Selection and Eugenics

Curt Stern

Department of Zoology, University of California at Berkeley

WHEN CHARLES DARWIN pointed out the role that natural selection of certain genetic types has played in the evolution of animal and plant species, it was soon realized that modern man may be subjected to similar selective influences. As a result, two slightly different considerations were advanced, each complementary to the other. One dealt with the changes in natural selection that civilization has brought about. Darwin had stressed the ruthless struggle, in which the genetically less fit has a poorer chance of reproducing his kind than the more fit. Did not civilization reduce or eliminate this selection against the less fit by improving his chance of reproduction through charitable care, or artificial remedies? Had not an ominous situation been created which would lead to an increase in undesirable genetic constitutions? But to counteract this pessimistic view another possibility fired the imagination toward hopeful perspectives. Could not man take the genetic fate of the species into his own hands? Could he not be more efficient and successful than nature, and by the use of his knowledge improve the genetic qualities of future generations? Francis Galton coined the word *eugenics* to cover the whole "study of agencies under social control that may improve or impair the racial [meaning hereditary] qualities of future generations, either physically or mentally."

In accordance with its two aspects, the field of eugenics has often been subdivided into two branches, called negative and positive eugenics. The first is concerned with combating the increase or the presence of alleles or of genic combinations producing undesirable phenotypes, the second with furthering the increase of alleles or combinations causing desirable phenotypes, or at least guarding against the decrease of such genotypes.

The facts that eugenists use as a basis for the discussions are worthy of the most serious attention. While estimates of the frequencies of subnormal traits, physical or mental, are not very accurate, the total number of affected individuals in the United States amounts to many millions. Of course, only in some of these individuals are the subnormal conditions due to heredity causes, but the sum of genetically defective persons may well be much larger than one million.

It is customary in this connection to emphasize what it costs the public to care for defectives. Some decades ago, a number of studies were published of families who, in the course of generations, had contributed a large number of undesirable individuals to the population of the United States. The Jukes and the Kallikaks—literary names assigned to these families—became household words in the discussion of eugenic problems. The recurrence, generation after generation, of various types of criminality and of mental deficiency was taken as proof of the hereditary nature of these traits. It is now recognized that the methods used in gathering these family histories were highly uncritical and that these studies therefore give a distorted picture. Moreover, even if the data were unbiased no valid conclusions regarding the genetic component of the traits in these families can be drawn, since it is impossible to judge separately the parts played by genetic factors and by the very unfavorable environments which persisted generation after generation. As for the cost to society of these families, who instead of adding to its economy required support from it or were directly destructive to its assets, it was estimated as amounting over the years to millions of dollars. The significance of such sums, however, must be judged in terms of the total national expenditure. Although any unproductive expenditure is undesirable, the costs of caring for defectives are small relative to the sums involved in the whole economy.

It is often stated that physical defectives, and especially the "insane," are on the increase in Western nations. If this implies that the absolute numbers of such persons are increasing, this may well be so, since populations have also increased. A statement of this kind is meaningful only when it refers to the relative frequency of defectives in the population. When the facts are stated in relative terms it is indeed found that the relative number of patients *in institutions* has steadily increased, but the interpretation of these data is by no means obvious.

This rise may reflect a change in attitudes and in opportunities for social care. Whereas in former times the mentally ill were kept at home, they are now sent to hospitals. Better diagnosis and better methods of obtaining full reports also result in adding to the census of defectives. In Sweden, with its highly developed census system, 4,349 epileptic individuals had been registered in 1940. Medical examination of

all Swedish men reaching the age for conscription, however, placed the total number of epileptics at about 12,000, indicating that the census had unearthed less than 40 percent of them. Apparently the accuracy of the census data depended on the willingness of people to divulge relevant information to the authorities. Dahlberg, to whom we owe this example, concluded that "there is plenty of room for an increased frequency through improved registration, even if the actual frequency of hereditary epilepsy were to decrease appreciably" (*1*).

One more factor may be mentioned that enters into an interpretation of increased frequencies of certain defects. Many pathological conditions, among them certain types of mental derangement, as well as organic diseases like cancer and diabetes mellitus, tend to make their appearance in the later periods of life. Without the recent prolongation of man's average life span, many persons who now reach an older age and become affected formerly would not have lived to the necessary age and would not have swelled the number of recognized defectives.

