*The genetic assimilation of adaptive characters*

Many authors have argued that truly exogenous adaptations are produced by selection for the capacity to respond developmentally to the environment in an appropriate way. Baur has already been cited to this effect. A much earlier author, Douglas Spalding, to whom Haldane has recently drawn attention, had thought along similar lines fifty years before. In a lecture given in 1872 (see Haldane 1954) he made the following amusing suggestion: 'Suppose a Robinson Crusoe to take, soon after his landing, a couple of parrots, and to teach them to say in very good English, "How do you do, sir?"—that the young of these birds are also taught by Mr. Crusoe and their parents to say, "How do you do, sir?"—and that Mr. Crusoe, having little else to do, sets



to work to prove the doctrine of Inherited Association by direct experiment. He continues his teaching, and every year breeds from the birds of the last and previous years that say "How do you do, sir?" most frequently and with the best accent. After a sufficient number of generations his young parrots, continually hearing their parents and a hundred other birds saying "How do you do, sir?" begin to repeat these words so soon that an experiment is needed to decide whether it is by instinct or imitation; and perhaps it is part of both. Eventually, however, the instinct is established. And though now Mr. Crusoe dies, and leaves no record of his work, the instinct will not die, not for a long time at least; and if the parrots themselves have acquired a taste for good English the best speakers will be sexually selected, and the instinct will certainly endure to astonish and perplex mankind, though in truth we may as well wonder at the crowing of the cock or the song of the skylark.'*

Neither Spalding nor Baur, however, find any good reason why selection should have gone so much further than would appear to be necessary, and genetically fixed a character which, it might seem, could have been directly produced in each generation. They suppose that such an evolutionary step must have required very many generations; but mere lengthening of the time does not really help. In its simplest form the problem is this. We have a stock of animals $P$ living in environment $A$. If some of them are caused to develop in environment $B$, they become modified so as to be adapted to their new situation; we might call these $P_b$ animals, and their progeny if brought back in $A$, will develop exactly like the original $P$ stock. But if a local race evolves in environment $B$ it will become of a type $P'_b$, which, ideally, are indistinguishable from $P_b$ except for the fact that if their progeny develop in $A$ they will not be identical with $P$ but will show some traces of the $P_b$ characteristics. What agent has operated to cause this genetic fixation of the $P_b$ features?

In my opinion, the solution of this problem can only be found in the context of developmental canalisation, an idea which was

* For a modern discussion of animal behaviour in relation to evolutionary processes of the kind with which we are here concerned, see Spurway (1955).

not known to the earlier authors. The phenomenon of pseudo-exogenous adaptation is esentially one in which the genetic constitution imposes a limitation on the degree to which the phenotype responds to environmental change. The race adapted to environment *B* fails to become completely altered back to the original form when taken back to environment *A*. Its development has been to some extent canalised towards the phenotype appropriate to *B*.

The question is, how has this canalisation been brought about? There are several possibilities. In the first place, a gene mutation may have occurred, by chance, which determines a canalised development suitable for *B*. This is the hypothesis known as 'organic selection', or the 'Baldwin effect'. It was advanced by Baldwin, Lloyd Morgan and others in the early years of the century. Simpson (1953), who recently devoted an article to a discussion of it, describes the Baldwin Effect as taking place in three stages, which put very shortly are: (1) Individual organisms interact with the environment in such a way as to produce non-hereditary adaptations; (2) Genetic factors producing similar traits occur in the population; (3) These factors increase in frequency under natural selection. The gap in the argument is between steps (1) and (2). Is there supposed to be any connection between the developmental adaptations and the genes with similar effect, and if so, what? Simpson says that either there is no particular connection, in which case the theory signifies very little, or the connection must be by way of a neo-Lamarckian causal connection. Huxley (1942) seems to put the point originally made by Baldwin and Lloyd Morgan more clearly when he writes that the adaptive modifications operate 'by holding the strain in an environment where mutations tending in the same direction will be selected'. Thus according to both Simpson and Huxley, the theory of 'organic selection' still leaves the actual nature of the adaptive changes produced to the operation of either random mutations or Lamarckism.

