Behavior Genetics and Individuality Understood

Behaviorism's counterfactual dogma blinded the behavioral sciences to the significance of meiosis.

Jerry Hirsch

Individual differences are no accident. They are generated by properties of organisms as fundamental to behavioral science and biology as thermodynamic properties are to physical science. Much research, however, fails to take them into account. The behavioral sciences have attempted to erect a superstructure without paying sufficient attention to its foundation. A uniformity of expression over individuals, and even across species, has too often been assumed for behaviors under study. The uniformity assumption is explicitly incorporated into a spate of mathematical models that have been developed to formalize the study of behavior: Bush and Mosteller (*1*), for example, built theirs for "organisms that can be considered 'identical' at the start of an experiment. . . ." Rosner (*2*) speaks of "a fundamental attitude" which keeps psychophysics (*3*) "oriented toward the sources of uniformity in behavior." In this article I consider some effects that such assumptions about heredity, individuality, and behavior have had on the behavioral sciences.

Three Approaches to Behavior

In the study of behavior, three points of view can be distinguished. (i) Only common properties of behavior are studied among individuals and species. (ii) Only common properties of behavior are studied among individuals, while both similarities and characteristic differences are studied among species. (iii) Similarities and differences are studied among individuals, populations, and species.

The first view prevails when an organism is used as a tool for studying behavioral correlates of stimulus conditions, reinforcement schedules, deprivation regimens, pharmacological agents, or physiological mechanisms. It is hopefully assumed that the form of any relation observed—for example, that between stimulus and response—will have universal generality. The organism's role is essentially that of an analyzer, like the role of the Geissler tube in physics. In their illuminating discussion "The misbehavior of organisms," the Brelands (*4*), drawing on over 14 years of faithful application of the methods and assumptions of behaviorism, show that behaviorism also assumes "that the animal comes to the laboratory as a virtual *tabula rasa,* that species differences are insignificant, and that all responses are about equally conditionable to all stimuli." They relate (*4*) a history of "egregious failures" which they feel "represent a clear and utter failure of conditioning theory."

From the second viewpoint the behavior of animals is as characteristic of their species as is their form. This view prevails in ethologically oriented studies —for example, studies of such instincts as reproductive, parental, or territorial behavior. All members of a species are assumed to manifest a given behavior pattern, in some typical way. In Mayr's cogent analysis (*5*) this represents typological thinking whose replacement "by population thinking is perhaps the greatest conceptual revolution . . . in biology."

The third approach characterizes behavior genetics: the study of the relations between the genetic architecture of a taxon and the distributions of its behavioral phenotypes. It employs the methods of both the behavioral sciences and genetics. The growth of this field can be attributed to protest against the counterfactual uniformity postulate, combined with the realization that we can now have a description and analysis of behavior based on a deeper understanding of the materials on which the behavioral sciences make their observations.

The key to our present understanding of the structure of life came during the first half of this century, from investigations of transmission cytogenetics (*6*) and population cytogenetics (*7*). Through study of cell division and reproduction (mitosis, meiosis, and fertilization), together with statistical analysis of variations in the expression of traits among offspring of specified matings, transmission cytogenetics gave us our first picture of the fundamental units of life (genes and chromosomes) and of the variation-generating probability mechanism (meiosis) by which lawfully combined random samples of these units are passed on from parents to offspring. Through study of (i) the distributions of genes in populations, (ii) the mechanisms responsible for both stability and change in gene frequencies, and (iii) the role of such mechanisms in evolution, population cytogenetics has given us some understanding of ensembles of these units that comprise the gene pools of populations and species—the taxa that are natural units of evolution.

Understanding Individuality

The phenotype (appearance, structure, physiology, and behavior) of any organism is determined by the interaction of environment with its genotype (the complete genetic endowment). Each genotype is the end product of many mechanisms which promote genotypic diversity in populations.

Ordinarily members of a cross-fertilizing, sexually reproducing species possess a diploid, or paired, set of chromosomes. Most species whose behavior we study are sexually dimorphic. The genetic basis of this dimorphism resides in the distribution of the heterosomes, a homologous pair of sex chromosomes (XX) being present in the mammalian female and an unequal pair (XY), in the mammalian male. Sexual dimor-

The author is professor of psychology at the University of Illinois, Urbana. This article derives partly from discussions presented in 1959 at the symposium "Roots of Behavior," program of the American Psychiatric Association at the Chicago meeting of AAAS; in 1960 at the American Orthopsychiatric Association symposium "Genetics of Mental Disease"; and in 1961 at a symposium, "Expanding Goals of Genetics in Psychiatry," of the Department of Medical Genetics, New York State Psychiatric Institute.

phism guarantees that any population will be variable to the extent of at least two classes. Whether sex-chromosome or other genotypic differences are involved in any particular behavior remains an empirical question to be investigated separately for every population. It can no longer be settled by dogmatic attitudes and assumptions about uniformity.

