

Ouachita Baptist University

## Scholarly Commons @ Ouachita

---

Honors Theses

Carl Goodson Honors Program

---

1-1970

### The Basics of Behavior Genetics: A Study of Heredity

William Edwin Walker

*Ouachita Baptist University*

Follow this and additional works at: [https://scholarlycommons.obu.edu/honors\\_theses](https://scholarlycommons.obu.edu/honors_theses)



Part of the [Biological Phenomena, Cell Phenomena, and Immunity Commons](#), [Genetic Phenomena Commons](#), [Genetic Structures Commons](#), and the [Medical Genetics Commons](#)

---

#### Recommended Citation

Walker, William Edwin, "The Basics of Behavior Genetics: A Study of Heredity" (1970). *Honors Theses*. 330.

[https://scholarlycommons.obu.edu/honors\\_theses/330](https://scholarlycommons.obu.edu/honors_theses/330)

This Thesis is brought to you for free and open access by the Carl Goodson Honors Program at Scholarly Commons @ Ouachita. It has been accepted for inclusion in Honors Theses by an authorized administrator of Scholarly Commons @ Ouachita. For more information, please contact [mortensona@obu.edu](mailto:mortensona@obu.edu).

H136.3  
WALK

The Basics of Behavior Genetics  
A Study of Heredity

Behavior  
Genetics

Presented to Dr. Weldon Vogt  
January, 1970  
In Fulfillment of the Requirements  
For Special Studies H492

by William Edwin Walker

H. 2. 1. 1

## INTRODUCTION

The general issue of the significance of genetic contributions to individual differences may be approached in two ways, through population genetics and through physiological genetics.<sup>1</sup> The first has no logical meaning when applied to an individual, for his whole genotype and total life experience contribute to every aspect of his behavior, and their influences cannot be separated. The second is rather light on the emphasis of environment and its influence.

The two approaches to the problem of individual differences complement each other. Knowledge of heritability is paramount when one attempts to change phenotypes by selection. Possibly the most significant contribution of behavior genetics is its documentation of the fact that two individuals of superficially similar phenotypes may be quite different genotypically and respond in completely different fashion when treated alike.<sup>2</sup> Knowledge of how genes produce effects on behavior is often sought for its practical importance. If one can counteract the effect of a genetic lesion by biochemical means, seriously defective individuals may be restored to health. The dual approach to the problem of individual differences has dictated a division of this paper into two sections followed by a general summary.

---

Footnotes for this paper have been combined in list form at the end of this paper preceding the bibliography.

## BEHAVIOR AND POPULATION GENETICS

The adaptive behavior of nature is almost a truism. In order to survive, organisms must respond to stimuli in a way which results in the satisfaction of tissue needs and the execution of reproductive functions. The accepted explanation for the correspondence between needs and behavior is the evolution of behavior mechanisms through natural selection.

The natural selection theory of behavioral evolution postulates three related processes. First, random selection and genetic variation occurs within a population. Second, this results in variable behavior some forms of which are better adapted than others to the environmental challenges which are encountered. Third, the better-adapted individuals are more successful in reproduction, and the genes which are necessary for superior adaptation increase. The process has no definite end point, and evolution is a contemporary process as well as a historical one. Obviously the evolution of behavior is explicable by this mechanism only to the extent that behavior is heritable. Superior adaptation not related to genes could be transmitted culturally but not biologically.

Two contrasting types of adaptive evolution have been recognized. In one, structures evolve which produce a relatively stereotyped response to critical stimuli impinging upon the organism. Through natural selection each stimulus-response pattern is stabilized as the one most likely to permit survival and reproduction. The second type of adaptation involves the evolution of structures which become organized in

the course of their functioning to produce the most adaptive response to particular circumstances. The stimulus-response patterns themselves are not stabilized by natural selection but by learning. The two forms of adaptation are not mutually exclusive, and man still depends on the innate protective reflexes, although learning plays so important a role in his behavior.

Allen has suggested that the central-nervous system of the higher mammals may show instability because of its rapid and recent structural evolution.<sup>3</sup> Not enough time has elapsed for natural selection to have eliminated genes with deleterious effects upon brain function. This has met with wide disapproval because of the noted increase of mental illness and retardation in our population. It is one of the problems which may be solved when more is known in the field of genetics.

Methods for determining the factual degree of genetic contribution to behavior variation in man differ with the nature of the gene-character relationship. A good example is a study on the frequency of the gene for microcephalia vera, a form of feeble-mindedness inherited as a recessive. Table i presents the data from this study.<sup>4</sup> The most interesting feature of this table is the tenfold difference in gene frequency between the larger cities, in which panmixia is a reasonable assumption, and a number of isolates with moderate numbers of consanguineous marriages.

It is clear from this one example that one cannot specify the risk of genetic disease population-wise in general terms. More extensive regional data collection is needed to determine the relative importance of genetic factors in the production of mental defect caused by specific genes.

Population-genetic models have been well tested with traits such as the ability to taste PTC and have also been employed with such characters as handedness, dyslexia or word-blindness, and schizophrenia.<sup>5</sup> In the last four characteristics, the expression of the trait is modifiable by the environment, and various assumptions regarding the penetrance must be made to achieve a fit to the standard models. The validity of such assumptions is difficult to prove, and their plausibility is somewhat dependent upon the nature of the trait investigated. The dominant mode of inheritance of PTC tasting has been established by population studies. Hallgren's hypothesis that dyslexia is inherited in a similar fashion has not been generally accepted, although the genetic evidence is of a nature similar to that of PTC. The point is that PTC taste-blindness is readily conceivable as the resultant of a rather simple metabolic variant. The relating of word-blindness, a variation which does not influence intelligence in general, to a unit process on the metabolic level is difficult to integrate with modern neurological ideas.