TABLE 1

RATIO OF THE NET REPRODUCTIVE RATE FOR SPECIFIC URBAN CLASSES TO THE CORRESPONDING RATE FOR THE TOTAL URBAN WHITE POPULATION OF THE UNITED STATES, 1935–1936*

Education of mother	Reproductive ratio
College	0.74
High school	0.97
Seventh or eighth grade	1.23
Less than seventh grade	1.39

* After Karpinos and Kiser (*2*).

The greatest concern expressed by eugenists is that the intellectual genetic endowment of Western populations is in danger of decreasing. This fear is based on the fact of differential fertility. If, for instance, the population of the United States is subdivided into different categories, according to occupation, educational background, income, or in other ways, it is found that the average number of children per family is different for the different groups. The striking fact regarding the average reproduction of the different categories is that it decreases with increase in socioeconomic status, as shown in Table 1.

The differential fertilities of groups of different socioeconomic status are of no concern to the geneticist if the genetic endowments of the different layers of the population are alike—that is, by and large, if the same allele frequencies for all loci hold for the different groups. If, on the other hand, different layers differ in their corporate genetic make-up, then differential reproduction constitutes a selective agent.

We cannot say now with any certainty whether or not different socioeconomic groups are genetically differentiated. The difficulties of research in this important field are great. The concept of socioeconomic levels itself is subject to various definitions, involving occupation, social prestige, amount of income, education, etc., and a simple scale of levels does not represent actualities satisfactorily. These, however, are minor difficulties as compared to finding out whether different groups are or are not genetically alike. It might be not too difficult to obtain data on this problem as related to various physical traits. But the question is primarily concerned with mental traits, since these may possibly be correlated with the socioeconomic status of the individuals. Mental traits are expressed very differently, according to environmental conditions—in the widest sense of the term environment. Undoubtedly, a large part of the variability in mental traits among human beings of different levels is therefore attributable to differences connected with their being in different environments as represented by these levels. Psychological tests which measure mental differences are imperfect indicators of the genetic nature of such differences, since psychologists have not fully succeeded in devising tests that are equally intelligible to individuals who have grown up in different social surroundings. Even with these imperfections of the tests in mind, however, there is evidence that strongly suggests hereditary influences.

TABLE 2

AVERAGE I.Q. IN THE CHILDREN (18–54 MONTHS OLD) OF FATHERS OF DIFFERENT OCCUPATIONAL LEVELS IN THE UNITED STATES*

Occupation of fathers	Average I.Q.
Professional	125
Business, clerical	120
Skilled workers	113
Semiskilled workers	108
Unskilled workers	96

* After Goodenough (*4*).

Intelligence tests of the children of parents belonging to different socioeconomic levels show a rather consistent phenomenon, whether, for instance, based on studies made in the United States (Table 2), or in Soviet Russia (Fig. 1). The mean scores decline consistently from groups of higher levels to those of lower levels. How much of this decrease is environmentally conditioned is difficult to evaluate. Various lines of evidence, however, particularly studies on twins reared together as compared with others separated early in life, and on the intelligence scores of adopted children in relation to occupational status of the foster father and as compared with own children,

make it hard to avoid the conclusion that environment is not the sole agent and that there *are* mean differences in the genetic endowment of different socioeconomic groups.

At each level scores vary greatly, much more than do the mean scores of the different levels. The result

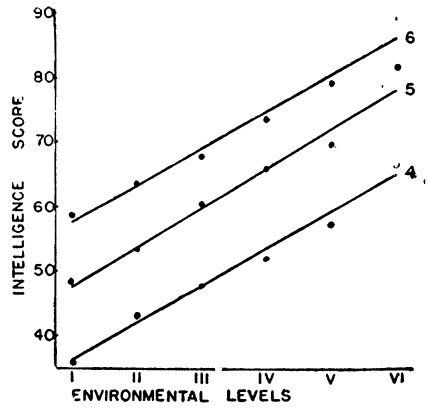

FIG. 1. Mean scores in intelligence tests administered in Charkow (USSR) to pupils in grades 4, 5, and 6, according to environmental levels as judged by parental education. Environment I applies to children of workers, one or both of whom were illiterate, VI children of officials who had university education (*5*).

of this intragroup variability is that an individual from a high level with a score at the average of his group will be surpassed by a great many individuals from a lower level; and, conversely, that an individual from a lower level with an average score for his group will be superior to many individuals of a higher level. The fact that there is a wide spread in I.Q. scores within each group mirrors to some extent the fact that different homes provide very different environments. Undoubtedly the spread in I.Q. scores is also owing to hereditary differences which express themselves in varying capabilities even within a single socioeconomic group.