Chromosomes other than sex chromosomes are called autosomes. Every autosome is normally represented by a homologous pair whose members have identical genetic loci. Alternative forms of a gene any of which may occupy a given locus are termed alleles. If an individual receives identical alleles from both parents at homologous loci, he is said to be homozygous for that gene. If he receives two alleles that differ, however, he is said to be heterozygous for that gene. The process by which a gene changes from one allelic form to another is called mutation.

When a gene is represented in the population gene pool by two allelic forms, the population will be genotypically polymorphic to the extent of at least three classes. That is, individuals may be homozygous for either of two alleles or heterozygous for their combination.

Study of populations has revealed that often extensive series of alleles exist for a locus. Well-known examples are the three (actually more) alleles at the ABO-blood locus in man and a dozen or more alleles at the white-eye locus in *Drosophila*. Benzer (*8*), in his study of the internal genetic architecture of *one* "gene" with a corresponding physical structure of probably less than 2000 nucleotide pairs, the *r*II region of the T_4 bacteriophage, found 339 distinguishable mutational sites, and he expects to eventually find some 428. There is no reason to believe that we shall find less complexity in cellular organisms as further refinement increases the resolving power of our techniques for analyzing them. In general, for each locus having n alleles in the gene pool, a population will contain $n(n + 1)/2$ genotypic classes. Mutation insures variety in the gene itself.

Sexual reproduction involves meiosis —a complex cellular process resulting in a meristic division of the nucleus and formation of gametes (reproductive cells) having single genomes (a haploid chromosome set). One homolog in every chromosome pair in our diploid complement is of paternal origin and the other is of maternal origin. In meiosis, the homologs of a pair segregate and a gamete receives one from each pair. The assortment to gametes of the segregating homologs occurs independently for each pair. This process insures diversity because it maximizes the likelihood that gametes will receive unique genomes. For example, gametogenesis in *Drosophila willistoni* produces eight alternative gametic genomes, which, if we represent the three chromosome pairs of this species by Aa, Bb, and Cc, we designate ABC, ABc, AbC, aBC, Abc, aBc, abC, abc. In general, n pairs of chromosomes produce 2^n genomes (if we ignore the recombination of gene linkages that actually occurs in crossover exchanges between chromosomes). Man, with 23 chromosome pairs, produces gametes with any of 2^{23} alternative genomes. This makes vanishingly small the chances that even siblings (other than monozygotes) will be genetically identical. Since the gamete contributed by *each* parent is chosen from 2^{23} alternatives, the probability that the second offspring born to parents will have exactly the same genotype as their firstborn is $(\frac{1}{2}^{23})^2$, or less than 1 chance in over 70 trillion! The probability that two unrelated individuals will have the same genotype, then, is effectively zero (*9*).

The argument for the genotypic uniqueness of members of populations is even more compelling, since other conditions also contribute to diversity. So, it is clear, the organisms which the behavioral sciences study are intrinsically variable before they undergo differentiating experiences. The mechanisms responsible for this variety are mutation, recombination, and meiosis. Add to these individual experience, and it becomes evident why individuals differ in behavior. In fact, the more reliable our methods of observation become, the more evident will this variety be.

The Abnormality of the Normal

For Watson, its founder, behaviorism was "a natural science . . . [whose] closest scientific companion is physiology. . . . It is different from physiology only in the grouping of its problems, not in fundamentals or in central viewpoint" (*10*). Assumptions about the uniformity and normality of material under investigation are often made in physiology, the science after which, more than any other, experimental psychology has attempted to pattern itself. We may, therefore, get a better grasp of the individuality-uniformity distinction by examining the differences between organisms whose behavior is studied by behavioral scientists and systems whose functioning is studied by physiologists.

Since the two disciplines are working at distinctly different levels of biological organization, the meaning of "normality" as operationally determined by them is quite different. Physiologists choose a normal organism to work with—one that looks healthy and does not appear unusual—and study one or more of its systems, such as the adrenals, gonads, or other endocrines, or regions of the nervous system. Either pre- or postexperimentally, anatomical, histological, or biochemical verification is made of the normality of the material under study, and sometimes of related or adjacent functions to boot. In the behavioral sciences we choose normal-appearing organisms to study too. We rarely perform biopsies unless there is a specific physiological interest, in which case we operate as the physiologist does.