A similar reliance on random mutations, occurring independently of the environmental stimulus, is implicit in the work which was carried out by a group of Russian authors between



1936 and 1944. Gause, who was one of the most important of them, has described these activities in a very interesting paper, published in English in 1947. He points out that the Baldwin-Morgan idea of organic selection, appearing at a time when Mendelism had only just been rediscovered, and when most biologists believed in the inheritance of acquired characters, rapidly fell into oblivion. The same principle was, it seems, independently arrived at in Russia by Lukin in 1936; and Gause and his fellow workers carried out a number of experiments inspired by it, which were published in 1941 and later.

The organisms studied were *Paramecium* and *Euplotes*. If vegetatively propagated clones of these Protozoa are cultured at different temperatures or in media of different salinities, any changes in shape or other properties that occur must be direct exogenous adaptations, since all the organisms will have the same genetic constitution and no natural selection can occur. If, however, a culture is started from the products of sexual conjugation between individuals, these will be genetically diverse owing to the segregation of genes, and natural selection will therefore be able to operate. Gause showed that in several cases natural selection in a particular environment operates to produce changes, in characters such as body size, which are similar to the direct adaptations to that environment exhibited by vegetatively propagated clones. This he considers to be 'organic selection' in the sense of Baldwin, since it involves the selection of genes which act in the same direction as the environment. But he does not seem to conceive of the genes as controlling the response of the organism to the external circumstances; he phrases his description always as though the action of the gene was quite independent of the environment. In fact, when the abnormal environment is so extreme that the initial vegetative clone cannot survive in it, although some segregating ex-conjugants succeed in doing so, he denies that this is also organic selection, writing: 'With increase in salinity of the medium to 7 per cent, adaptive modifications in *Euplotes vannus* are no longer formed and various strains entirely die out. Among ex-conjugants direct natural selection of viable individuals occurs . . . In this case there is clearly no organic selection'. Thus he

does not consider that the segregated genotypes which are favoured by natural selection operate by making possible an adaptation of some kind to the environment; he seems to think of their phenotypic effect as merely selected by the environment, but not otherwise modified by it. In this he is probably giving to the phrase organic selection, though in modern Mendelian terms, precisely the significance which Baldwin and Morgan intended it to bear; and although he does not discuss the problem of why an adapted race, when replaced in its original environment does not always return completely to its initial form, the general tenor of his thought would undoubtedly lead him to a reliance on 'random mutations'.

The whole train of thought found in Gause, and implicit in the old idea of organic selection, is based on the over-simplification of forgetting that the environment is one of the determinants of the phenotype. It is more realistic to envisage these phenomena as the selection, not of genes whose effects, though not modified by the environment, happen to be parallel to the direct adaptations, but rather of factors which control the capacity for response to the environment. In these terms, we can go further in finding an explanation for partial irreversibility of adaptation. It has become a commonplace that natural selection for any character will alter the general 'genetic background' of the population. If the selection is for the capacity to respond adaptively to the environment, it will mould the epigenetic landscape into a new form, in which this response is facilitated and perhaps adjusted so as to reach the most favourable end-result. There will then be two ways in which genetic fixation of the originally acquired adaptive character may take place. Either a gene-mutation occurs which suffices to direct development into the channel which has been prepared for it; or the remodelling of the epigenetic landscape goes so far that what was initially the side-valley, reached over a threshold, becomes the most easy path of change, so that one cannot point to any particular genes as being responsible for switching development into it (Fig. 30).

