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Genetic Assimilation, or the «Baldwin Effect»

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Abstract. — It is not necessary to suppose that « exogenous » characters become fixed in the genetic composition of a species by an accumulation of independent mutations that eventually produce an imitation of those characters. Instead, the characters are already under genetic control, requiring at first a particular stimulus to make them manifest, but by means of selection the range of sensitivity of the organism to stimuli is widened so that the characters then appear under « normal » conditions.

Adaptations may develop in an individual by the direct response of organs or tissues to environmental stimuli, or to physiological stimuli within the body. The size, strength and facility of muscles increase as a result of exercise. One kidney, if the other has been removed, increases in size and in its capacity to handle the complex process of excretion and water balance. The soles of the feet become thick and tough as an effect of walking. But all these adaptations are **exogenous** or physiological, and nothing is inherited except the ability of the body to make such responses.

Then how does it happen that an unborn fetus has soles that are already thicker than other parts of the skin? How do the pectoral muscles and associated skeletal parts of a mole become heavy and powerful before the individual uses them? How can an ostrich hatch from the egg with callosities on its belly and breast, such as it would receive from resting on them later? At first glance this looks like evidence for Lamarckism, the inheritance of acquired characters, for there is no doubt that these latter examples are genetically determined. On account of their resemblance to the non-hereditary traits mentioned first, they are called **pseudoexogenous.**

It was suggested by Mark Baldwin in 1896, in the American Naturalist, that hereditary variations which happen to coincide with acquired, non-hereditary modifications would thereby be favored by natural selection and would be sustained until they became fully established in a species. C. Lloyd Morgan (1900) accepted this explanation, saying, «survival would in the long run be better secured, we may suppose, where the two methods of adjustment were coincident and not conflicting.» Morgan applied this idea especially to the evolution of animal behavior in an attempt to explain the origin of (inherited) instincts by fixation of (acquired) habits in the genetic constitution of the species. «There would be a distinct advantage in the struggle for existence when inherited tendencies of independent origin coincided with acquired modifications of behavior; a distinct disadvantage when such inherited tendencies were of such a character as to thwart or divert the action of intelligence. Thus any hereditary variations which coincide in direction with modifications of behavior due to acquired habit would be favoured and fostered.»

Simpson (1953) expressed the hypothesis in this way: (a) non-hereditary adaptations are produced by interaction between organism and environment, (b) genetic factors then produce traits similar to these, and (c) these factors are favored by natural selection. The Baldwin «effect,» if there is any, means that the non-hereditary adaptations, including those of behavior, provide a means of sustaining a species during such time as may required for independent mutations to appear and spread through the population, causing the same adaptation to have a genetic basis. Presumably at this point direct environmental control might be relinquished, or «habit» might become «instinct.» But a careful consideration of the problem in reference to particular cases suggests that the explanation may be simpler. Incidentally, the belief of nineteenth-century biologists that instinct (inherited patterns of behavior) evolved from habit (learned behavior) is contrary to the evidence and is not held by modern students of animal behavior. Neither Simpson nor any other recent evolutionist applies his discussion of the Baldwin effect to behavior.

Considering, first, the formation of callosities, these are due to an increased rate of proliferation of cells in the germinative layer of the skin, and of their conversion to stratum corneum. But obviously the skin that is not callused has its own normal rate for these processes, and this is certainly under genetic control; it differs in various species of mammals, and it is subject to occasional heritable abnormalities within one species (man). The inherited characters here are two: a normal rate of growth, and the ability to increase this in an area mechanically stimulated, thus forming an «exogenous» callus. The non-inherited character is the callus itself, which results from mechanically-induced acceleration of growth. Pseudoexogenous callosities are undoubtedly the result of some genetic change. But this need not, and probably cannot, be a random, «independent» mutation of genes

having no previous relation to the character. Instead they result from an increase of the normal growth-rate, affecting a particular area. The effect of the genotype previously responsible for normal growth-rate has been heightened, the growth accelerated, and it seems unlikely that any other genes than those already involved would enter the picture, for only a slight variation of these would produce the change. It could probably be expressed also as increased sensitivity of the tissue to whatever agent controls its normal rate of proliferation.