Physiological systems are variable, not uniform. Williams (*11*) amply documents this and points out that implicit in our use of "normal" is reference to some region of a distribution arbitrarily designated as not extreme—for example, the median 50 percent, 95 percent, or 99 percent. We choose such a region for every trait. Among n mathematically independent traits—for example, traits dependent on n different chromosomes—the probability that a randomly selected individual will be normal for all n traits is the value for the size of that region raised to the nth power. Where "normal" is the median 50 percent and $n = 10$, on the average only 1 individual out of 1024 will be normal (for ten traits). When we consider at one time the distributions throughout a population of large numbers of physiological systems, we should expect negative deviates from some distributions to combine with positive deviates from others, both kinds of extreme deviates to combine with centrally located ones, and deviates of similar algebraic sign and magnitude to combine. Each individual's particular balance of physiological endowments will be the developmental result of the genotype he draws in the lotteries of meiosis and the mating ritual. Because of crossing over, most genes assort independently. Hence, we cannot expect

high correlations among the systems they generate.

If, underlying every behavior, there were only a single such system—for example, if the male "sexual drive" were mainly dependent on the seminal vesicles (*12*) or if escape behavior were mainly dependent on the adrenals—then the same kind of distribution might be expected for both the behavior and the underlying system. Whatever uniformity might exist at one level would be reflected at the other. The last few decades of research on the biological correlates of behavior have made it increasingly clear that behavior is the integration of most of these systems rather than the expression of any one of them. Therefore, there is little reason to expect that the many possible combinations and integrations of those systems that go to make up the members of a population will yield a homogeneously normal distribution of responses for many behavioral measures. An organism richly endowed with the components of one subset of systems and poorly endowed with those of another is not to be expected to behave in the same manner as an organism with an entirely different balance of endowments. The obviousness of this fact is well illustrated by the differences in behavior among the various breeds of dogs and horses.

Reductionism

Another conviction, strongly held by some, is that *real* explanations must be reductionistic. Those who hold this view in its most extreme form assert that no behavior can be understood until its physical basis has been unraveled. And the search for the physical basis proceeds along physiological, biochemical, biophysical, or genetical lines, depending on the skills and predilections of the investigators.

In laboratory experiments, some rats learn mazes more readily on the basis of visual cues while others do better with predominantly kinesthetic cues (*13*). The kinds of differences in organization that can coexist as alternative forms within a species, as well as some relations between one behavior and the component subsystems that are alternative possibilities, have been further revealed in a series of studies of the effects of domestication. In some domesticated rats, activity in a revolving drum was controlled by the gonads: control rats had daily activity scores as high as 18,000 revolutions, while gonadectomized rats scored only a few hundred revolutions. Cortisone therapy restored a high activity level in the gonadectomized rats. When the same experiment was repeated on wild Norway rats, however, the presence or absence of gonads made no detectable difference in measured activity. Further study of differences between these domesticated and wild rats revealed larger adrenals in the wild rats and larger gonads in the domesticated (*14*). So it appears that activity may be under the control of adrenal output in one case and gonadal output in the other—that behavior is not a univocal index to an organism's balance of endowments. The fallacy of reductionism lies in assuming a one-one relation between different levels of organization. With degeneracy already demonstrated in the genetic code of messenger RNA base triplets for the amino acids of proteins, we should be surprised not to find it at the levels of complexity we are considering (*15*).

Behaviorism and Introspection

According to my naive picture, the pyramid of sciences forges links of knowledge "out" from the periodic table: on the one hand, "down" into atomic structure through advances in physics; on the other, "up" into life through the genetic code and organic structure by advances in biophysics and biochemistry. The place of the behavioral sciences in the outline of that pyramid has been clearly demarcated for some time (*16*). Our models and assumptions must be consistent with the knowledge that is burgeoning at other levels. This means doing our homework and learning (*17*) about developments in fields which may once have seemed remote from behavior, but which clearly are not. Unfortunately, we are still plagued by a legacy of pseudo-problems which, like MacArthur's old soldier, seem to be slowly fading away instead of discreetly dying.