Another use of population genetics is exemplified by Slater's computations from data on incidence of schizophrenia in a number of countries.<sup>6</sup> He has undertaken to test Book's hypothesis, that the disorder is caused by a partially dominant gene with complete manifestation of the disease only in homozygotes, by comparing results of several family studies. The consistency of the results from independent surveys suggests common etiological mechanisms in all populations. Psychiatric characters do not follow the classical models of population genetics perfectly, but the models do appear useful in computing morbidity risks.

The heritability of intelligence has been variously estimated. The highest heritability values proposed are those of Burt and Howard who ascribed about 69% of variance in intelligence to genetics, 17% to assortative mating (also genetic), and only about 14% to environmental factors and unreliability.<sup>7</sup> These estimates may impress some as being over high, but it must be remembered that the equalization of educational opportunities will have the effect of increasing heritability, since environmental sources of variance will be simultaneously reduced.

On the other hand today it is also known that intelligence of infants can be increased by environmental setting for a majority of individuals.

Even though the heritability of intelligence under certain circumstances is high, too little is known of the interaction between heredity and environment to make accurate predictions concerning the effects of natural selection on this trait or even to classify it as a single trait. We live today in an era of rapid cultural and educational change, and the effects of these factors upon intelligence probably obscure any effects of genotypic changes.

The racial diversity of man has long been recognized, but population genetics provided the first quantitative means of evaluating such differences. Races have been defined as relatively homogenous groups of interbreeding individuals characterized by a particular set of gene frequencies. Do the well-established differences in gene frequencies imply psychological differences as well? Strains of animals show behavioral differences correlated with their diversity

in genotypes, and it can be argued that the same must be true of human races. Such a view need not imply racial superiority, merely racial differences. (In most cases it is taken the wrong way). The evidence to prove the point one way or the other does not exist, although there are numerous contentions. There are reasons to discount the likelihood of such differences being very important. The most diverse human cultures have common features related to the perpetuation of the species. It is difficult to conceive of a society in which intelligence, cooperation, and physical vigor would not have positive selective value. Hence it is likely that natural selection tends to oppose the establishment of major heritable behavior differences between races.<sup>8</sup>

A similar question may be asked regarding the genetic basis of intelligence differences between social classes. Although there is great overlap in the intelligence-test scores of individuals from different social classes, there are real differences in average performances on various psychological tests.<sup>9</sup> In the opinion of some investigators, social class differences in intelligence are simply a reflection of cultural stimulation. This is very widely accepted in the field of sociology and education today. It is still very difficult, though, to distinguish the environmental factors responsible for genius in the slum area and a mediocre student from a professional family. In view of the strong evidence for the heritability of intelligence and the occurrence of assortative mating with respect to intelligence, it is possible that some social class differentiation exists with respect to genetic factors affecting intelligence, and this divergence may in-



crease if social-class membership becomes more dependent upon competitive effort in a society with high social mobility.

These opinions regarding race and class differences may appear to be contradictory. The point is that natural selection in man operates at the level of whole societies. Intelligence is necessary for survival of a society, but it is not necessary that all members of the society be superior, and in fact a complex society has niches for its dullards as well as its geniuses. The less able members of a society benefit from association with the talented leaders.

EUGENICS---The essential idea of eugenics is that artificial selection be substituted for natural selection in the evolution of man. The program has generally been divided into negative eugenics, concerned with the elimination of major defects, and positive eugenics, the encouragement of reproduction by the most able elements of the population. Negative eugenics is considered now to be chiefly a matter of counselling with voluntary action based upon genetic predictions. The counselor can be definite only with those characters which show single-factor patterns of inheritance, and these are relatively rare in behavior genetics.

Positive eugenics is less direct in its approach and might actually be described as an attempt to give direction to natural selection.<sup>10</sup> Osborn has given an excellent account of the modern eugenics movement. The basic idea is to work toward a social organization which promotes the formation of stable families and provides satisfactory niches for those who are incapable of these responsibilities.

The population aspects of behavior genetics have not been widely studied with quantitative techniques. Nevertheless, it appears that a considerable portion of the behavioral variability of both wild and laboratory races is attributable to heredity. Surveys of genetic variation in behavior in populations of small mammals would be very useful in developing general laws for the nature-nurture relationship.

In man the adaptive nature of behavior is largely insured through the process of learning. Genetic variation, however, provides a second mechanism for adjusting to different environmental conditions including perhaps different social roles. Both modes of behavioral adaptation are the product of organic evolution through natural selection. Since natural selection differs in several important ways from artificial selection as usually practiced in laboratory experiments, it would be highly instructive to study the evolution of behavior in the laboratory using natural selection instead of directed selection. Such experiments would test the hypothesis that major changes in the nature of selection will always influence behavior in a relatively permanent fashion by changing the composition of the gene pool.

Finally the eugenics movement has been considered as a proposal to substitute directed for natural selection in human populations. As applied to deleterious characters inherited in simple Mendelian patterns, it is reasonable that man should use his scientific knowledge to prevent the conception of children likely to be severe social burdens. Beyond this, our knowledge of human genetics is insuf-

ficient to base further recommendations, particularly since we do not know the nature of the future society to which our descendants must be adapted.

#### BEHAVIOR AND GENETIC TRANSFER

Although it is possible to demonstrate hereditary effects without understanding the mechanisms involved, there are good reasons for probing more deeply. The modification of heritable defects is more likely to be successful if we understand how the causative genes are acting. Furthermore, the discovery of a pathway for gene action gives more concreteness to the concept of heritable behavior. Proof that a particular psychological difference between strains fits a one-factor Mendelian model is more convincing when some physical link can be found between the presumptive genes and the observed behavioral variation. In short, behavior genetics becomes intellectually more satisfying as it bridges the gaps between genes and psychological traits.

