

Is Natural Selection still have to be Regarded A Foundation Stone of Evolutionary Process?

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Abstract

Natural selection is a buzzword used to describe the main driving force of evolution. Its creative role is believed to be based on: a) an unlimited variety of organisms caused by hereditary variation and b) a direct connection between hereditary changes and their phenotypic expression. These are the two requirements that can lead to the genetically based changing modalities of characters through "iterations" of natural selection in the series of successive generations. Are these two requirements fulfilled in the nature, however? The present study focuses on the analysis of these two "foundation stones" of natural selection. Firstly, hereditary variation is shown to be essentially non-homogenous. New hereditary characteristics of individuals fall onto a narrow "strip of land" in the sea of potential possibilities. Secondly, the consequences of changes in the genotype of an organism are involved into a system of hierarchical multiple compensation, from the molecular to the biocenotic level. In a way, the signal of hereditary change passes through a series of "system filters" at epigenetic, ontogenetic, physiological, behavioural, populational and biocenotic level. Each filter is represented by multiple feedbacks maintaining the integrity of systems at each level and at all the hierarchical levels taken together. It is in these "system filters" the adaptive nature of characters is formed representing the every individual as a subject to the Law of Multilevel Self-Organization. The emerging understanding of this provides a strong reason to change the evolutionary paradigm from the mainly selectogenetic to the mainly orthogenetic one.

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Introduction

Natural selection, laconically expressed by Spenser and Darwin as "...the survival of the fittest"^{23, 115}, has become a key concept in the interpretation of microevolution events. In various forms, it has been used to explain: adaptive shifts in the modality of character expression under changing environmental conditions (directional selection); maintenance of optimal values of characters in stable environments (stabilizing selection); formation of bimodal frequency distributions followed by the divergence of forms in situations when several optimums are present in the environment (disruptive selection); formation of adaptations directly associated only with the reproductive success during sexual reproduction (sexual selection). The idea of natural selection is the fundamental component of population genetics and generally of the 20th century evolutionary synthesis^{24, 30, 67, 72, 129}. Natural selection underlies generally accepted speciation models⁶⁷.

However, in recent years, the notion of NS as an universal mechanism of microevolution has raised more and more questions. There are at least three directions of a critical rethinking of NS. First is a discussion about considering NS as a putative evolutionary mechanism from the point of view of a new mechanistic philosophy^{6, 45, 81, 114}. Second is expanded understanding of phenotypic plasticity and transgenerational inheritance^{25, 62, 64, 93, 127, 130} that dramatically change the relationship between mechanisms of phenotypic plasticity and genotypic adaptations. The last in its turn can change the evolutionary paradigm to the "extended evolutionary synthesis". Third is growing attention to the mechanisms of self-organization of molecular complexes, cell organelles, cells and multicellular organisms^{49, 63, 69, 82, 83}. From this point of view, NS is much more restricted than it was previously assumed. It can operate only to form specific conditions for the implementation of self-organization processes. Thus, generally, the last decades again put the notion of NS in the discussion plane and my work represents one more step in this direction.

Within the framework of the modern evolutionary synthesis, studies of the intensity of natural selection are based on the assessment of "fitness", the degree of reproductive input of an individual into the next generation⁴. The idea of "reproductive success", a greater contribution of an individual into the gene pool of the next generation as compared with the carriers of other genotypes, is central here. It is unimportant whether it is achieved through differential mortality (the survival of the fittest) or in some other way. What is important is that individuals carrying a certain genotype in the series of successive generations ("iterations" of the selection) change the representation of this genotype in the population*.

Can we prove that different individuals in the population contribute unequally into the next generation? Indeed, we can. That is what all studies of the reproductive structure and the dispersion of fertility indices in populations point to. Many of the individuals simply do not live long enough to reach the reproductive stage. The dispersion of individuals within populations as to the numbers of surviving offspring is always high^{19, 21, 36, 84, 92}. The length of the pre-reproductive phase varies³⁸, and the reproductive period itself may be longer or shorter in different individuals in the same population^{14, 20, 29, 74}. We may, therefore, be fairly sure that the contribution of individuals into the gene pool of the next generation is unequal. Whether this unequal contribution is connected with evolution, a vector process involving series consisting of hundreds and thousands generations is, however, far from being evident. Such a connection, if any, has to be separately proved. It may well turn out that we observe mere stochastic or cyclic populational-genetic processes¹ rather than changes at the evolutionary scale allegedly caused by "natural selection".