Recently, in *Science* (*18*), immediately following Wilkin's exposition (*19*) of his magnificent work on nucleic acids that led to the Watson-Crick model, Skinner heeded a call to issue "a restatement of radical behaviorism. . . ." It may be recalled that behaviorism bears its title to call attention to the fact that it studies behavior objectively rather than mind subjectively. Under Watson, in 1913, it wished to distinguish itself from unreliable (?) introspectionist psychology, whose findings lacked intersubjective agreement. Under Skinner, 50 years after, it is still worried about "the dimensions of the things studied by psychology and the methods relevant to them."

Starting from the uniformity assumption, the introspectionists were attempting to study the generalized human mind by analyzing the contents of their own consciousness. Of course, the study of mind through analysis by different individuals of the contents of their consciousness inevitably revealed individual differences. Under a given set of stimulating conditions, different people reported different sensations. According to Boring (*20*) "there is always to be remembered that famous session of the Society of Experimental Psychologists in which Titchener, after hot debate with Holt, exclaimed: 'You can see that green is neither yellowish nor bluish!' and Holt replied: 'On the contrary, it is obvious that a green is that yellowish-blue which is just exactly as blue as it is yellow.' That impasse was an ominous portent. . . ."

In over 50 years no one has suggested that Titchener and Holt might *both* have been making reliable observations. The event Boring bemoans would not be looked upon as an "impasse" that represents "an ominous portent" by a behavioral science that understands the structure of the materials it studies. Until recently, some of our best information on the assignment of genes to human chromosomes came from introspective behavioral observation. We know that genes affecting red-green color discrimination are carried on the X chromosome. We know it because some people fail to report differences in sensation easily observed by others, and the determining factors are transmitted to sons by mothers but never by fathers. Furthermore, Graham (*21*) has made excellent use of the introspections of one individual whose two eyes receive different color sensations from the same stimulus. Wouldn't it have been of great interest to learn how colored stimuli appeared to other members of the Titchener and Holt families? How many more potentially fruitful leads have been lost in the behavioral sciences because of rigid adherence to the counterfactual uniformity assumption?

Now, what was really wrong with introspection? Is there any other method by which Penfield could have made the startling discovery that apparently

long-forgotten experiences remain stored in specific regions of our brain? He succeeded in restoring "lost" memories to introspectively observed consciousness by electrical stimulation of appropriate regions of exposed human brains (*22*). If the behaviorists had scrutinized the assumptions, which they shared with introspective psychology, they might not have been so quick to condemn its method. Every method has limitations, which it behooves its users to understand.

Behavior Genetics

There now exists a substantial and rapidly growing literature on the behavior genetics of many organisms, from *Drosophila* to man—what Tryon (*23*) calls "the basic science of individual differences." It comes from research far less hampered by unsound premises. In Fuller and Thompson's useful summary (*24*) we can see "its documentation of the fact that two individuals of superficially similar phenotype may be quite different genotypically and respond in completely different fashion when treated alike." This field, like others, is passing through stages.

The goal of the early work was a genetics *of* behavior. It took a while to learn that heritability is a property of populations and never of behaviors: the relation between behavioral variation and relevant genetic variation is never constant. It must be measured in specific populations under specific conditions, because it varies with both. Tolman (*24*), Tryon (*24*), and Heron (*24*) each measured individual differences in rats' ability to learn and then, by selective breeding, produced strains of "maze-bright" and "maze-dull" rats. Hall (*24*) and Broadhurst (*24*) selected for differences in emotional responses. Analogous studies have been made of performance on an animal "intelligence test" (*25*).

Many strains of small mammals (mice, hamsters, rats, guinea pigs, and rabbits) are maintained under varying inbreeding regimens for purposes of medical and other research. When different strains within a species are compared, it actually becomes a challenge *not* to find differences in one or more behaviors. When strain comparisons are followed by appropriate genetic crosses, genetic correlates of behavioral differences are demonstrated. Such experiments have been performed for a large variety of behaviors: alcohol preference (*26*), hoarding (*24*), mating competition (*24*), susceptibility to audiogenic seizure (*24*), exploratory tendency (*24*), and various learning measures (*24*).

Paralleling the animal research are studies of human pedigrees, studies of family resemblances, twin comparisons, population surveys, and studies of race differences. Again, heritabilities have been demonstrated for many behaviors; for example, nature-nurture ratios were computed for intelligence-test and personality-test performance. Kallmann and his associates have pioneered, and others have joined, in collecting an impressive body of evidence on genetic factors in schizophrenia and other psychopathologies (*27*).

In 1963, with the wisdom of hindsight, we can ask why so many demonstrations were necessary. Should it not have been common knowledge that within each population the variation pattern for most traits will be conditioned by the nature of the gene pool, and that this will differ among populations? The answer lies in one phrase: the heredity-environment controversy.

