, four had 45, three had 44, and two had 43 chromosomes. One appeared to have 47 but the photograph of this figure showed that a chromosome from a neighboring metaphase plate had joined the group.

No clinically typical case of Mongolism has yet been reported which cytologically has shown a normal chromosomal complement. The cases in which 46 chromosomes were found, all show chromosomal abberations, which with good reasons are being interpreted as translocations. The supposition at the end of the cytological paper with other causes for the development of syndromes of the Mongolism type has not been supported.[6]

---

[6]Werner Schmid, Chi Hao Lee, and Priscilla Smith, "At the Borderline of Mongolism: Report of a Case with Chromosome Analysis." American Journal of Mental Deficiency, LXVI (1961), p. 449.

Phenylketonuria

Phenylketonuria (phenylpyruvic amentia) is a striking instance which a definite relation is shown between a biochemical error of protein metabolism and imbecility. It seems clear that the mental defect is a direct result of the metabolic arrangement. Follings recorded ten inmates of a Norwegian Hospital who excreted phenylpyruvic acid in the urine. Nine of them were undoubtedless feeble-minded, the tenth was only a year old and its mentality was uncertain. This condition was not found in any mentally normal person, including the normal relatives of those affected. Penrose tested the urine of 500 inmates of the Royal East Counties Institution and found two brothers, idiots, excreting phenylpyruvic acid. The parents and the two siblings were normal. This type of amentia is caused by failure of the body to oxidize phenylpyruvic acid and is inherited as a simple recessive condition.

Later, Penrose found a family in which there had been four idiots and an imbecile in the offspring from two cousin marriages. The surviving imbecile, age forty-one, excreted phenylpyruvic acid. The latter condition was regarded as the heterozygous state of a single recessive gene which disturbs the metabolism.

In affected individuals of phenylketonuria, the condition is present from birth. They are also usually short, with smaller heads, the reflexes are generally

very active and show dermatological peculiarities (eczema) and abnormal skin pigmentation. The mental defect ranges from profound idiocy to a very mild impairment. Another feature of interest is that the hair is usually light, so that phenylpyruvic oligophrenia appears to be related biochemically to albinism.

Jarvis examined 8043 inmates representing all forms of mental defect in Chicago. He found 59 affected in 37 families with 176 siblings. He applied Hogben's correction for families in which it fails to appear, and confirmed a recessive inheritance. Of these case, forty-two had a peculiar syndrome dominated by mental deficiency with phenylpyruvic acid excretion.

Such results emphasize that mental diseases are physiological disturbances of the whole organism. It is pointed out that the mental effects of anoxemia (lack of oxygen in the blood) are well known. Disturbances in the nervous system will result in a diminished rate of oxidation and produce mental disorder.[7]

Recent work made it clear that there is a causal relation between mental retardation in phenylketonuria and the existence in these patients of an inborn defect in the enzyme system of the liver for the conversion of phenylalanine to tyrosine. The exact nature of the casual relationship is unknown but it is evidenced

---

[7]Gates, Reginald R, Human Genetics (New York, 1946), pp. 1088-1090.

by:

(1) The demonstration of the enzymic defect in mentally retarded phenylketonuric patients.

(2) The almost invariable occurrence of a low grade mental deficiency in persons with this chemically demonstrated metabolic block.

(3) Mental improvement seen in many phenylketonuric patients when placed on a phenylalanine low diet.

In vast studies a confirmed correlation of the biochemical and psychological attributes of phenylketonurics. The data showed mental retardation to be marked in the first born, slight in the second, and nil in the third.

The older boys are used in the studies and were chemically phenylketonurics while younger boys show no evidence of this metabolic block. All three boys are of similar abnormality but the youngest has the best regulated pattern of the three. The children placed on a phenylalanine restricted diet indicated some subsequent improvement in I. Q. and behavior. The other children were followed in an attempt to assess any progression of the disease.

The findings imply that a trial of therapy for phenylketonurics of I. Q.'s in the moron range will be worthwhile even if they are identified at a relatively advanced age.[8]

---

[8]B. Tischler et al., "Degrees of Mental Retardation in Phenylketonuria," American Journal of Mental Deficiency. LXV (1961), pp. 726-734.

The Degeneration Theory

A hundred years ago it would have been taken for granted that the connection between fertility and mental disorder could be summed up in the word "Degeneration." In some stocks or families, French psychiatrist B. A. Morel declared that a morbid deviation from the normal type could occur, most often in the form of mental abnormality. It would then exhibit progressive degradation. In the first generation the abnormality would become manifest as nervous temperament, moral deflect, instability, and excess psychopathic personality. In the second generation it would appear as severe neuroses, alcoholism, and general paralysis. In the third generation it would burst into full bloom as melancholia, paranoia, and hyperchondria. A fourth generation, if the stock survived, would consist of people with idiocy, gross malformation, and other barriers to procreation.