Investigations restricted to the subnormal categories of intelligence point to the same conclusion. It was found in several studies that the relative frequency of feeble-minded children was considerably higher in the lower socioeconomic groups than in the upper ones. Thus the lower average I.Q. scores of the lower levels is in correlation with the greater frequency of *very* low I.Q. scores among the children. This correlation undoubtedly has an environmental component in that the restrictions of a lower socioeconomic status tend to relegate a child of low intellectual potentiality to the feeble-minded group whereas the opportunities provided by a higher status would tend to shift the same child into the range of better I.Q.'s. It seems unlikely, however, that such environmental factors are solely responsible for the high rate of feeble-minded children in the lower levels. It is more likely that the segregation of genetic constitutions involved in low I.Q. scores in the parents results not infrequently in genotypes among the children that place them in the range of feeble-mindedness. Probably, too, expression of genetic constitutions involved in low scores may vary from feeble-mindedness on up and the same genotype that in a parent permits somewhat higher intelligence may produce some feeble-minded children.

To reach the conclusion that there are probably genetic differences in intellectual endowment of the different socioeconomic levels is one thing, but to determine the specific type and magnitude of these differences is quite another. Undoubtedly, the differences are not absolute in the sense that any layer of a population is in the exclusive possession of alleles effective in the control of intelligence. There are no sharp boundaries between layers, since many individuals in each generation rise from a lower to a higher status, while others fall from a higher to a lower status. Some of these shifts may be explained by genetic segregation—of better genotypes in the lower levels and of poorer genotypes in the upper levels—but even if this interpretation should be true, there is a lag brought about by environment that keeps many with higher genetic endowment from rising and others with lower endowment from falling.

Whatever the genetic details and however important they may be for a complete insight, one fact is already apparent: If there are genetic differences between different socioeconomic layers, then differential fertility will result in selective increase of some allele frequencies, and decrease of others in the population as a whole. Since the differential fertility acts in favor of high reproduction of the intellectually more poorly endowed groups, and against high reproduction of the bettter endowed groups, a deterioration of the genetic endowment of the population should result.

The degree of this deterioration from one generation to the next could be determined only on the basis of exact data on the genetic basis of the intelligence scores and their nature-nurture interdependence. Lacking such data, attempts have been made to calculate expected phenotypic changes—that is, in the I.Q. scores—in successive generations. Using the observed mean scores in different socioeconomic levels and the observed reproductive values of these levels, various authors have arrived at estimates for the decrease in I.Q. for the population as a whole, from the present generation to the next. These estimates vary from about one to around five points. The calculations are based on many uncertainties and cannot be checked with observation, since no actual tests of successive generations have yet been made.

Historically, differential reproduction, on a large scale, of the type under discussion is a rather recent phenomenon. It is primarily the result of birth control, which became an important social practice in the second half of the 19th century. Since birth control measures are more frequently used by the upper and middle groups of Western countries than by the lower ones, the limitation of births becomes a selective process. There is reason to believe that the use of contraceptive measures will spread over the population as a whole and that the differential in the fertility of different groups will be diminished. From a eugenic point of view such a result is desirable.

There is a possibility that the differential fertility of the different groups is less significant even now than it appears. Some data indicate that the most successful members of the upper groups (success being estimated in various ways) are more fertile than the less successful ones. The reasons for this higher fertility may be related at least partially to the favorable financial status of these successful families, which permits them to bring up children under good conditions without undue strain. If the relatively high fertility of the upper subgroups within the higher levels should be a general phenomenon, and if success *within* a group is positively correlated with intellectual genetic endowment, then the higher fertility of the most successful would counteract, to an unknown degree, the low fertility of the group as a whole.

No studies have been made which give information on the fertility of different subgroups within the middle or lower groups. It does not seem unlikely that here the correlation between success and fertility is again negative. In the upper subgroups of the lower or middle groups, with their relatively small financial resources, the desire to provide for one's children the most favorable conditions may lead to particularly stringent birth limitation.