The more thorough the remodelling of the landscape, the more likely will it be that some random gene-mutation will be able to



take over the switching function of the original environmental stimulus. The type of hereditary change envisaged by Baldwin is, therefore, much more likely than he could have realised. But one may still ask why, even if such a mutation occurs, should it be

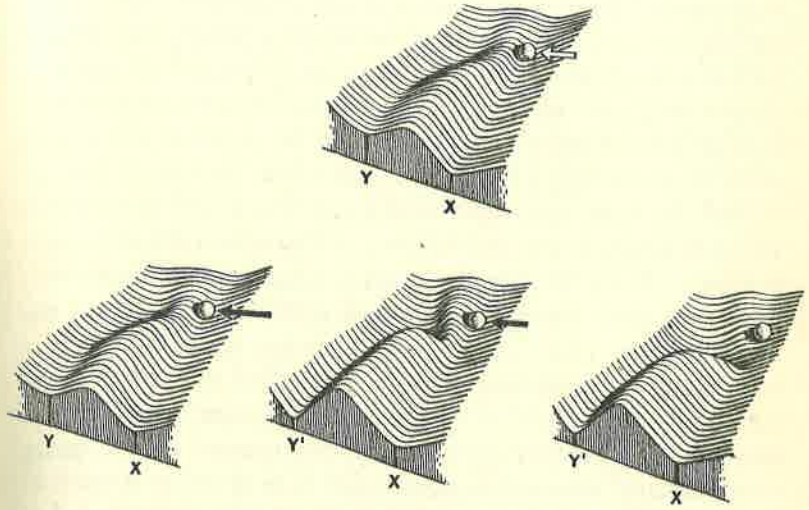


FIGURE 30

*'Organic selection' (the Baldwin effect) and genetic assimilation.* The diagram above shows part of an epigenetic landscape, with a main valley leading to the adult character *X* and a side branch leading to *Y*; the developing tissue does not get into the *Y* path unless an environmental stimulus (hollow arrow) pushes it over the threshold. The three diagrams below show ways in which the 'acquired character' *Y* might become incorporated into the genotype. On the left, the original environmental stimulus is replaced by a mutant allele (dark arrow) which happens to turn up; this is 'organic selection'. On the right are two modes of 'genetic assimilation'. In the central one, the threshold ptotecting the wild type is lowered to some extent, but there is an identifiable major gene which helps push the developing tissues into the *Y* path. On the right, the genotype as a whole causes the threshold to disappear and there is no identifiable 'switch gene'. Note that in both the genetic assimilation diagrams there has been a 'tuning' of the acquired character, i.e. the *Y* valley is deepened and its end-point shifted from *Y* to *Y'*.

selected? Provided natural selection so improves the capacity to respond to the environment that the adaptive change is regularly performed, is there any advantage in going further, so that the adaptation becomes genetically fixed and occurs even in the absence of the stimulus? The answer, I think, must be an appeal to the same type of consideration which we have to call upon to explain the evolution of canalisation in general. The almost universal occurrence of developmental buffering, and the generality of some degree of genetic fixation of adaptations, suggests that it is an advantage if individuals tend to produce the modal adaptive phenotype even if their particular life-history has involved circumstances which were somewhat unlike those which are most common for their fellows, and to which the adaptation is adjusted. If, for instance, there was no canalisation of the growth habit of a plant ecotype, every cold spring would convert the lowland forms into alpine types unable to take advantage of a succeeding warm summer. It looks as though it must be too difficult for natural selection to produce organisms which always respond in a perfectly adjusted adaptive manner to fluctuating environmental circumstances, and that *faute de mieux* it tends to fix, by canalisation, a type which is reasonably well adapted to the situation it will most frequently encounter. When this occurs in a population living in an environment which remains relatively unchanged for considerable periods, it is the process which we have called canalising selection. When it happens to a subpopulation which is carrying out exogenous adaptation to a new environment, it converts this into a pseudo-exogenous adaptation, and the 'acquired character' becomes genetically assimilated.