The "opinion leaders" (*28*) of two generations literally excommunicated heredity from the behavioral sciences. Understandably, they objected to amateurish labeling of behaviors as instincts without proper experimental analyses. Also, they were repelled by the pseudogenetics of Hitler and other purveyors of race prejudice (*29*). On the other hand, impressed with the power of conditioning *procedures*, they proclaimed their faith in analysis of experience as the starting point for behavioral science—as though experience, like the Cheshire Cat's grin, could exist without the organism. "Our conclusion . . . is that we have no real evidence for the inheritance of traits," said Watson (*10*). While acknowledging that there are heritable differences in form and structure, he claimed there is no evidence that those differences are related to function, because "hereditary structure lies ready to be shaped in a thousand different ways" (*30*). Behaviorism still makes the gratuitous uniformity assumption that all genetic combinations are equally plastic and respond in like fashion to environmental influences (*31*).

We are now in a more fruitful period. Experimental analysis is yielding information about genes and chromosomes and how they act. The way is open to understanding molecular—ultimately submolecular (*32*)—mechanisms and to following metabolic pathways between genes and phenotypes. In the honey bee, Rothenbuhler (*24*) found that resistance to foulbrood disease (a bacterial infection of the larvae) depends on homozygosity of the worker bees for recessive alleles of at least two genes: one which enables them to uncap compartments containing infected larvae and another which enables them to remove those larvae from the hive (*33*).

Médioni (*34*), in his studies of phototaxis (light-oriented locomotion) in *Drosophila*, employed genetic, physiological, and stimulus variables in an exquisitely detailed analysis articulating relations between components of behavior, components of the organism, and stimulus properties of the environment. Behaviorally, phototaxis is resolvable into five components: (i) a photopositive phase; (ii) a sensory adaptation factor [Viaud's *capacité photopathique* (*35*)]; (iii) an exploratory phase; (iv) a photokinetic factor; and (v) a photoinhibition phase. The interplay of the behavioral components depend on (i) the intensity and wavelength of light, (ii) the differential effects of stimulation through the ocelli and through the compound eyes, (iii) sex, and (iv) genetic background and geographical region of racial origin. Races in 17 regions of the Northern Hemisphere, from Japan across Eurasia to America, arrange themselves into two distinct North-South clines, an Eastern and a Western, in which light preference diminishes with latitude of origin.

Our laboratory has made the most detailed analysis, to date, of relations between the genome and a behavioral phenotype in studies of geotaxis (gravity-oriented locomotion) in *Drosophila*. Behavioral distributions for populations are obtained in the apparatus shown in Fig. 1. Selective breeding from a geotactically and genetically heterogeneous foundation population has produced the two strains shown in Fig. 2, which have diametrically opposite response tendencies. Other methods produced three populations differing with respect to both degree and kind of similarity in chromosome constitution among their members. Two parameters of their behavioral distributions were thus controlled. The least dispersion occurred in the population in which all members carried two of the three large chromosomes in identical form. The other two populations, differing from each other with respect

to the single chromosome distributed in identical form to all their members, differed in central tendency but not in dispersion, which was twice that of the first population. Figure 3 shows, for this model situation, the kind of prediction and control that an understanding of population structure and its genetic basis may yield.

Erlenmeyer-Kimling's subsequent chromosome analysis shows that genes influencing the response to gravity are distributed throughout the genome. The first two chromosomes in the unselected foundation population contribute to positive geotaxis, and the third to negative. Selection pressure both enhances and reduces their effects, depending on the direction of selection (*36*).

At the molecular level, an exciting development is the measurement, by Hydén and Egyházi (*37*), of changes, with learning, in the RNA base ratios in nuclei of specific mammalian nerve cells and in their glia. This work, if confirmed, represents a major advance in our search for the physical basis of experience. Hydén's speculative, but interesting, suggestion is that the electrical disturbance of the nerve impulse releases, in some as yet unspecified way, a repressed region of chromosomal DNA. This DNA henceforth produces, on demand, its characteristic RNA to code the protein that facilitates forward transmission of the particular temporal pattern of electrical frequencies that first released the DNA. This suggestion is the first to be made that appears capable of reconciling the universal feature of improvement with practice with the idiosyncratic features of individual performance. In this schema the individuality encoded in the chromosomal DNA of each genotype at meiosis and fertilization is propagated directly into the learning and memory mechanism by means of the established sequence of DNA producing RNA producing protein. Such a schema could thus accommodate the distributions of individual differences invariably found in studies of learning and memory.