So, the differential contribution of individuals into the gene pool of the next generation as such does not prove that the mechanism of natural selection is at work. This begs the question: can natural selection be considered as the main driving force of evolution? This is

Note: In our opinion, the notion of selection should not be expanded to mean the universal synthesis of the oppositions of "prohibition and permission" (see⁷⁷, p. 252). Such an expansion makes it impossible to disentangle the processes of structural self-organization at various levels and the processes ruled by the mechanism of selectogenesis and, thus, to distinguish selectogenetic and orthogenetic evolutionary mechanisms.

the key issue addressed in the present study. To solve it, I will briefly characterize the primary material of natural selection, that is, hereditary variation (section 2, Potential material for natural selection). Then I will analyse, in a series of hierarchical systems, how probable is the formation of a correlation between variations of the genotype and the reproductive parameters of an individual (section 3, System filters...). If found, a distinct correlation of this kind would provide compelling proof of the effectiveness of natural selection as an evolutionary factor.

Potential Material for Natural Selection

Stating the Question

Natural selection acts upon the entire range of hereditary variation in a given population. There are, however, certain requirements to the nature of this variation. If these requirements are met, we can consider natural selection as an important mechanism of changing the modality of the genotypic composition of the population (to be entirely sure about its effectiveness, one should also analyse the importance of "system filters" – see section 3).

There are two major requirements. Firstly, the variation should be to more or less "homogenous" (or isotropic⁵³). This means that it should be characterized as unlimited, potentially representing a broad range of

variants. The manifestations of hereditary deviations should be distributed more or less evenly across the axes of characters' variation. The second requirement is as follows: the primary act of hereditary variation should not depend on the *need* in the act and the *nature* of this need*.

If both these requirements are met, we may be fairly sure that the subsequent changes in the frequencies of characters indeed can be, in principle, be formed by natural selection. On the other hand, if these requirements are not met, we should rethink the leading role of natural selection in microevolutionary events.

For instance, the "non-homogeneity" of the character space (the first requirement) — in other words, canalized nature of hereditary variation, considerable differences in the frequency of manifestation of the hereditary variants, the discreteness of forms in the hereditary variants — testifies to the importance of the "construction-determined" evolutionary mechanisms and to the prevalence of orthogenesis (Box 1). The greater "non-homogeneity" is observed, the more important orthogenesis is (Fig. 1). The role of natural selection is reduced to eliminating non-viable variants and the work within the range of the origin of forms, which is limited and predetermined by the variation^{8, 12, 17, 53, 70, 79, 85}.

Box 1 *Three types of evolutionary concepts.*

*Indirect adaptogenesis, syn. **selectogenesis** (term^{12, 70}), from Latin *selectio* — choice, selection; syn. **tychogenesis** (term⁹⁰, p. 813), from Greek *týche* — luck, fortune.*

Heterogeneity of organisms in respect of reproductive characters — random, unlimited and independent of the environment — is the cause of differential input of individuals into the next generations. The major mechanism of transformation is selection of the "fittest", or natural selection. Cumulatively, in the series of generations, it results in adaptive changes of the modal species characteristics. The environment determined the direction of selection, and therefore the

*model is **ectogenetic**: it admits transformism under the influence of external factors. The model is also **idiographic**, as it focuses on the uniqueness of transformation acts and their probabilistic character. The ideas of indirect adaptogenesis underlie the "modern evolutionary synthesis" of the 20th century.*

Direct adaptogenesis.