The problem of the relationship between gene and character is central to physiological genetics, and the difficulties are great even when concern is limited to physical traits. With respect to behavioral traits, there is relatively little which has been firmly established. Nevertheless, there is value in summarizing and generalizing to the extent now possible, in full realization that changes may soon be required. Experimentation in the area is desirable, for genetics can

become a useful tool for the behavioral scientist seeking to find a physiological explanation for individual differences. //

The ordinary technique of physiological genetics research is to start with a specific well-defined phenotypic difference and work backward toward genetic sources of variation. The reverse order is more suitable for presentation of general principles. Behavior is the response of an organism to stimulation of external or internal origin. Genes operate at the molecular level of organization, but they are peculiar kinds of molecules, highly individuated carriers of information, whose effects are describable in psychophysiological as well as chemical terms. Enzymes, hormones, and neurons may be regarded as successively complex intermediaries between genes and psychological characters. We shall investigate each of these.

ENZYMES ...According to the gene-enzyme hypothesis, the effects of genes upon behavior must always be related eventually to some metabolic effect of the gene within the cell. In this sense variations in hormones, nerve structure, and the like are the outcome of more basic enzymatic differences. It is convenient to consider these more complex pathways separately, since the links between the primary gene functions, and their structural consequences are known only in the most rudimentary fashion. The classic example of a gene controlled metabolic lesion with important behavioral effects is phenylketonuria. A block in the oxidation pathway of phenylalanine leads to the accumulation of phenylketone and related substances. That the

cell

effects upon intelligence are produced by toxic action of the abnormal metabolics is indicated by the fact that afflicted individuals with reduced phenylalanine intake are psychologically improved. Without the raw material no toxic substance is produced.

Phenylketonuria and hereditary obesity are examples of metabolic lesions which are compatible with life, but which produce phenotypic differences far beyond the ordinary range of the species. Both conditions are inherited as simple Mendelian recessives. The concept of less drastic metabolic lesions is also fundamental in Williams' genothropic theory of alcoholism. He speaks of partial genetic blocks which can apparently vary quantitatively and thus be responsible for biological and psychological behavior variation within the normal range.<sup>12</sup>

One of the most direct attempts to link body chemistry with heritable difference in behavior has already been described as cholinesterase, an enzyme which catalyses the breakdown of acetylcholine to choline and acetic acid. Acetylcholine is one of the chemical mediators in the peripheral and central nervous systems. High concentrations of an enzyme are taken to indicate a high level of the metabolism of the enzyme substrate. In this instance, the concentration of cholinesterase might be taken as a measure of readiness of synaptic transmission. Within limits ease of transmission might be conducive to adaptive learning.

/ The approach to the gene-behavior character relationship

through enzyme studies has the advantage of being close to the gene end of the chain, but this advantage is counterbalanced by distance from behavioral events. One may employ genetic lesions, using Ginsburg's phrase, to "naturally dissect" the nervous system at the metabolic level. But this dissection is not the same as separating out natural units of behavior. More must be learned regarding the relationship between biochemical individuality and behavior before the findings of the biochemist can have psychological meaning. In the expanding area of psychochemistry, genetics will have a unique role, for genes are the only way in which permanent chemical characteristics can be built into an organism. Selective breeding for biochemical characters is well known in plants and can be achieved with animals for characters of psychological interest, such as the cholinesterase concentration. The methods are laborious, but some shortcuts may be possible through the use of strains already available.

HORMONES....The relationship between hormones and behavior was reviewed a few years ago by Beach.<sup>13</sup> The potential mechanisms through which hormones might control behavior were grouped under four headings.

- 1-Hormones may affect behavior through effects upon the organism's normal development and maintenance activities. Such effects, exemplified by the multiple deficiencies of the cretin, are relatively non-specific.
- 2-Hormones may control behavior through stimulation of structures employed in specific response patterns. For example, the postnatal growth of genital organs is dependent upon hormones, and adult sexual behavior cannot occur until these

structures are fully developed.

3- Behavior may be controlled through effects upon peripheral receptors, sensitizing them to particular forms of stimulation. This possibility has not been much explored, but there is positive evidence for it.

4- Behavior may be controlled through effects of hormones on the integrative functions of the central nervous system. This possibility has attracted considerable attention since Beach's review, and a number of studies have dealt with the effects of hormones directly injected into the brain.

The fact that evidence can be found for each of these possibilities does not mean that all are involved in the production of heritable individual differences in behavior. A distinction must be made between psychophysiological actions of hormones and psychopharmacological effects of large doses applied in artificial ways. The latter type of effect has little significance for the genetics of normal variation.

An additional complication in the analysis of the gene-hormone behavior relationship is that genes might operate upon a source of the hormone, affecting the quality and the quantity of the product or upon the target organs, affecting their response. Furthermore, the endocrine system is physiologically complex, with much inter-

action between components, None of the four types of mechanism described by Beach or the two means by which genes might act are mutually exclusives. The choice of pathways is more than adequate.

Since courtship behavior is intimately dependent upon hormones, it might be expected to provide good evidence on the points in question. Young's group has maintained that individual and strain differences in the sexual activity of guinea pigs are functions of target-organ sensitivity rather than amount of a sex hormone produced.

The relationship between sex hormones, experience, and behavior varies widely among species, and generalizations from guinea pigs to man should be made cautiously. Many studies have shown wide variations in sex hormones in humans, but there is no real evidence that such variations have any direct effect upon sexual behavior, provided they are not so extreme as to interfere with normal development. <sup>it</sup> On the other hand, it would be wrong to conclude that, because even pseudo-hermaphrodites adopt the gender role of their rearing in spite of genetic or endocrine discordance, endocrine variations have no effect. Feminization of the male, or masculinization of the female features have obvious social repercussions.

A decrease in the size of the adrenal glands has accompanied the domestication of the Norway rat. Among domestic strains, rats of a very emotional nature had larger adrenals and thyroids than non-emotional stock. Variation in the thyroid glands of dog breeds was reported by Stockard. Some of the subjects of his experiments were conditioned in the classical Pavlovian manner.



Thyroidectomy was deleterious to establishment of a conditioned response, but it is not clear from the published report that individual differences in conditionability were directly correlated with thyroid status.