The study of man is also moving beyond the stage of wondering whether we can find a heritability for this or that behavior. Phenylpyruvic oligophrenia, a form of mental deficiency accompanied by a high concentration of phenylpyruvic acid in the urine, had early been traced to a gene-controlled enzymatic deficiency in phenylalanine metabolism (*27*). Now, Down's syndrome (mongolism) has been associated with the presence of extra chromosomal material (*24*).

Human populations are dimorphic for taste sensitivity to certain bitter compounds. Different races show different distributions with respect to this trait, as well as to almost every other trait that has been genetically analyzed. On the basis of behavioral observations indicating that an individual's ability to taste certain compounds depends upon the presence of his own saliva, Cohen and Ogdon (*38*) suggested that components of the saliva might play a critical role in tasting ability. Lately, Fischer and his coworkers (*39*) have shown in vitro that the bitter-tasting thioureas are oxidized faster by the saliva of nontasters than by that of tasters. Presumably, at low concentrations so much of the compound is oxidized in a nontaster's mouth that the few molecules which might reach their receptor sites remain undetectable. Furthermore, Fischer and his associates have now confirmed Cohen and Ogdon's finding that, in order to taste certain compounds at all, even a taster requires the presence of his own saliva. Superficially at least, it appears that saliva, like many body tissues, cannot be transplanted. A valuable observation here would involve an exchange of saliva between identical twins, who are presumably alike in body chemistry.

The ramifications of the taster phenomenon appear to be legion. There is a significantly higher incidence of non-

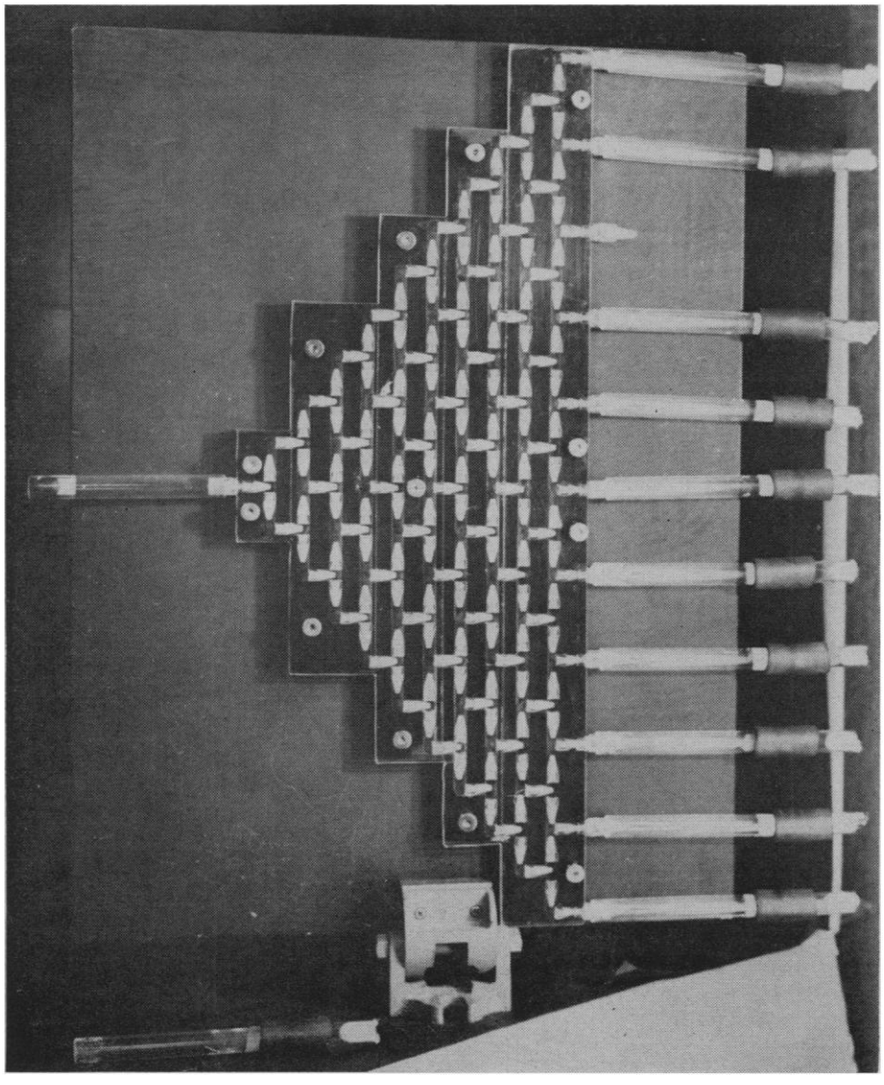

Fig. 1. Vertical ten-unit plastic maze facing a fluorescent tube. Squads of flies introduced in the vial at left are collected from the vials at right. They are attracted through the maze by the odor of food and by light. Small trap-like funnels, having a larger opening continuous with the alley surfaces and a small one debouching in midair, discourage backward movement in the maze. [Hirsch (*51*)]