If this explanation is accurate, natural selection could of course favor the spreading of the changed genetic control of the character through the population. But this is very different from, and much simpler than, imagining an accumulation of clandestine mutations under cover of a protective exogenous adaptation until somehow they are able to produce an imitation of the exogenous adaptation itself. If the result were to produce the same character, precisely, it is difficult to conceive of natural selection playing any part whatever, because the character was already produced in response to appropriate stimuli. But in fact the character is not the same; it differs in its earlier appearance, more rapid development, and the heightened sensitivity of the tissues to the same stimulus. This is the essential change and the reason for selection.

As a second case, Waddington (1957) discussed the problem in relation to natural selection, proposing ways in which the exogenous adaptive characters can be «assimilated» into the genetic constitution of a species. In brief, granting that a species inherits the ability to respond to an environmental stimulus by producing a phenotype, he proposes that natural selection will «mould the epigenetic landscape» (i.e., the processes of development) into a new form, which increases the organism's ability to reach a favorable result, perhaps by means of genes that «switch» development into an easier pathway. He cites several experiments to indicate how this takes place. A study of Waddington's experiments suggests that his results are open to a simpler, more direct explanation, that appears to serve also for other examples of «genetic assimilation.» His account of the work is quoted here:

«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 25 hours were subjected to a temperature of 40 degrees 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. »

Although one may not, on first reading, understand how these results could be brought about without subscribing to some Lamarckian effect, the key lies in the statement that « the capacity to respond was under genetic control, » even at the beginning, and in the further fact that the stimulus, a higher than normal temperature, used during each of the 14 experimental generations, differs only in a quantitative way from the normal. It is not as if the normal flies were produced under no influence of temperature at all, but merely at a temperature a few degrees lower. The genotype was such that it permitted a certain frequency of the phenotype « broken or absent crossvein » to appear when the pupae were treated, but it lacked the capacity to make this response at the normal temperature. By selection under the experimental conditions a greater frequency of this response was brought about in the course of 14 generations. A part of this result (not an additional result) is that a normal temperature could now also induce the response occasionally. The range of sensitivity to temperature had been extended slightly downward. After this was done, selection for the character « broken crossvein » could of course be carried on without regard for the experimental condition of temperature, because it had become patent (phenotypic) without that condition.

It seems highly misleading to use the term « assimilation » for this phenomenon, since the word implies that the effect of an environmental influence has been incorporated somehow into the genetic system of the flies, but this has not happened at all. The capacity to respond to temperature per se has been shifted by selection in a perfectly normal manner. To say « the character became assimilated » obscures what actually took place.

A similar explanation will probably account for other examples of « pseudoexogenous » adaptations without assuming any independent, random mutations to « fix » genetically a character that was produced « by the environment » initially. A character that is in some measure adaptive, but that does not appear unless certain interactions of the organism with the environment take place, does not on that account lack a genetic basis. We can say that a genotype exists which, given

a certain environmental stimulus, is capable of producing the adaptive phenotype (eg., calluses) or for that matter, one that is non-adaptive (broken crossvein). Initially this genotype may only occasionally have responded in this way, and may not have been common to the whole population. If the phenotype in question has adaptive value (or if, in an experiment, artificial selection favors it), two results may be brought about. One is to increase the frequency of the genotype in the population. The other is to favor a wider sensitivity or responsiveness of the organism to the stimulus in question. Environmental stimuli are rarely if ever discrete units or unvarying quantities, nor is the exposure of the developing organism to them an all-or-none situation.

It seems, outwardly, that an « exogenous » adaptation has come to be hereditary. The basis for this character was genetic in the first place, but the sensitivity of the tissues involved was not then adequate to include response to the « normal » as well as the « abnormal » stimulus.

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