At the time of this research, radioactive-tracer methods for studying thyroid function were not available. Using such techniques, inbred mouse strains have been found to differ widely in rate of thyroid-hormone output. The high output strains are those found in other studies to be more active.

. A number of endocrine disorders in man, diabetes mellitus and Grave's disease among them, are heritable. In untreated diabetes mellitus blood-sugar concentration fluctuates widely with accompanying changes of mood and appetite. The victim of Grave's disease is hyperactive, sometimes to the point of mania. Extreme variants in the endocrine system do have behavioral consequences related to the physiological disturbance, but the opposite relationship, that between extreme behavioral deviation and endocrine disorders, is not as clear.

To review adequately the literature of endocrinological psychiatry would lead too far afield, but a few comments will suffice as illustration. Schizophrenics are frequently extreme deviants in endocrine function tests, but the relationships are not perfect; observers are not in agreement as to the nature of the deviations, and the effects of institutionalization are confounded with possible genetic effects. Familial investigations have generally shown a hereditary basis for both the psychosis and the endocrinopathy, but

except for acromegaly, the correlations of the psychosis with the endocrine dysfunctions were no greater than those predicted from random association of independent variables. In acromegaly, a behavior syndrome attributable to diencephalic disturbance was prominent. Although not causally related, an endocrine dysfunction may affect the course of psychotic disease. For example, Bleuler reports that schizophrenia in a physically infantile person differs from the disease in one whose genital development is normal. Sexual fantasies and aberrations are less frequent in infantile persons.

In summary, the pathway from genes to behavior through the endocrine system is real but narrow. Although much exploration is yet to be done, it is likely that variations in target-organs response will have greater significance for behavior genetics than variations in hormone output. In fact, strain differences in response to hormones seem to be very common.

THE NERVOUS SYSTEM....Despite the importance of variation in the nervous system as a path whereby genes might come to influence behavior, few studies have dealt directly with the problem. We have previously given cursory attention to the large variety of heritable neurological defects which produce profound changes in behavior due to interference with anatomical pathways. Genes which lead to major neurological defects have been found in many species. They show considerable

uniformity in their manifestations. One group of these, the lipidoses, is characterized by abnormal lipid disposition in the brain, but these have not yet been related to specific enzymatic processes. An interesting feature for some lipidoses and Huntington's chorea is their long latency. Onset of the disease follows long periods of apparently normal functioning. How the presence of the causative genes becomes manifest only at a late stage of development is not clear. Perhaps a developmental error occurs early, but function is adequate until a defective part wears out. Many neurological diseases are progressive, and it is often difficult to specify exactly when they began. In these instances of gross defect in the nervous system, the behavioral correlates depend upon the region of the nervous system affected. At present the primary metabolic lesions have not been identified.

The simplest quantitative attribute of the nervous system is its size. Mere mass of the brain is considered to be good measure of the psychological capacity of the related species. But brain weight by itself has not provided a reliable indicator of psychological differences within a species.<sup>16</sup> It is natural, therefore, to look for less crude morphological differences which might be correlated with behavior. A number of interrelated questions must be asked. Is there substantial individual variation in the fine structure of the central nervous systems of the higher vertebrates? Is such variation heritable? What significance does it have for behavior?

Evaluating the behavioral significance of fine neural-structure variation when there is so little evidence must be speculative. It seems reasonable that the functions of a nerve network would be altered when the number of connections is increased, and it is conceivable that the structure of the network is a heritable character. A full-scale research program in the area would repay the effort, for even negative results would be important.

Heritable peripheral variation is well known to affect behavior. A simple example is the differential thermal preference of mice shown to be dependent upon skin thickness. There is, however, no convincing evidence that differences in temperament and intelligence are related to any obscure inherited sensorimotor deficits or advantages. Tryon was unable to show any correlation between sensory factors and the maze-bright and maze-dull differentiation in his rats. In human beings, even extreme sensory deficits are compatible with high intelligence if adequate educational procedures are employed. <sup>17</sup>

Peripheral variations have specialized significance in social species, for they serve as cues for discriminatory responses or determine success in competition. The heavy dog wins his fights and becomes dominant. The myopic boy reads because glasses interfere with sports. These events affect the development of personality, yet genes affecting weight and the eye are not usually considered "behavior genes".

The search for anatomical and physiological channels through which genes contribute to variation in behavior has been successful to a limited extent. A few enzymes have been implicated; hormones play a significant role; neurological defects have behavioral consequences. But many behavioral differences clearly shown to be heritable have not been reduced to problems in biochemistry or electrophysiology. Perhaps investigators have not looked in the right places. Or it may be that behavior measures are the only reliable indicators of certain kinds of inherited organic characters. Physiological and anatomical techniques have limitations, since the measuring devices themselves impair the intactness of the subjects.

These limitations have stimulated some psychologists to use behavior tests themselves to find psychological components which could have genetic significance. The idea is that the traits might be found by methods such as factor analysis which are biologically more real than test scores chosen empirically. Results of these observations on the heritability of factor scores are now primarily concerned with the general implications of the method in behavior genetics.

Factor analysis begins with a matrix of intercorrelation between a number of measures and by a series of statistical manipulations determines a smaller number of factors which can explain the variances of the original scores. There is no mathematically unique solution of such a matrix. Many psychologists have employed

Thurstone's concepts of "simple" structure and "positive manifold". The first means that each test shall have loadings on as few factors as possible; the latter requires rotation of axes to eliminate significant negative factor loadings on all tests. Thus the description of the traits is the most parsimonious possible, and high ratings on factors never imply low scores on any test. This requirement is probably defensible in the area of intelligence testing in which Thurstone was particularly interested, but its validity in the realm of temperament is less obvious. Both these criteria are intrinsic to the original matrix; that is, they are applied to the relationships between the dependent variables as expressed in the test intercorrelations. Having no definite relationship to causal factors, they do not necessarily lead to factors which make biological sense. By itself, factor analysis leads to more parsimonious description, not to hypothesis testing.