It is likely, then, that the present differential fertility of the different groups has a dysgenic effect in regard to intelligence. But the question may be raised whether there are desirable mental traits under gene control whose frequencies may be positively correlated with fertility. The upper layers of Western societies apparently have a relatively high frequency of genetic constitutions favoring intelligence, but they seem to be no better off than other strata in their frequency of alleles which lead to idiocy. Could there be still a third type of genetically controlled mental traits for which the upper groups are relatively deficient, but which constitute assets to the individual and to society? An answer to this question cannot be given. It would depend on many factors, of which only a few may be mentioned. The first difficulty arises in defining a desirable trait. Emotional stability might be desirable, but it is a trait that does not accompany many types of genius which enrich civilization. Altruism may be another desirable trait, but acquisitiveness and egocentric ambition have not produced misery exclusively but have also led to advances which have contributed to the welfare of the masses. It will be hard to agree on definitions of desirable traits—and it is clear that the ideal does not lie in uniformity.

Even if some agreement could be reached, a second difficulty is that measures of the genetic component of men's variability in regard to these traits are not available. The social plasticity of mankind is very great and different societies and groups within societies mold the attitudes of their members in most diverse ways. Many facts of psychology show that cooperativeness or aggressiveness can be produced by environmental influences acting on the same individuals, but these facts do not preclude the possibility that certain genetic components, yet unknown, may bring out one particular trait more readily than certain others.

There is some relevant evidence from two genetically different strains of laboratory mice. Under certain conditions, the males of one strain react peaceably to a strange mouse and the males of the other strain are highly aggressive. Yet within a few days specific training can transform both types of mice into either peaceful or fiercely fighting individuals.

We have stressed the tentative nature, and the frequent absence, of knowledge regarding the genetic basis of differences among human beings. Even if the information were more complete we would still have to make a detailed investigation of the effects selective agents would have on the genetic and phenotypic composition of later generations, before we could fully understand the dynamics of populations. A few theoretical situations will be discussed in which selection for or against various genotypes is active.

The simplest situation is that of selection against a single factor, autosomal dominant genotype *DD* or *Dd,* or conversely, selection for a recessive genotype *dd.* Practically all persons carrying a rare dominant allele are heterozygous, so that we may restrict our discussion to a population consisting of only *Dd* and *dd* individuals. If the dominant allele is fully penetrant and causes its phenotypic effect to appear before the reproductive age has been reached, then suppression of reproduction of all affected individuals will lead to elimination of the dominant condition from the next generation (Fig. 2, dotted line) except for new mutations from the recessive to the dominant allele. If, on the other hand, selection, in terms of suppression of reproduction of *Dd* individuals, acts only on some