*The impact of the environment results in adequate changes in the morpho-functional inheritable characteristics of the organism. In this way, adaptive changes of organisms are not mediated by selection but form directly under the influence of the environment in a series of generations. This means that the model admits the existence of **ectogenesis**, transformation under the*

Note: In essence, these requirements have been formulated by Meyen and Chaikovsky⁸⁰, p. 12, paragraphs 5, 6, and 9 in their review of the ideas of A.A. Lyubishchev concerning selectogenesis.

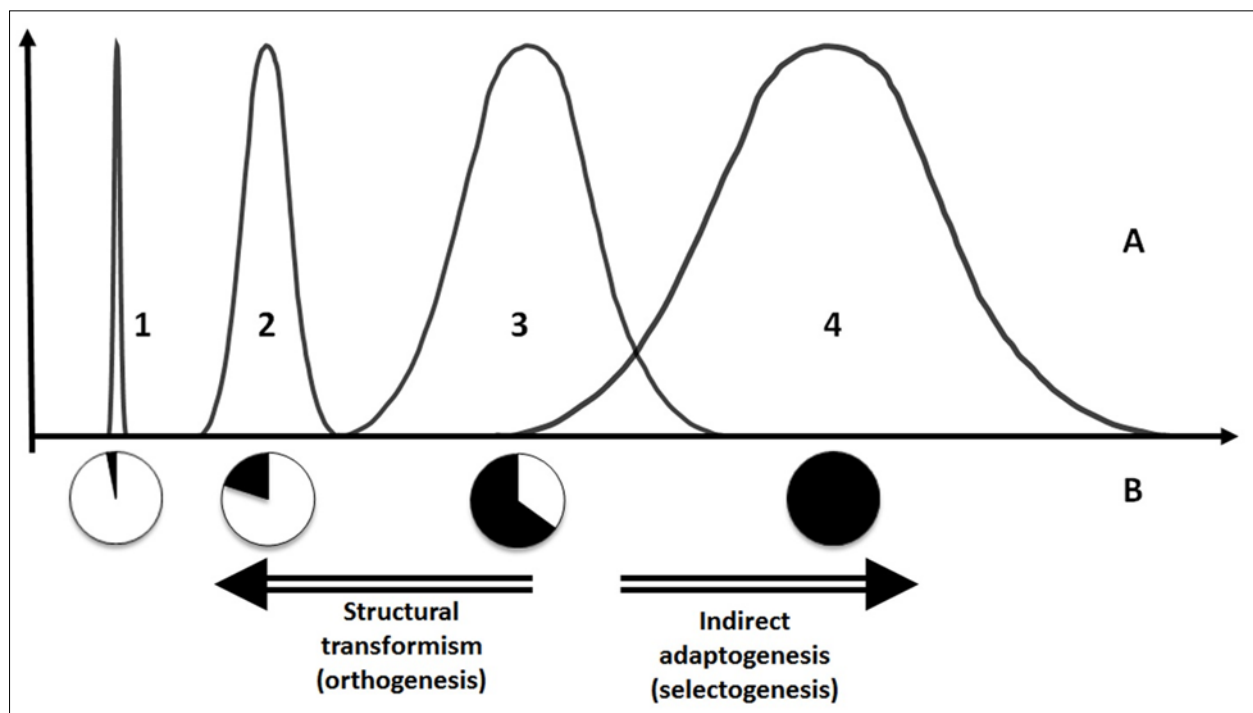


Figure 1. Schematic representation of: A - frequency distributions of a character with a narrow (1,2) or broad (3,4) range of potential values; B – proportion of implemented variation (black sectors) in the potential range of variation of this character (circles under the abscissa). The proportion of implemented variation increases in the series 1 – 4. If the progeny of organisms is characterized by a small proportion of implemented variation (1, 2 B) and a narrow range of character implementation (1, 2 A), the evolutionary mechanisms of structural transformism (orthogenesis) prevail. If the progeny of organisms demonstrates a large proportion of potentially possible variants of a character (3, 4 B) and the values of the character vary broadly (3, 4 A), indirect adaptogenesis (selectogenesis) is possible. Accordingly, the role of natural selection increases from left to right along the abscissa.