An attempt to relate factor theory to genetics emphasizes the multiple factor control of independent processes which can collectively be called intelligence. Royce's model (Table ii) assigns blocks of genes to various group factors.<sup>18</sup> The relationship between the genotypes, S, M, etc., and their respective mental traits, space, memory, etc., is not stated in the theory. Presumably the action is direct, since other genes are postulated to have indirect effects through the nervous or endocrine systems. The most notable feature of the Royce model is the idea of congruence between genetic and psychological elements, a concept which is implicit in much hypothesizing in behavior genetics. In the Royce model,

it leads to a distinction between direct and indirect (nervous and endocrine) actions of genes upon intelligence, but the nature of direct action is not defined.

In the hands of more biochemically oriented investigators, all genetic effects are considered to be chemical. When combined with the concept of congruence, however, this leads to a sort of biochemical phrenology in which the enzyme systems replace bumps on the cranium, each enzyme controlling a psychological function. But when single genes are found which affect behavior, they affect not only one but a variety of intellectual and temperamental traits. Phenylketonurics are low in all the primary mental abilities. About two-thirds show abnormal neurological symptoms in addition to mental defects; bizzare behavior such as echolalia and echopraxia may be more common than in some other defectives. Psychotic episodes and epilepsy are relatively common. A simple biochemical lesion does not affect a limited segment of behavior but modifies development in many ways. Since biological genes systems and psychological factors are not congruent, factor analysis will not automatically yeild a genetic analysis.

Correlations between traits may rise from genic, chromosomal, gametic, or environmental communalities. A diagram of genic commuality is shown in Table iii. The correlation between traits  $\hat{a}$  and  $\hat{e}$  is a function of the contribution of physiological character 1 to each. This character is, in turn, controlled by gene D. Both  $\hat{a}$  and  $\hat{e}$  have genetic variances (from genes A, B, C, E, F) which are either specific or shared with other traits. The short arrows extending

from physiological-level traits are considered to run to other behavioral traits omitted from the figure.

On the right side of the table (iii) is a diagram of chromosomal communality. The covariation between traits  $\hat{e}$  and  $\hat{o}$  is dependent upon the linkage of genes F and G. It will not be important in large random-breeding populations, but may be significant in small groups of related individuals.

Gametic communality is illustrated in Table iv. The associations of traits  $\hat{a}$  and  $\hat{e}$  and their opposites  $\hat{a}'$  and  $\hat{e}'$ , are maintained only as long as a non-random mating system is followed. Since assortative mating is characteristic of humans (with respect to social class, intelligence, etc.) it is conceivable that factors could be generated by the gametic correlations produced. The critical issue is whether assortative mating is partly based upon genic and gametic communalities or solely upon environmental ones.

The diagrams of genetic communalities were drawn without reference to environmental variance. In Table v, traits  $\hat{a}$  and  $\hat{e}$  are shown with both environmental and genetic contributions to variance. A portion of each type of variance is common to the two traits; other portions are independent. Some such arrangement is probably representative of the actual situation. If traits  $\hat{a}$  and  $\hat{e}$  are subsumed under a common factor, z, because of their covariance, this is a function of event II as well as gene C. If this figure is representative of the true relationships between variables affecting behavior, one would not expect a simple factor analysis to lead to purely biologically or purely environmentally determined factors. Possibly this limitation can be removed by developing



new techniques which include genetic characteristics in the original correlation matrix. <sup>19</sup>

Genetic effects upon behavior are sometimes mediated through metabolic lesions which interfere with specific enzymatic reactions. Partial genetic blocks have also been implicated as causes of behavioral variability, but the evidence for this is less clear.

Endocrine disorders are generally coordinated with behavioral changes, but this does not mean that quantity of hormones-- provided it is within the normal range-- has critical effects upon behavior. Heritable variations in target-organ sensitivity are probably more important than variations in hormone output as sources of individual psychological differences.

Except for gross defects which impair normal functioning, little is known of the behavioral significance of structural variation in the nervous system. The relationships of neuron density and patterning to individual psychological differences may be worth exploring.

The search for more suitable means of behavioral description for use in genetic studies has converged on factor analysis. The method has promise, but must be interpreted with full understanding of the possible genetic meaning of correlation coefficients.

A working model for the gene-behavioral character relationship emphasizes the non-congruence of the two levels of description. Non-congruence implies multiple factor control of psychological traits and the existence of complex gene interactions in the development of

phenotypes. In spite of the complexity the evidence for lawful genetic effects upon behavior has been amply demonstrated. Further analysis in the gene-character relationship may be possible from experiments in which genotypes are manipulated and phenotypic effects measured.

Now that scientists have at last isolated the "gene" much study is indicated to related behavior and heredity.

TABLE i

Estimates of the Frequency of the Gene  
for *Microcephalia vera* in the Netherlands  
(van den Bosch, 1957)

Place	Gene frequency	Total Population
Four large cities (Amsterdam, Rotterdam, Den Haag, Utrecht)	0.000187	2,333,346
Eight Isolates	0.0285	68,427
Huissen	0.0151	
Etten	0.0263	
Lenmen	0.0250	
Elst	0.0285	
Putten	0.0285	
Didam	0.0181	
Hardinxveld	0.0200	
Enkhizen	0.0164	