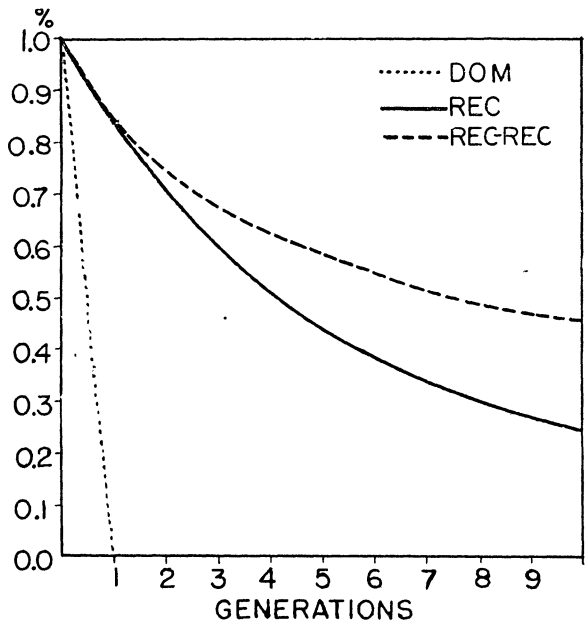

FIG. 2

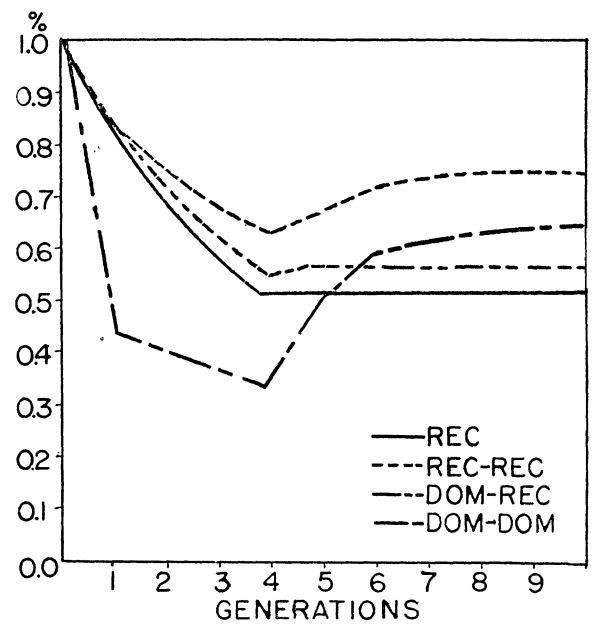

FIG. 3

FIG. 2. Complete selection for ten successive generations against a rare dominant genotype (Dom), a recessive (Rec), and a double homozygote (Rec-Rec). Initial frequency of the selected genotypes 1% (*3*).

FIG. 3. Complete selection against certain genotypes for four successive generations and cessation of selection during the following six generations. Rec = homozygous recessives; Rec-Rec = double homozygotes (e.g. $A^2A^2B^2B^2$); Dom-Rec = dominant phenotype in regard to one pair of alleles, recessive in regard to a second pair (e.g. *AAbb* and *Aabb*); Dom-Dom = dominant phenotype in regard to two pairs (e.g. *AABB, AaBB, AABb* and *AaBb*) (*3*).

and not on all these individuals, either because penetrance is incomplete or for other reasons, then a fraction of affected persons will reappear in successive generations. If, for instance, half of the carriers of a dominant allele are selected against, while the other half reproduce at the rate of the genetically normal population, the number of carrier individuals will be reduced to one-half in the first generation following selection, to one-quarter in the second, and in general to $(\frac{1}{2})^n$ of the original number where n equals the number of generations.

Complete selection against a single dominant factor is thus 100 percent effective in a single generation, and even partial selection accomplishes much. For example, with a selection factor of one-half, the number of dominants is reduced to a little more than one-tenth of the original number in three generations, and has practically disappeared in ten generations. If, for instance, all dominant chondrodystrophic dwarfs, or all individuals with dominant juvenile cataract would not reproduce, then the unhappiness caused by the birth of affected individuals in these families would be completely eliminated in one generation. For dominantly inherited diseases like Huntington's chorea, which often sets in after the reproductive age has begun, the reproduction of the disease through cessation of procreation by phenotypically affected persons will follow the experimental decrease.

Even with incomplete penetrance of a dominant allele, a complete elimination in one generation could be accomplished if *all* children who had an affected parent remained childless, regardless of whether they were phenotypically healthy or affected. This would involve not only the *Dd* individuals who might later become diseased, but also their *dd* sibs who are genetically normal. Such a situation is fraught with tragedy. A person who knows that he is the carrier of a genotype that leads to a very serious disease later in life will undoubtedly not wish to risk the chance of producing potentially affected children, but the personal sacrifice in remaining childless will appear very heavy if the potential parent finds out later in life that he was free from the dreaded allele. A means of distinguishing between *Dd* and *dd* individuals when both are still normal would be of great benefit to such people.

Selection against a single-factor, homozygous, autosomal genotype *dd*, or conversely, selection for a dominant *DD* or *Dd*, is less effective than the type of selection just discussed. The decrease of *dd* individuals from one generation to the next in a population in which all *dd* adults have been excluded from procreation can be calculated on the basis of the known frequencies of the alleles *D* and *d*. If the two frequencies are p and q respectively ($p+q=1$), then the frequencies of the three genotypes *DD, Dd,* and *dd* in

the original population are p^2, $2pq$, and q^2. After complete selection against *dd*, new *dd* can be produced only in $Dd \times Dd$ unions. The frequency of *Dd* in the total population is $2pq$. Since under complete selection against *dd* only *DD* (p^2) and *Dd* ($2pq$) contribute to the next generation, the relevant frequency of *Dd* among the *fertile* population is

$$\frac{2pq}{p^2+2pq} = \frac{2q}{p+2q}$$

which, because $p = 1 - q$, becomes

$$\frac{2q}{1+q}.$$

Therefore the frequency of *dd* in the new generation as a result of marriages of $Dd \times Dd$, amounts to

$$(q_1)^2 = \frac{1}{4} \cdot \left(\frac{2q}{1+q}\right)^2 = \frac{q^2}{(1+q)^2} \qquad (1)$$

The significance of the formula becomes apparent if some specific values for the initial frequency, q^2, of *dd* individuals are used. If, for instance, this frequency is 1 percent then, after one generation of complete selection against the affected individuals $(q_1)^2$ amounts to 0.83 percent. If the initial frequency is 0.83 percent, selection in one generation will reduce it to 0.69 percent; if the initial frequency is 0.01 percent the reduction will lead to 0.009803 percent.