Since the concept of the epigenetic landscape is not well known, and since some people find it difficult to follow an argument expressed in terms of a three-dimensional diagram, it may be well to discuss these ideas in another way. Let us consider first a population of animals living in some environment $A$, in which some important character, subject to natural selection, takes values within the range $a$ (Fig. 31). Suppose that the character is influenced by the environment. The genotypes composing it will react each according to its appropriate sensitivity, and if,



in a later generation, the conditions change from those of $A$ to those of $B$, some individuals may be supposed to react according to the line $P$, others say to $Q$, to give only two examples. Now if the optimum for the character in environment $B$ falls within the range $b$, $Q$ will be favoured by selection but $P$ rejected. On return to $A$, however, a population of Q's would again exhibit the character within the range $a$, and no assimilation towards the modified $b$ range would have occurred. The simplest possible

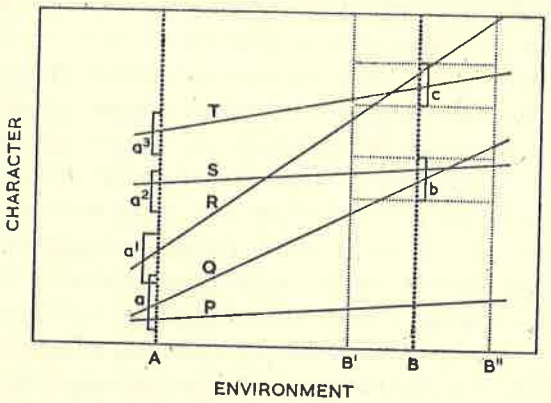


FIGURE 31
*The assimilation of a character which varies in a quantitative manner.*

type of genetic assimilation would occur if selection favoured the building up of genotypes which produced the character within the range $b$ even though the environment might vary from $B'$ to $B''$. This would lead to the appearance of individuals which reacted more like $S$; and a population of these, on return to $A$, would show the character within the range $a^2$. In this case, the rejection of $P$ in favour of $Q$ is an example of 'normalising selection' (p. 72); and the formation of genotypes which react like $S$ rather than like $Q$ is what we have called canalisation; while the almost complete retention of the modified $b$ range when the population returns to $A$ is the phenomenon which leads one to speak of assimilation.

It is also possible for normalisation in $B$ to take place around some optimum value which lies outside the range which can be

produced by direct adaptations such as are indicated by the lines *P* and Q. It might be, for instance, that the optimum fell in the range *c*. Then if we had purely normalising selection, with no canalisation, the result might be genotypes which react like *R*, in which the 'strength' of the genotypic tendency for the character, and the sensitivity to the environment, have both been increased. On return to *A*, they would regress towards the range *a*, but would not quite reach it, coming say to $a^1$. If, in similar circumstances, canalising selection also operated, we might get genotypes reacting like *T*, which would regress only to $a^3$.

In Fig. 31, an individual of the initial population whose reaction to environmental change is indicated by a rather steeply sloping line, such as Q, would be exhibiting only very loose canalisation. It is worth considering the situation when the character of the original population is strongly canalised. As an extreme example, we can consider a character which is entirely unexpressed in the original population in environment *A*, and which is developed only when some threshold is exceeded, as, we will suppose, it is for some individuals when the population is transferred to environment *B*. We may then postulate that the original population would react as indicated by line Q in Fig. 32; but note that here

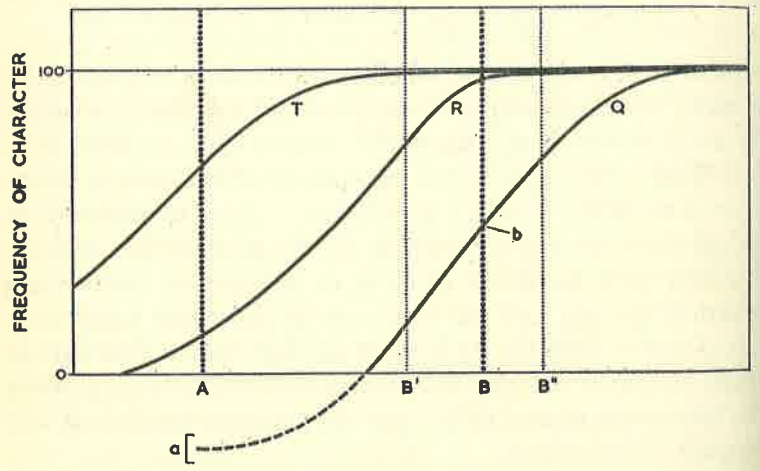


FIGURE 32
*The assimilation of a quantitative character which involves a threshold.*



the ordinate is the *frequency* with which the threshold is exceeded and development is diverted out of the canalised normal path into the abnormal path. If the abnormal phenotype has an advantage in environment *B*, there will be normalising selection to increase the frequency with which it occurs, so that the population will be changed into one which reacts like *R*.