TABLE ii

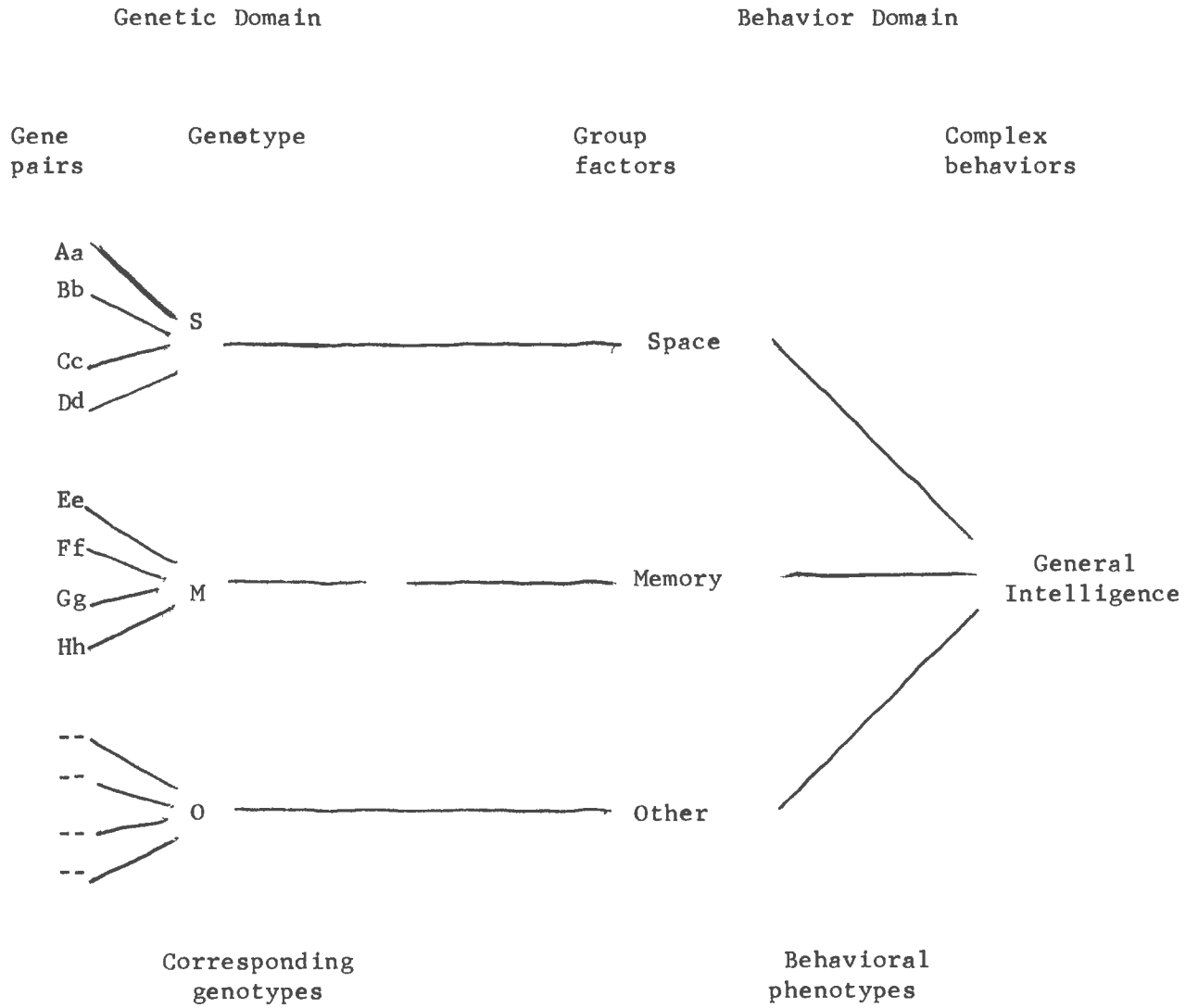


TABLE 441

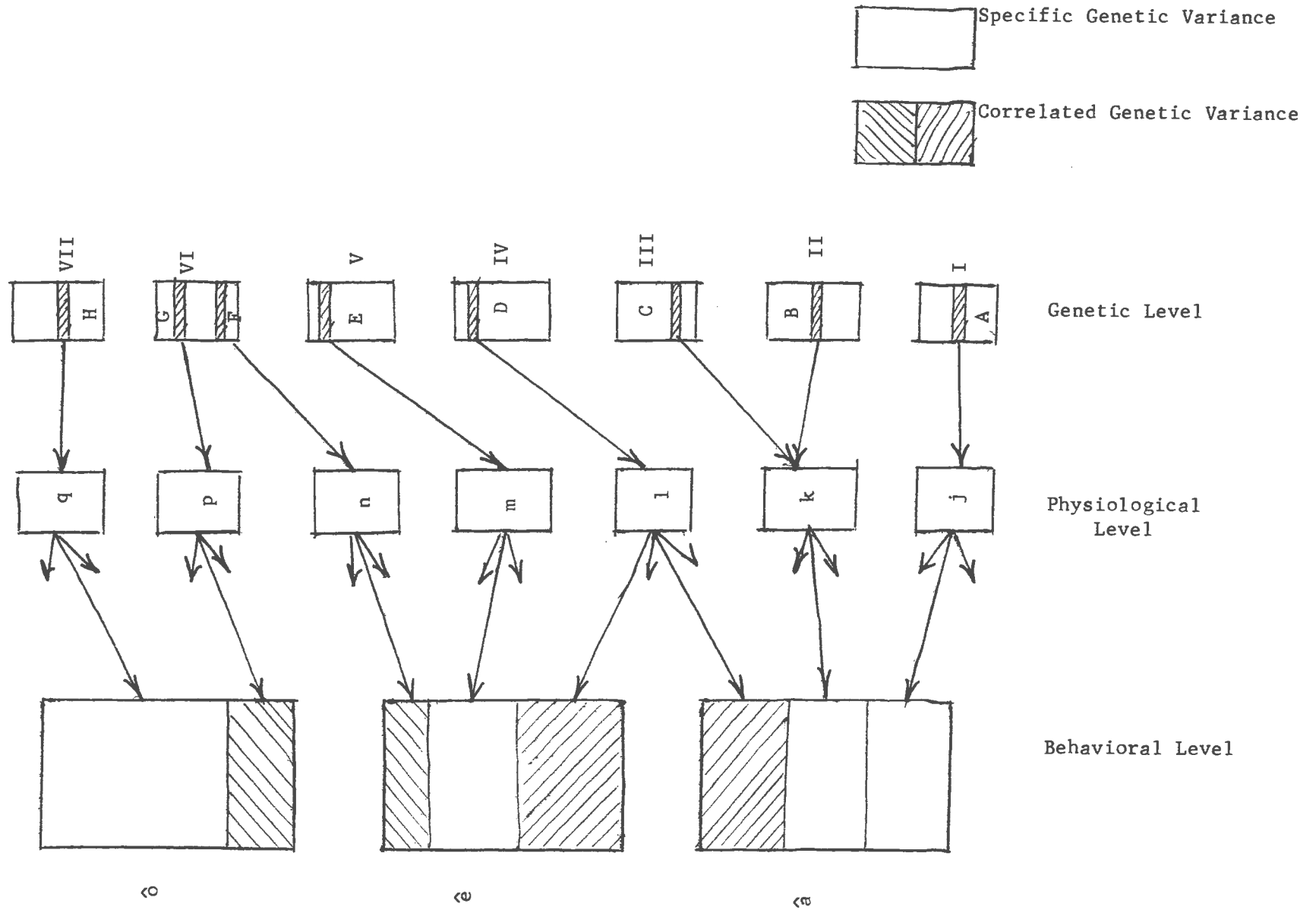