These figures show two main facts: (1) that the lowering of the frequency is only a fraction of any initial frequency and (2) that the relative efficiency of selection against recessives lessens with a decrease of the initial frequency. The second point is well illustrated by a comparison between the first and last examples. The reduction from 1 to 0.83 percent represents a lowering of the initial frequency of *dd* by 17 percent, while the reduction from 0.01 to 0.009803 percent represents a lowering by only 2 percent.

This decrease in the effectiveness of selection against recessives with a lowering of the initial frequency is of great significance if one considers the results expected from selection continued over many successive generations. In Fig. 2 (continuous line) the results of selection over ten generations are given, starting with an initial frequency of 1 percent. It can be seen that the reduction in frequency of *dd* becomes less in each successive generation and that, after ten consecutive generations of total selection, it is still nearly one-quarter of the initial frequency of 1 percent. To reduce it to one-tenth, that is 0.1 percent, would require 22 generations. If the selection against recessives is not complete but reduces the average reproduction of *dd* individuals to some fraction of the normal rate, then obviously the effect of the selective process is even less.

Selection would be more effective if it included not only the homozygotes themselves but also certain of their close relatives who are likely to be heterozygotes. The immediate effect, however, would be very small, since in the case of rare alleles most marriages of heterozygous persons are with homozygous normal ones. It is true, nevertheless, that for any two heterozygotes who do not reproduce, two recessive alleles are eliminated, and this elimination, on the average, is equivalent to the nonappearance at some future time of one affected person.

The foregoing analyses of the effect of selection against simple dominant or recessive traits apply primarily to specific abnormal traits. From a general point of view, the significance of these traits is small if compared to such traits as intelligence, for which a whole series of expressions exists and which are rightly of greater concern to the student of human genetics and to the sociologist. These quantitatively varying traits, insofar as their variation is genetically conditioned, are the results of expression of *multifactor genotypes*. If, for instance, a trait is controlled by two pairs of genes A^1, A^2 and B^1, B^2, then nine different genotypes may be present in the population: $A^1A^1B^1B^1$, $A^1A^2B^1B^1$, $A^1A^1B^1B^2$, $A^2A^2B^1B^1$, $A^1A^1B^2B^2$, $A^1A^2B^1B^2$, $A^2A^2B^1B^2$, $A^1A^2B^2B^2$, and $A^2A^2B^2B^2$. If $A^1A^1B^1B^1$ and $A^2A^2B^2B^2$ are phenotypical extremes—for example, if they represent the lowest and highest endowment in the population—and if every substitution of A^2 for A^1, or of B^2 for B^1 increases the endowment to a certain degree, then nine different degrees of endowment will exist.

The results of selection against one or more of these multifactor phenotypes are different from those against single-factor genotypes. This may be seen in Fig. 2 (broken line), which shows the decrease of $A^2A^2B^2B^2$ individuals during ten consecutive generations of complete selection against this type. From an assumed initial frequency of 1 percent the frequency drops after one generation to 0.83 percent—just as in selection against a single-factor type *dd*. However, in later generations the decrease of $A^2A^2B^2B^2$ is much less than of *dd*. After ten generations the $A^2A^2B^2B^2$ class still recurs in 45.5 percent of its initial frequency, in contrast to 25 percent of the *dd* class. The slower progress in the transformation of a population in case of multifactor as opposed to single-factor inheritance is owing to the fact that the populations contain different frequencies of carrier individuals. In single-factor inheritance only the heterozygous *Dd* individuals form a reservoir from which a *dd* may be reconstituted. In two-factor inheritance against $A^2A^2B^2B^2$ *all* genotypes except $A^1A^1B^1B^1$ are carriers for one or the other, or for both of the alleles A^2 and B^2 against whom selection is directed. In a population which contains 1 percent *dd*, 18 percent are *Dd* carriers; but in a population

which contains 1 percent $A^2A^2B^2B^2$, 77.1 percent are $A^1A^2B^1B^1$, $A^1A^1B^1B^2$, $A^2A^2B^1B^1$, $A^1A^1B^2B^2$, $A^1A^2B^1B^2$, $A^2A^2B^1B^2$, or $A^1A^2B^2B^2$.