By the turn of the century, the fatalistic picture of diminishing fertility, decay, and then extinction of the degenerate stock had been pretty well dropped. An entirely opposite and more alarming view took its place. Instead of nature wiping out the hereditarily diseased and insane by rapid, benignant reduction of their fertility, man was now seen as the blind agent of a social and biological process which would increase the relative fertility of the mentally ill, and consequently the total amount of mental illness.

Dr. David Heron concluded that pathologically abnormal stocks are not less but rather more fertile than the normal stocks in the community. Dahlberg set about an inquiry into the fertility of insane women. His propositi were 2,200 women admitted to the Upsala Mental Hospital. Of these, 1,219 had been under observation until they had passed the age of forty-five and were therefore unlikely to have any further children. These women had born in all 2,407 children. The average number of children per woman was 1.97. This 1.97 was in contrast to 3.78 for the general population of Sweden and indicated that the insane have a lower fertility than the sane.

These and other investigations by Dalton and Essen-Moller show that of the two commonest and gravest mental disorders, manic-depressive psychosis is characterized by approximately normal fertility, and schizophrenia by a much lower fertility than normal, largely because of a lessened capacity for marriage.[9]

The strong evidence for the presence of a single Mendelian unit in many families with mental defect was shown. Sjogren gives a brief account of twenty-five families in which there were thirty-four cases of oligophrenia combined with congenital bilateral cataract.

---

[9]Aubrey Lewis, "Fertility and Mental Illness," Eugenics Review, L (1958), p. 566.

The inheritance was recessive, affected individuals showing strong psychic inhibition of mental development. A study was conducted of the mental defects in Denmark, which has a school for high grade oligophrenics with an I. Q. for 70-90 (dullards and simpleminded) and an asylum for low-grade imbeciles and idiots. They investigated fifty families in each group. They found the asylum material distributed more or less at random in the general population while the higher grade defectives were drawn largely from the unskilled laborers. This general survey confirms that of the Mental Deficiency Survey of England.

Feeblemindedness has been considered as a simple recessive Mendelian trait, but there is much evidence that it is usually the product of a variety of genetic factors.

There has been a great problem of the classification of the mentally deficient. Objections have been raised as to the use of such terms as "idiots," however most of the literature uses such terms and they are followed in this study as the most acceptable and widely used terminology in the literature.

CHAPTER V

BRAIN AND CENTRAL NERVOUS SYSTEM OF MENTAL DEFECTIVES

Many important studies have been made of the brain of mental defectives. They seem to show that visible and palpable defects are present, corresponding roughly to the degree of mental deficiency. As early as 1895, Hammarberg showed by counting the cells that imbeciles are deficient in the number of cells in the cerebral regions. Ellis found that the same was true to the cerebellum.

Evidently microcephaly (pinheadedness) can arise from different causes. Spencer described the case of two microcephalic sisters from normal parents who were first cousins. The gene for this defect was evidently recessive. The development of the calvarium was arrested, and without an operation the children would continue as idiots. Bernstein described the family of German descent in New York State with five cases of microcephaly in the sibship of ten with normal parents and grandparents (apparently recessive). All of the relatives were of normal intelligence, but the father is said to have had a markedly low forehead.

Whitney gives a pedigree of two feebleminded immigrant

families, one Swedish, and one German, who intermarried. In four generations they produced at least twenty-eight feeble-minded and three normal children. The inheritance was recessive except for these three, who were perhaps illegitimate.

Benda proposed to distinguish oligoencephaly (stunted brain) from microcephaly. He made 130 autopsies of mental deficients and an anatomical analysis of 20 familial cases with a mental age of four to ten years. In these familial cases there were many developmental anomalities of the spinal cord and brain. He connected nine pedigrees of the condition in two to five generations with forty cases (inheritance dominant) and regards oligoencephaly as a hereditary developmental disorder.

Grebe described two cases of microcephaly in a pedigree with much consanguinity, indicating recessive inheritance. Among the relatives were found asymmetry, deaf-mutism, congenital dislocation of the hip, as well as anomalities of the fingers, toes, and teeth, together with debility, epilepsy, and high grade myopia.