*influence of external factors. The model is **idiographic**: each transformation act, being determined by a unique combination of the environmental conditions, is considered as unique. The mechanism of direct adaptogenesis underlies the ideas of various Lamarckian evolutionary hypotheses.*

Structural transformism, syn. orthogenesis (term ⁴⁰), from Greek ὀρθός — straight.

The model considers internal patterns of morpho-functional organisation of living systems as the driving force of transformation. Its logic can be characterized as "self-assembly" (rather than selection!) of increasingly complex systems. Therefore, it is the only model that

*consistently admits the existence of **autogenesis**: the change based on internal patterns of the organisms' structure. According to this model, variation of organisms is strictly directional rather than random. The model is **nomothetic**: it searches for strict laws of evolutionary transformations and envisages the possibility of predictive interpretations. On the whole, it admits the existence of internal directionality of the evolutionary process, in other words, orthogenesis. The main problem of orthogenetic interpretations of evolution is associated with the explanation of the formation of adaptations in the course of evolution, that is, their correspondence to the environment ⁷⁰.*

Failure to fulfil the second requirement spells the acknowledgement of direct adaptogenesis (see Box 1. Three types of evolutionary concepts). Direct adaptogenesis occurs when the impact of the environment on the individual is accompanied by the formation of the hereditary variants of the progeny that are, in general, consistent with the impact (inheritance of acquired characters). This situation is essentially different from that envisaged by the natural selection model, where adaptations are formed indirectly, by selection from the available variants (indirect adaptogenesis).

What are, in fact, the manifestations of the hereditary variation? Are we in the position to say whether the two requirements are fulfilled and to provide the rationale of the effectiveness of natural selection?

Arguments in Favour of Macro-Evolutionary Approaches to the Assessment of Variation Structure

The most general approach to the assessment of the "homogeneity" of the character space is the construction of potentially possible (mathematically calculated) spaces of certain characters. Within this approach, the calculated "morphological space" ("morphospace") of the potential variation of a character is compared with that actually implemented in a certain species of organisms, group of species or group of organisms united into taxonomical categories of a higher rank. In all these cases it has been shown that only a minor proportion of the potential diversity is actually implemented in the nature ^{18, 75, 78, 100, 101, 102, 113, 118, 119, 121}. This discrepancy between the potential and the actual diversity may be an argument in favour of essential structural constraints of the origin of forms (that is, the primary "non-homogeneity" of variation). At the same time, this assessment is not necessarily incompatible with a different viewpoint: the initial variation is reasonably "homogenous" and the subsequent non-homogeneity is the result of the mechanism of differential mortality, that is, natural selection.

The analysis of hereditary variation of characters in closely related species allows one to make more definite conclusions. The range of variation and particular characters turn out to be rather conservative in species of the same genus and even the same family.

Manifestation of the same range of forms in the variation of different species may concern both particular morphological characters and complexes of correlated characters representing certain "morphotypes". This phenomenon is best described by Vavilov's law: the law of homologous series of hereditary variation ¹²³. It emphasizes, firstly, the similarity of series of variation in closely related species and genera (the similarity being directly proportional to the relatedness of the taxa under comparison) and, secondly, the similarity of cycles of variation in subordinate taxa within the category of a higher rank (such as a family). Noteworthy, discussing the formation of such parallelisms of variation, Vavilov admitted the possibility of a similar direction of the mutation process in closely related species (see ¹²³). Homologous series reflect the nomothetic nature of variation: it is not stochastic but subject to general rules. Such ideas are incompatible with the initial hypothesis about the "homogeneity" of variation. Nomothetic nature of variation is discussed in detail by Vasil'yev and Vasil'yeva ¹²² and widely discussed in modern literature (see, for example ^{8, 53, 85}).

The nomothetic nature of variation at the level of macrotaxa is strongly supported by paleontological data on the evolutionary formation of large taxonomic groups of organisms: archaeocyathids ¹⁰⁴, arthropods ^{95, 96}, mammals ¹¹⁷, echinoderms ¹⁰⁵ and birds ⁷¹. Limitations applying to the origin of forms and numerous parallelisms indicate that Vavilov's law of homologous series is expressed at the scale of evolution of macrotaxa ¹⁰⁶. Therefore, the "non-homogeneity" of variation may play a pivotal role in directed character of the evolution.