In single-factor inheritance with random mating, a change in an allelic frequency as a result of selection immediately leads to the establishment of a new constant distribution of the three genotypes *DD, Dd,* and *dd*. This is an expression of the Hardy-Weinberg rule, $p^2\ DD : 2\ pq\ Dd : q^2\ dd$. This immediate establishment of an equilibrium implies that a lower frequency of *dd* attained after selection will remain at its level after selection ceases. It was Weinberg himself, however, who realized soon after his discovery of the situation relating to one gene pair, that for multiple pairs, if the equilibrium were disturbed, a new equilibrium would be established only gradually. This may be shown for the $A^2A^2B^2B^2$ class. After one generation of selection this class is reduced from an initial frequency of 1 percent to 0.83 percent. If selection is discontinued, reconstitution of the class from the large reservoir of carrier individuals leads to a new rise, which gradually brings its frequency up to 0.91 percent—that is, to a recovery of nearly one-half of the originally lost frequency.

This "comeback" phenomenon in two-factor pair inheritance is obviously not restricted to cases in which selection ceased after one generation. Fig. 3 shows its existence and extent for some other examples, giving the changes in the frequencies of various phenotypes after complete selection had acted for four generations and then ceased.

Multifactor inheritance is usually based on many more than two pairs of factors. The foregoing discussions, modified, apply also to inheritance involving three or more pairs. Since in a population the frequency of individuals who are carriers for at least one of the alleles concerned in selection increases with the number of loci, the speed with which selection permanently accomplishes specific results decreases with increasing number of loci.

The numerical data presented for selected cases of multifactor inheritance are examples of the kind of information needed for a detailed understanding of the effect of differential fertility in man upon the phenotypic and genotypic composition of later generations. The model examples used in our discussion cannot be regarded as representing adequately the still unknown genetic situation in respect to such traits as performance in intelligence tests, or other genetic components believed to participate in the variability that places different individuals into different socio-economic layers. It already seems possible, however, to say that genetic changes brought about by selective agents are small from one generation to the next, and that the effectiveness of selection cannot be judged solely from a consideration of immediate phenotypic changes.

The effectiveness of selection has been discussed for populations which at the beginning of the selective processes were in equilibrium. Specific alleles or combinations of alleles, however, frequently occur in relatively high concentration in "pockets" of the population. The efficiency of selection is *increased* if there is a relatively high concentration of alleles in isolates, or more than random frequencies of homozygotes or of selected multifactor combinations.

In another respect the effectiveness of selection may often be *lower* than predicted at first. If an inherited trait has a certain frequency in a random mating population it must be ascertained whether the appearance of the trait is due to alleles at the same locus in each affected individual or whether it may be caused by different loci. If the trait is the result of two or more different genotypes based on different loci, then the effect of selection has to be considered separately for each genotype.

This may be shown by an example. Assume a population in which 1 in 10,000 individuals is blind for genetic reasons—that is, a frequency of 0.0001. If the blindness of all individuals were caused by the same homozygous recessive allele, then one generation of selection against the trait, according to equation 1, would reduce its incidence to 0.00009803. On the other hand, if there were ten different, and equally numerous genotypes, each of which produces blindness, then the initial frequency of any one of them would be one-tenth of 0.0001 or 0.00001. Selection, for one generation against each homozygous genotype, would reduce it to 0.000009937, so that the frequency of the sum of all individuals affected with any one of the ten genotypes is 0.00009937. Selection against the trait would thus lead to a reduction of only 0.63 percent of the original frequency as compared to 1.97 percent when a single genotype is involved. This hypothetical example is probably representative of many actual situations, since many results suggest that similar or apparently identical hereditary phenotypes are the result of diverse genetic constitutions.