Berry cites the work of von Economo in Vienna on the cyto-architectonics of the brain. He found that in an unselected series of 1,391 mental deficients in Melbourne Hospital, 62.2 percent were definitely microcephalic. Of 8,533 normal school children, 19.5 percent were of similar microcephalic dimensions, while of 131 adult female defectives at Stoke Park Colony, 67 percent

were microcephalic. No particular shape of head was associated with mental deficiency, but the shape of the head tended to be abnormal, and besides the two-thirds who were microcephalic, another 25 percent were found to be abnormal macrocephalic levels.[1]

From this evidence it will be seen that widely different views have been expressed regarding the method of inheritance of some types of mental defect. Yet practically all are agreed that heredity is mainly concerned in their production. Increasing volumes of evidence indicate that mental defectives have smaller brains with visible defects in the cells of the cerebral cortex.

---

[1]Gates, pp. 1134-1139.

CHAPTER VI

SUMMARY AND CONCLUSION

Most people would agree that at the present time mental health is one of the most important aspects of health as a whole. To measure it is difficult. What criterion can be used to say that a man is not healthy.

The symptoms of diptheria are the same in patients of different ages, but in mental disorders and deficiencies a galaxy of conditions, ranging from the temper tantrums of childhood, through the emotional immaturity of adolescence, the schizophrenia of the young adult, the depression of the middle aged, to the confusional state of the aged arteriosclerotic.

The repercussions of mental illness are bound to affect the whole population. In addition to the heart-break of families or friends when a loved one is a victim, an account of the financial burden which mental illness places on a community is more than substantial.

In America, a recent report sponsored by the Joint Committee on Mental Illness of the United States has assessed the cost 3,000 million dollars a year.

In this paper, an attempt has been made to show the powerful effects of genetics on mental disorders and

deficiencies. Attempts were made to point out the grave problem it imposes. Also the unique method of transmission was considered.

We, nevertheless, are living in times of great significance, and many efforts are being made to tackle this problem in all aspects.

Many hospitals are changing their institutional appearance completely. The drab wards of yesterday are giving place of gay and cheerful surroundings. New drugs are enabling many to maintain themselves in the community, who otherwise would have had to go into a hospital. The ugly image of mental illness is being slowly eradicated.

Mental illness follows the pattern taken in the conquest of many other diseases; first we find how to cure the disease and then how to control it. Geneticists and psychiatrists can unite their efforts to combat this national problem.

Because of its difficult origin, eradication is perhaps many years away, but new scientific methods and more complete findings by scientists can be coupled with a better informed public in being an efficient tool in effectively utilizing modern methods in combating mental illness.

SELECTED BIBLIOGRAPHY

Brothrell, D. R. "A Possible Case of Mongolism in a Sax Population." Annals of Human Genetics. XXIV (1960), pp. 141-149.

Gates, Reginald R. Human Genetics. New York: The MacMillan Company, 1946.

Holmes, S. J., Human Genetics and its Social Import. New York and London: McGraw-Hill Book Company, 1936.

Kallman, F. J. Heredity in Health and Disorder. New York: W. W. Norton and Company, 1953.

Lewis, Aubrey. "Fertility and Mental Illness." Eugenics Review. L (1958), 209-213.

Montagu, Ashley. Human Heredity. Cleveland and New York: The World Publishing Company, 1959.

Osborn, Frederick. Preface to Eugenics. New York: Harper and Brothers, 1951.

Schmid, Werner, Chi Hao Lee, and Priscilla Smith. "At the Borderline of Mongolism: Report of a Case with Chromosome Analysis." American Journal of Mental Deficiency, (LXVI (1961), p. 449.

Stern, Curt. Principles of Human Genetics. San Francisco: W. H. Freeman and Company, 1960.

Wolfensberger, Wolf. "Schizophrenia in Mental Retardates." American Journal of Mental Deficiency, LXIV (1960), p. 704

VITA

James Baker Abram, Jr.

Candidate for the Degree of

Master of Science

Report: THE ROLE OF HEREDITY IN INFLUENCING MENTAL DISORDERS AND DEFICIENCIES

Major Field: Natural Science

Biographical:

Personal Data: Born at Tulsa, Oklahoma, December 5, 1937, the son of James B. and Beulah Tibbs Abram.

Education: Attended grade school at the Roosevelt School, near Ardmore, Oklahoma; graduated from Douglass High School, Ardmore, Oklahoma in May, 1955; received the Bachelor of Science degree from Langston University, Langston, Oklahoma, in May, 1959; did graduate work at Prairie View A&M College, Prairie View, Texas, and Texas Southern University, Houston, Texas; completed requirements for the Master of Science Degree in May, 1963.

Professional experience: Began a teaching career in September, 1959, at the Douglass High School, Ardmore, Oklahoma, and taught general science, biology, chemistry, physics, and mathematics from 1959-1962.

Professional organizations: Kappa Delta Pi, Alpha Kappa Mu, Phi Sigma.