tasting of the bitter compounds among persons with nodular goitre (*40*), among patients with congenital athyreotic cretinism, and among parents of the latter as well (*41*). In another study (*42*) it was found that, among 38 parents of children with Down's syndrome, none was able to taste quinine. Furthermore, all but one of the fathers in that sample were unable to taste a bitter thiourea. Finally, a correlation exists between taste sensitivity and dislike of foods: the more sensitive tasters find more foods objectionable (*43*).

Race Differences

A problem of continuing social importance, for an understanding of which most behavioral scientists have lacked a proper conceptual basis, is the question of race differences. To the liberals this question has been a continuing source of embarrassment (*44*). They have made little progress in answering it since the signing of our Constitution and Bill of Rights, when it was *asserted* that all men are created equal. To the prejudiced the question has presented no difficulties, because they *know* other races are inferior to their own; this seems as obvious to them as the flatness of the earth did to our ancestors.

This question appears in another perspective when it is examined in the light of current knowledge of population structure. Dobzhansky (*45*) has clearly called attention to the difference between equality and identity. Genotypic uniqueness creates biochemical individuality. Without enforcing conformity—irrespective of heredity, training, or ability—a democratic ethicosocial system offers to all equality of opportunity and equal treatment before the law. Genetics explains both individual and population uniqueness. Even though reproductively isolated populations belong to the same species and have the same genes, the relative frequencies of different alleles of genes in their gene pools are almost certain to differ. Mutations and recombinations will occur at different places, at different times, and with differing frequencies. Furthermore, selection pressures will also vary (*46*). In analyzing data from such populations we have learned to ask, not whether they are different, but, rather, in what ways they differ.

Races are populations that differ in gene frequencies. Observations on populations are summarized in distributions, so often assumed to be normal (*47*). When we add the assumption of common variance, or make transformations to obtain it, the data fit into the ever popular analysis-of-variance models. *The* difference between two populations must then be a difference between means, because the assumptions of normality and homogeneity of variance for the model leave no other property with respect to which the distributions can differ. The final step in this fantastic chain of reasoning has recently been taken in *Science* by Garrett (*48*). He ignores individual differences and claims that wherever two populations differ on some scale of measurement, no matter how vague, any individual from the population with *the* higher mean is better than any individual in the other population, and that intermarriage will "be not only dysgenic but socially disastrous"!

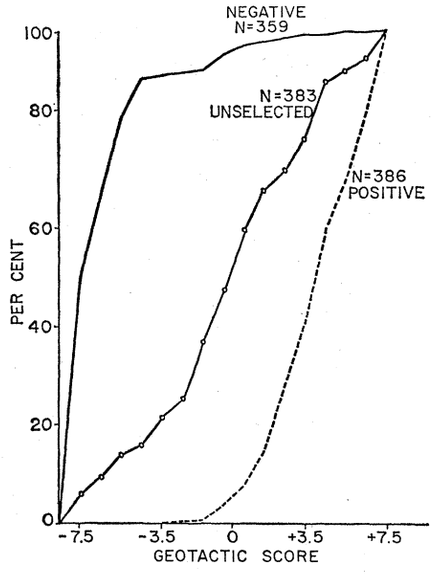

Fig. 2. Cumulated percentages of animals (males and females) that received geotactic scores in a 15-unit maze, from an unselected foundation population (middle curve) and from the two selected strains (outer curves). [Hirsch and Erlenmeyer-Kimling (*52*)]

Distributions have other properties, such as dispersion, skewness, and kurtosis (peakedness), and no single one is exclusively important. Where these other properties have been examined, the inadequacy of a preoccupation with the central tendency and a hasty assumption of normality has been easy to document (*49*). There is no reason to expect two populations with different heredities and different environments to have precisely the same distribution for any trait. We can expect to find varying combinations of similarities and differences in the several properties of distributions when we compare different populations for a given trait, or any set of populations for different traits. Furthermore, the number of traits for which we could make comparisons is effectively unlimited, and many of the traits will be uncorrelated (*11*). Again, a lack of intrinsic correlation would come as no surprise to a behavioral science that understands its materials, because traits are the devel-