The slowness of selective processes against recessive single factors and against multiple factors is both a blessing and a curse. It is a fortunate feature in that it forms a powerful buffer against sizable, undesirable effects of selection, as in differential fertility directed against intellectually well-endowed groups. It is an unfortunate feature in that selective measures aimed at eliminating undesirable traits are rendered relatively ineffective. In either respect the population at large serves as a huge reservoir for alleles from which

the desirable and undesirable genotypes can be reconstituted.

If the hopes and fears of the eugenic movement seem greatly exaggerated in the light of a numerical treatment of the problems, it should not be forgotten that the idealism which concerns itself with the genetic fate of future generations has a sound core. To say that the loss of supposedly desirable genotypes in one or even many generations of differential fertility is small does not remove the fact that it *is* a loss, which may be regrettable and possibly even have serious consequences. Although reproductive selection against severe physical and mental abnormalities will reduce the number of the affected from one generation to the next by only a few percent, nevertheless these few percent may mean tens of thousands of unfortunate individuals who should not be born. Conversely, even a slight increase of desirable genotypes, through positive eugenic measures, would be a social gain.

Eugenic and dysgenic selection are concerned with genetic constitutions which under present physical, mental, and social circumstances may lead to desirable or undesirable phenotypes. Since many genotypes express themselves differently under different environmental conditions it is possible that new kinds of environments can make genotypes which now lead to undesirable phenotypes shift their developmental reactions so as to result in desirable phenotypes. Thus, if a certain genotype formerly caused the serious disease diabetes mellitus, by way of a deficiency in internally produced insulin, the same genotype can now be made compatible with nearly normal living, by means of injection with insulin. Or it may be assumed that one genotype gives its carriers, in their specific educational and social environment, a lower I.Q. than another genotype gives to other individuals in their different environment. In spite of these circumstances, it might be possible to bring the carriers of the "lower" genotype to the same high achievement or even higher than the others, by means of a particularly appropriate environment.

Does substitution of a special environment to make up for deficient gene function necessarily lead to a weakened constitution of mankind? This question is frequently asked, and if it means that man may become more dependent on his environment than he was when all deficient genotypes were wiped out by natural selection the answer is yes, as far as the specific trait is concerned. Such dependence on special environments, however, did not start with the advent of civilization. When, in earliest evolutionary times, animals first developed, a new dependence of organisms on the environment arose. Instead of being able to synthesize their protoplasm from inorganic sources, animals had to rely on other organisms for food. When, much later, man's ancestors lost most of their mammalian body hair another, but this time minor, step was taken. Man had to rely on fur from other mammals and on fire to keep his temperature at the necessary physiological level.

In no instance, however, has man's greater dependence on specific outside sources for his survival been equivalent to degeneration. On the contrary, the dependence has often resulted in greater freedom from the restrictions of the external world. The loss of ability to use inorganic material for food became correlated with the evolution of nervous systems and sense organs, which make possible the many autonomous adjustments of animals. The dependence on clothing and fire enabled man to occupy regions of the globe where he could not have survived earlier. The passing of the primitive stages, in which each man was to a large extent independent of the help of others, gave rise to the complex interdependence of men in modern civilization. This new dependence has released man from the physical and mental starvation of earlier times. It is true, however, that man's freedom from his many limitations can persist and grow only if he retains and extends his slowly acquired control over his environment and over himself.

Human genetics concerns our own as well as future generations. Genetic counseling is largely devoted to individual problems, but the social implications of specific advice usually have not been disregarded. Eugenic thinking has always emphasized the well-being of mankind, even though much eugenic counseling was based on inadequate knowledge and has been harmful. In the future more knowledge will be gathered and will aid wise planning. Then genetic and eugenic counseling will become the foundation of human genetic engineering. Although eugenic problems are not as urgent as the pessimists believed, their ultimate importance can hardly be overestimated.

References

1. DAHLBERG, G. *Mathematical methods for population genetics.* New York: Interscience, 1948. Pp. 1–182.
2. DORN, H. F. *Milbank Mem. Fund Quart.*, 1947, **25**, 359.
3. KOLLER, S. *Z. Konstitutl.*, 1935, **19**, 253.
4. OSBORN, F. *Preface to eugenics.* New York: Harper, 1940, 312.
5. SIRKIN, M. *Sch. & Soc.*, 1929, **30**, 304.

This article is a condensed version of the chapter on selection and eugenics in Dr. Stern's forthcoming book, Principles of human genetics, *to be published by W. H. Freeman and Company in September.*