This may already show some assimilation, in the sense that the abnormal phenotype may appear, perhaps only in a low percentage, when a return is made to environment *A*. Further assimilation could occur if canalising selection becomes important. It could do so in two rather different ways. In the first place, it might be advantageous to produce the abnormal phenotype in high frequency even if the environment varied throughout the range *B'* to *B''*. We should then build up a population reacting like the line *T*; and this corresponds in its mode of formation to the population also indicated by *T* in Fig. 31. But there might also be another form of canalising selection. We have so far considered the abnormal character as unvariable, but there might be some optimum degree of expression of it in environment *B*, and if there were, canalising selection would tend to build up genotypes which produce this grade of expression throughout the *B'* to *B''* range. This possibility cannot be indicated in Fig. 32 which deals with the frequency of the abnormal phenotype, but not with its degree or kind of expression. In Fig. 30, however, the results of the canalising selection which brings the abnormal phenotype to its optimum expression have been indicated by a change in the course of the side valley and a shift in the end point which it finally reaches.

An evolutionary process which results in the genetic assimilation of a character which was originally produced as a direct adaptive response to the environment must, in fact, be envisaged as a somewhat complex set of changes, which may proceed in sequence, or, more probably, concurrently. One phase in the process is the appearance of genotypes which direct development into the 'adaptive' path, or something like it, without requiring the assistance of any special environmental stimulus. This may sometimes be accomplished by quite straightforward progressive

selection of genes of appropriate activities. This would suffice when the epigenetic systems in the initial population possessed a canalised system in which the adaptive phenotype could be elicited merely by pushing development out of its normal path. Assimilation would then require no more than that the gene-dosage was raised above the threshold which protects the initially normal developmental path (i.e. it would only be necessary to steer development over the saddle in Fig. 30). Often, however, assimilation would require something more, namely the building up of a new canalised path. In its simplest form this involves the selection of genotypes with only restricted responsiveness to the environment (e.g. those like *S* in Fig. 31). In such cases, not only progressive, but also canalising selection would be operative. Finally, canalising selection would, in many if not most cases in Nature, have still another task to perform, namely to guide the new path of development so that it reaches exactly the most valuable end. We might call this the 'tuning' of the adaptive phenotype.

It will be argued later (p. 188) that this process of tuning is, from the theoretical point of view, perhaps the most important aspect of genetic assimilation. So far, however, most of the experimental work which has been devoted to exploring these possibilities has been devoted to the other aspects of assimilation, and has touched only incidentally on the tuning of the phenotypes.

These experiments were made with *Drosophila*. Very strong environmental stimuli were used, which pushed development over well-marked thresholds into quite definitely abnormal channels. In a first series of experiments (Waddington 1954), pupae aged about 21 to 23 hours were subjected to a temperature of 40° C. for four hours. In the foundation stock, a number of aberrations in the wings were produced. One of these, a breaking or even complete absence of the posterior crossvein, was selected for study. Selection was applied for (and also against) the capacity to react to the environment in this manner, the 'upward' selected stock being carried on by breeding in every generation from flies in which the crossvein was broken, while the 'downward' selected were bred by taking in every generation those which



failed to respond. It was immediately apparent that, as might be expected, the capacity to respond was under genetic control and became strengthened (or weakened, as the case might be) as the experiment proceeded. The important point then emerged that genetic assimilation began to occur. After about 14 generations, flies of the upward selected stock were found to produce a small number of offspring which developed broken crossveins even when they were not given the temperature treatment. In order to speed up the further progress of assimilation, these flies were bred from and selected in normal temperatures, when stocks were rapidly produced which had a broken or absent crossvein in a high percentage of individuals.