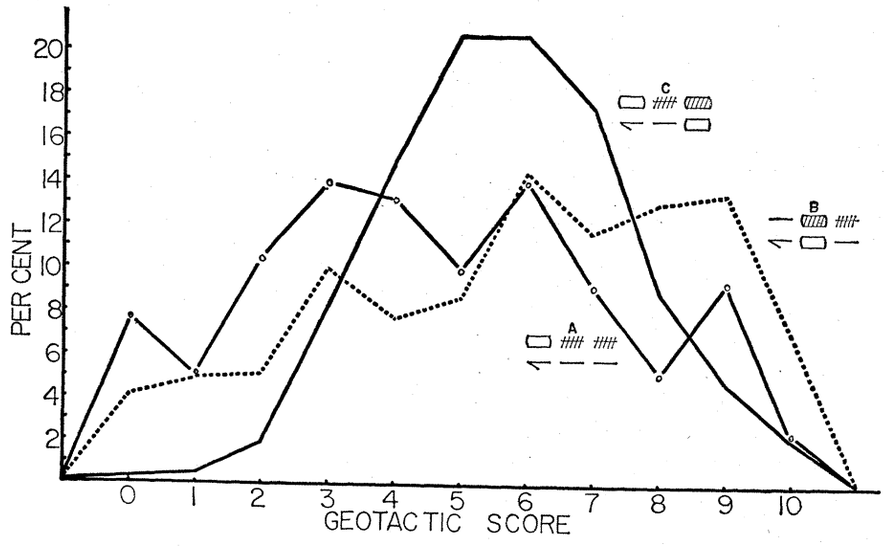

Fig. 3. Distributions of geotactic scores in a ten-unit maze for males of three populations (described in text). (Rectangles) Chromosomes carried in identical form by all members of a population; (dashes) chromosomes varying at random; (hatching) heterozygosity; (half-arrowhead) the Y chromosome of males. (In Fig. 2 the abscissa scale was reversed and the zero point was shifted to the center of the distribution.) [Hirsch (*51*)]

opmental result of thousands of genes, most of which, because of crossing over, sooner or later undergo independent assortment.

For ease of exposition, I have not considered environment in discussing race. Certainly, it is no less important than genetic endowment. The ontogeny of a responsible and effective citizen requires prolonged socialization, highly dependent upon the socializing agency. A genotype must have an environment in which to develop a phenotype. But the same genotype can produce quite different phenotypes, depending on the environments in which it may develop. Furthermore, a given environment can nurture quite different phenotypes, depending on the genotypes which may develop there. This fact is attested daily by parents and teachers who find that a method of tuition admirably successful with one child may be worthless with another, who nevertheless can learn by a different method. So, while environment makes an undeniably important contribution to the particular values obtained in phenotypic measurements, consideration of particular environments should not change our general picture of population structure. Without an appreciation of the genotypic structure of populations, the behavioral sciences have no basis for distinguishing individual differences that are attributable to differences in previous history from those that are not, and no basis for understanding any differences whatsoever where there is a common history.

Conclusions and Summary

Traditionally, many behavioral scientists have assumed that individuals start life uniformly alike, and that individual differences result only from differentiating experiences. To assume this is as contradictory to the established fact of uniqueness at conception as to assume that entropy is as likely to decrease as it is to increase. Recognition of the contradictory nature of this assumption does not make the role of experience in ontogeny any less important, but we now realize that the effects of experience are conditioned by the genotype. Therefore, a careful reconsideration of our statistical tools, experimental methods, theoretical models, and research goals is in order.

Many problems that have generated violent controversy now appear in totally different perspective. Introspection may provide a legitimate probe into subjective experience, without requiring intersubjective agreement. The concept of a normal individual has no generality. The outlook for understanding the physical basis of behavior has never been more promising. Awareness that a multiplicity of variable systems comprise its substrate, however, emphasizes the integrity and importance of the different levels of biosocial organization at which the several sciences work. In place of reductionism, we may now think of studying correlations between phenomena, reliably observed and analyzed at various levels, and of assessing the correlations over an ever-widening range of conditions. The controversial aspects of the heredity-environment question and of the race-differences question arise from failure to understand the genetics of individual and population differences and the rationale of their statistical analysis (*50*).

References and Notes

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