TABLE iv

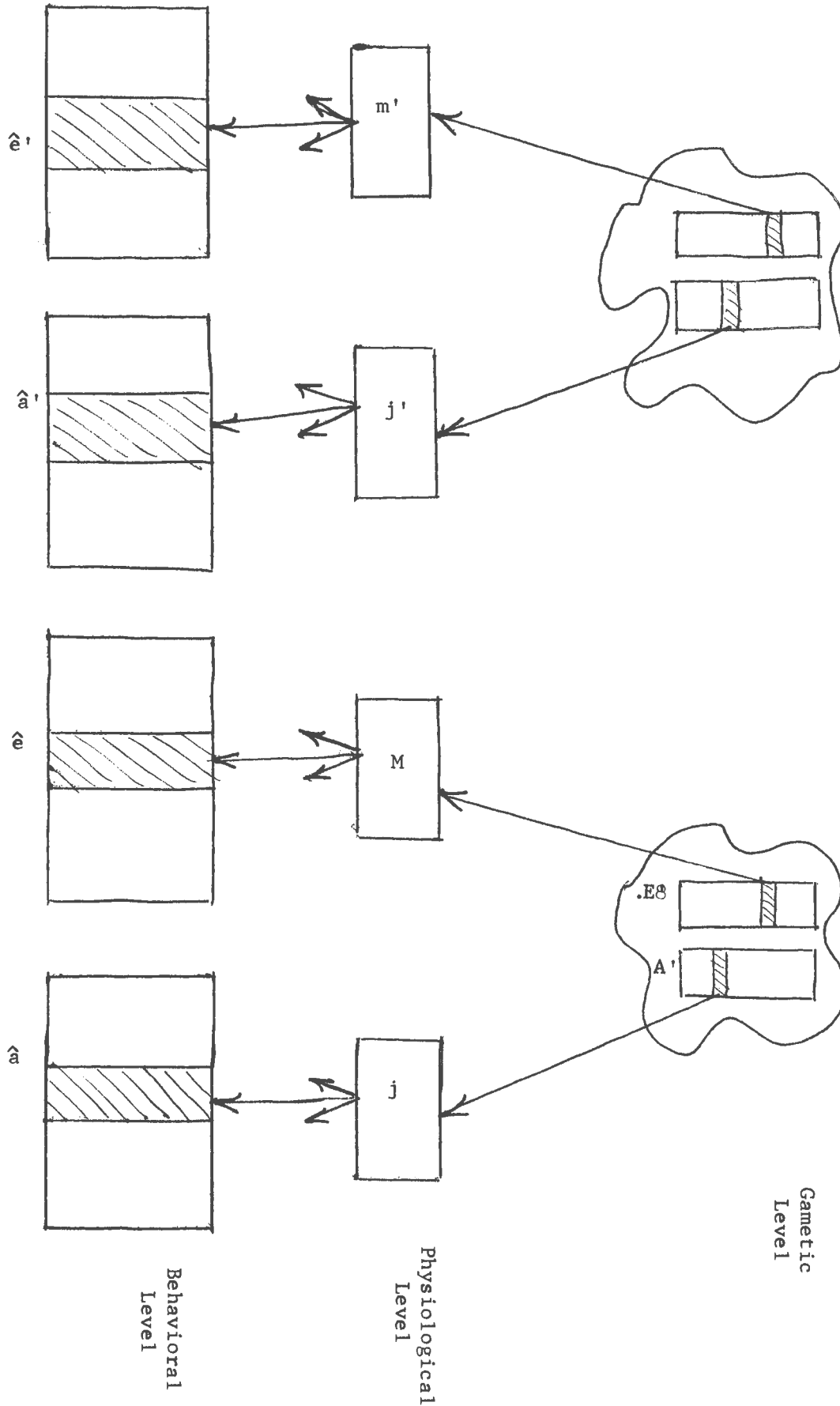
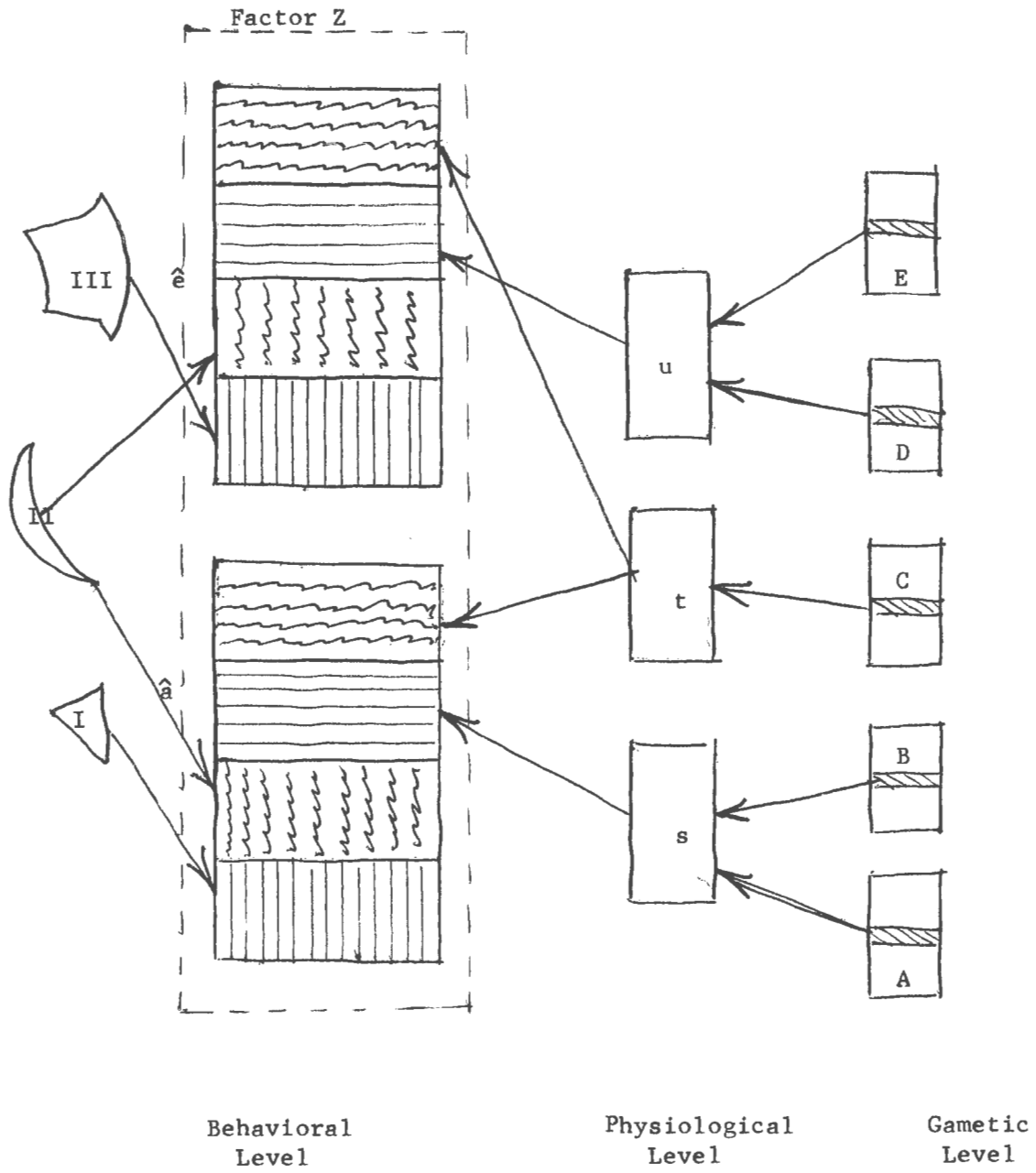