Similar experiments have been made by G. Bateman (1956), using a number of different foundation stocks (Fig. 33), and in

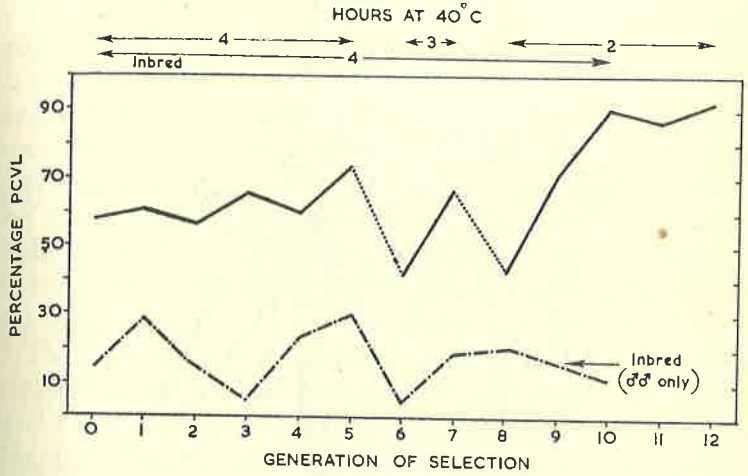


FIGURE 33

*Selection for response to an environmental stimulus.* The graph shows the frequency of flies which showed a broken posterior crossvein (*PCVL*) following exposure to 40° C. for periods of 4, 3 or 2 hours, as indicated at the top; in each generation, only flies exhibiting the modification were bred from. The lower dot-dash graph shows the lack of response to selection of an inbred strain (derived from the same original stock); the inbreds were treated for four hours throughout the experiment. (From Bateman 1956.)

some cases selecting for different environmental responses. Perhaps the most extravagant character which has been assimilated in this way is the well-known bithorax phenotype, in which the metathoracic imaginal buds develop into an accessory mesothorax. This effect is not only produced by the bithorax gene, but can be called forth by various environmental stimuli (ether vapour, high temperatures) applied to the egg shortly after laying (Gloor 1947, Maas 1948). In an experiment in which ether was used as the inducing agent, the character eventually became assimilated after some 28 generations of selection (Waddington 1956). The resulting flies are highly bizarre (Fig. 34). If such a change occurred in

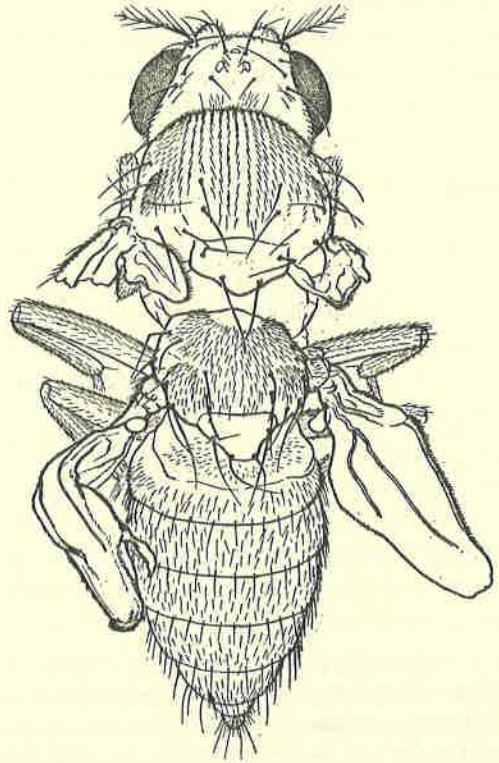

FIGURE 34
*A fly of the 'assimilated bithorax' stock*, with the normal wings removed to show the metathorax transformed into a very complete secondary mesothorax. (From Waddington 1956*a*.)



Nature, it would certainly rank as a 'macro-evolutionary' step. It seems doubtful whether in any other selection experiment such a large change has been produced so rapidly from a normal wild type stock.