TABLE v



FOOTNOTES

- 1-Anastasi; Differential Psychology; pp.234-245
- 2-ibid.pp.203-219
- 3-Allen;"Genetic Aspects of Mental Disorder" from The Nature and Transmission .....; pp.324
- 4-van den Bosch; Microcephaly in the Netherlands
- 5-Slater; The Heredity of Behavior
- 6-ibid.;Slater
- 7-Burt;"The inheritance of mental ability"; The American Psychologist; ppl-15, Vol.13.
- 8-Ashley-Montagu
- 9-Anastasi, ibid.pp.162-174.
- 10-Osborn; Preface to Eugenics; pp.214-234.
- 11-Ginsburg; pamphlet- Genetics as a Tool in the Study of Behavior.
- 12-Williams; Biochemical Individuality
- 13-Beach; Hormones and Behavior; pp.165-176.
- 14-Williams; ibid.
- 15-Kety- Science; Vol. 129, pp.1528-1532.
- 16-Lashley- Psychological Review; Vol. 54, pp.325-334.
- 17-Anastasi; ibid.
- 18-Royce; "Factor Theory and Genetics" from Educational Psychological Measurement; Vol.19, pp.361-376.
- 19-Hull; Essentials of Behavior



## BIBLIOGRAPHY

- Allen, George; "Genetic Aspects of Mental Disorder" from The Nature and Transmission of the Genetic and Cultural Characteristics of Human Populations; New York; Milbank Foundation; 1957.
- Anastasi, Anatol; Differential Psychology; 3rd edition; New York Macmillan, 1958.
- Beach, F.A.; Hormones and Behavior; New York; Paul B. Hoeber; 1948.
- Burt, Carl; "The inheritance of mental ability"; The American Psychologist; Vol. 13; 1-15; 1958.
- Ginsburg, William V.; Genetics as a Tool in the Study of Behavior; a pamphlet; 1968.
- Hull, C.L.; Essentials of Behavior; New Haven; Yale University Press; 1951.
- Kety, S.S.; "Biochemical Theories of Schizophrenia"; Science; Vol.129; 1528-1532.;1959.
- Lashley, K.S.; "Structural Variation in the Nervous System in Relation to Behavior"; Psychological Review; Vol. 54; 325-334; February, 1947.
- Lerner, I.M.; The Genetic Basis of Selection; New York; Wiley Press; 1967.
- Osborn, Franklin; Preface to Eugenics; New York; Harper and Brothers; 1951.
- Royce, J.R.; "Factor Theory and Genetics"; Educational Psychological Measurement; Vol. 19, 361-376. June, 1967.

Stockard, C.R.; Genetic and Endocrinic Basis for Differences in  
Form and Behavior; Philadelphia; Philadelphia Wistar Institute  
Press; 1957, 1968 revision.

Williams, R.J.; Biochemical Individuality; New York; Wiley Press;  
1956.