These experiments are, of course, artificial in the first place because the responses to the environmental stimuli are not actually of any natural selective advantage, but are only treated as such by the experimenter. This, however, does not seem likely to affect the general principle of the process. It is rather difficult to think of an experimental material which would be convenient for breeding through a large number of generations and which also shows clear-cut environmental adaptations of real value to the organism. Attempts are now being made to study the lengthening of the posterior tracheal horns in *Drosophila* larvae kept in a liquid medium; and it seems possible that the haemoglobin formation induced in certain small crustacea by low oxygen tension might also be favourable experimental material.

Another point to note about the experiments is that the later stages of the selection were based on the assumption that any tendency towards assimilation can be treated as favourable. That is to say, as soon as slight assimilation occurs, and the 'acquired character' begins to appear in unstimulated individuals, these have been isolated and bred from. Thus what the experiments demonstrate is that, *if* genetic fixation of an ecotype is valuable, this mechanism can bring it about.

There are several other interesting points about these experiments. We may ask what is the genetic constitution of the assimilated stocks. Is their epigenetic landscape like that in the centre of Fig. 30, with a modified genetic background but also one relatively important switch gene, or is it like that on the right, in which there is no particular gene which can be singled out as more important than any other for the switch? In the first assimilated crossveinless stock which was made, there was little evidence for the existence of any definite switch gene. In assimilated bithorax, on the other hand, there is a gene (or genes) on the *X* chromosome with rather strong effect. This factor is recessive, and acts by 'maternal effect', causing females homozygous for it to

lay eggs which produce a moderate proportion of bithorax phenotypes. It could be regarded as a switching mechanism; but it only succeeds in giving a really high percentage of bithorax individuals if the rest of the genotype determines a suitable epigenetic landscape with a low threshold for this modification; and some bithoraces are formed even without it.

The process of selection for the ability of an animal to react adaptively to its environment, leading to the genetic assimilation of the adaptive character, provides us with a way of understanding how 'acquired' characters may become inherited without our having to suppose that the external conditions have been responsible for calling into being the necessary genetic determinants. There is no *a priori* theoretical reason which would prevent us imagining that all the genes which eventually make up the assimilated genotype were already present in the population before the selection began, and only required bringing together. Equally, however, it must be admitted that this supposition does not necessarily follow from any of the facts which have yet been related. Most previous discussions of acquired characters and their possible inheritance have turned around the question whether the environment can provoke the appearance of new hereditary variation of an appropriate (i.e. adaptive) kind. It seems to be the opinion of nearly all recent authors, with the exception of Lysenko and his followers in Russia, that the lack of conclusive evidence for such effects, and the difficulty of envisaging, even in theory, a mechanism by which they might operate, justify one in completely rejecting this possibility. Nevertheless, the clear-cut evidence for the conversion of acquired characters into inherited ones which has been provided by the experiments on genetic assimilation make it necessary to discuss the matter again, specifically in connection with them.

If one looks at the facts concerning the assimilation experiments with an open mind, one will find in several cases rather strong indications that the genetic basis for the assimilated genotype was in fact present in the initial population; while there is little that positively suggests—and that not very compellingly—that the environmental stress has called forth the new variation. In the



case of the crossveinless phenocopy, the evidence for the preexistence of the genetic variation is indeed convincing. In the population from which Waddington's experiments were started, no crossveinless flies were found before the experiment began, but the number of individuals examined was not very large. Bateman (1956), who repeated the work using a different stock, checked over very many more of her initial population, and found a low percentage (<1 per cent) of spontaneous, naturally occurring low grade crossveinless individuals (Fig. 35). Selecting

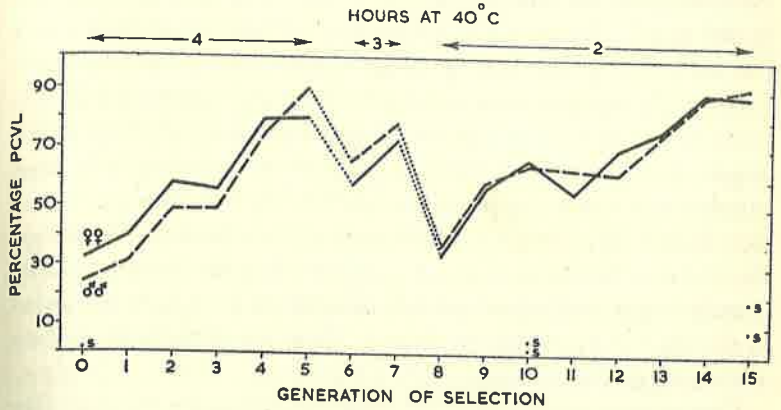


FIGURE 35

*Selection for liability to heat-induced crossveinlessness.* In this stock ("Edinburgh") unlike that shown in Fig. 33, there were a very few spontaneously crossveinless flies in the population from which selection started. The frequency of these is shown by the points marked *S*; their numbers increase as the selection for heat-sensitivity proceeds. (From Bateman 1956.)

these, she was able to build up a crossveinless stock, and when this was tested against the assimilated crossveinless stock eventually derived after the temperature treatment, they appeared to be identical in genetic constitution. It seems certain, then, that all the genes which enter into the genotype of the assimilated race were already present in the initial population, though in such low frequencies that it was only in a very few individuals that sufficient of them occurred together to give a crossveinless phenotype.

What the environmental treatment has done in this case is to reveal sub-threshold concentrations of these genes, and thus made it possible for selection to get a hold.

Attempts to carry out genetic assimilation starting from inbred lines have remained quite unsuccessful (Fig. 33). This provides further evidence that the process depends on the utilisation of genetic variability present in the foundation stock with which the experiment begins.

In most of the other phenocopies studied by Bateman, no individuals of the abnormal type could be found in the initial populations. This does not, of course, prevent us supposing that the necessary genes were present; if the assimilated genotypes involve the co-operation of large numbers of genes, and if each of these was present only in low frequency before the experiment began, it would require a search through an unmanageably large number of the starting population before an abnormal individual was found. We are only confronted with a positive suggestion that new variation has arisen if it appears that the assimilated race contains a gene which we cannot imagine to have been concealed in the stock with which we began. There are actually three cases which suggest something of the kind.

One of these arose during Bateman's selection for a dumpy-like phenocopy provoked by a temperature shock to the pupa. An assimilated stock was eventually produced, and it was shown that an important element in its genotype was a gene with a rather strong tendency to produce dumpy wings. It is probably an allele of the well-known dumpy locus. Although when this gene is crossed out of the assimilated genotype into a wild-type background it causes dumpy wings to appear only in a low percentage, it seems most unlikely that it could have been concealed in the initial population; it is more probable that it has arisen by mutation during the course of the experiment. The same conclusion applies even more forcibly to a dominant (with recessive lethal effect) which appeared during the selection of the bithorax phenocopy and caused a slight enlargement of the halteres. This gene, which seemed to be identical with the previously known $bx^D$, actually appeared twice, once in each of the two selection



experiments which were being carried out simultaneously; but there is always the possibility that the second appearance was due to contamination by a fly which had escaped from the stock of the mutant which had by that time been established. The gene did not produce a full assimilation of the phenotype, of the kind which the experiment was attempting to produce, and it was therefore eliminated from the line which was carried on and which eventually gave rise to the assimilated stock described and figured above. Its importance in the present connection is that even when transferred into a wild-type background the gene gave a well-marked effect, and it certainly was not present in the original population; it must have arisen by mutation.

The third suspicious case also occurred in the bithorax experiments. In the final assimilated bithorax stock, an important element in the genotype is a sex-linked recessive condition which produces a 'maternal effect'; that is, the homozygous mothers lay eggs which tend to develop into bithorax phenotypes. When *X* chromosomes containing this genetic condition are transferred into a wild-type background the homozygous females still produce some, though rather few, bithorax offspring. It is quite certain that this did not happen in the original population. The only way to escape the conclusion that the genetic determinant has arisen by mutation during the experiment is to suppose that the maternal effect is caused by two or more linked genes in the *X* chromosome, in which case the frequency of the complex in the initial population might have been so small that it would be overlooked. This is, however, rather a forced explanation.