Ideas about limitations of variation find an even more general expression in the concept of repeating polymorphic sets ^{78, 113}. The emphasis here is on the distributions of the modalities of separate characters (rather than character sets as in Vavilov's law) and their similarity ("isomorphism" according to ⁷⁸) in different taxa. Moreover, the notion of repeating polymorphic sets falls outside the scope of phylogenetic homology. Their repetition "...can be observed in obviously non-homologous parts ..." (⁷⁸, p.165). This is equivalent to an outright acceptance of the existence of some laws of variation and origin of forms that are not subject to

“phyletic” relationships. From this viewpoint, the potential role of natural selection in origin of forms is reduced to secondary corrections.

An important argument in favour of a considerable “non-homogeneity” of the space of hereditary variation is the wide distribution of agamic, and parthenogenetic species among protists and multicellular organisms^{39, 44, 107}. Their morpho-functional distinctness is not associated with any special mechanisms uniting their gene pool and at the same time isolating it from other genetic entities. The very existence of distinct species in such organisms is an evidence of the discreteness of the origin of forms and its canalized nature.

Another argument jeopardizing the hypothesis about the “homogeneity” of the space of hereditary variation is provided by the existence of numerous so-called cryptic species. These are genetically separate groups endowed with all the prerequisites for morpho-functional divergence on the basis of their genetic separateness. The scale of genetic differences in such groups may vary; in fact, they are often more than sufficient for these species to be considered as reliable “biological species”¹⁶. However, despite the genetic divergence, the representatives of these groups are characterized by a conservative structure and an extreme paucity of morpho-anatomical differences. Cryptic species occur in a variety of taxonomic groups of eukaryotes, from protists to multicellular animals⁴⁴, for a review of multicellular animals see¹²⁰. The importance of “non-visual signals” for the establishment of reproductive isolation and the stabilizing selection for a certain morphotype have been used to explain the evolutionary formation of such complexes of species¹⁶. However, the problem of their morphological conservatism remains. Actually, under conditions of an acquired (no matter how) reproductive isolation there should be an extremely effective mechanism of maintaining the conservativeness of morphological characters (stabilizing selection?), also explaining an impressive intraspecific plasticity in some species (directional selection at the same time?)^{48, 97, 108}, (“anti-cryptic selection?”)¹⁶. The explanations of this phenomenon from the point of view of limitations in the origin of forms, the “non-homogeneity” of character space, are more parsimonious.

Variation at Early Ontogenetic Stages

Several attempts have been made to assess directly variation in organisms at the early stages of the ontogenesis (that is, before the time when variants may be eliminated by natural selection). An example of such an analysis is the study of variation of postcranial skeleton (spinal column, sacrum, pectoral and pelvic girdle, limbs) of tailless amphibians^{58, 59, 60}. The results indicate that a) the range of actual variation is considerably more narrow than that of the theoretically calculated potential variation; it is represented by series of discrete variants; b) there exist “prohibited” variants, which are never implemented, and the set of such variants is different in different species; c) under extreme developmental conditions the proportion of anomalous individuals increases but the general set of variants remains the same. Therefore, both the “non-homogeneity” of the character space and the influence of environmental conditions on the frequency of actual anomalous variants is observed.

In many instances hereditary variation is characterized by a considerable discreteness of the manifestation of the entire complexes of characters in an organism^{2, 17}. A theoretical basis of these observations can be found in the ideas about correlative connections in an organism and the canalization of the ontogenetic ways^{109, 125}.

Consistent Formation of Variation at the Molecular Level

Selectogenetic model of evolution is based on the idea that potential hereditary changes are in principle unlimited and multidirectional. However, ideas about systemic organization of genomes^{34, 51, 112, 128} leave little space for interpreting molecular mechanisms underlying hereditary variation as stochastic processes. Failures of matrix processing in prokaryotes and eukaryotes are mitigated by an array of reparation systems^{26, 50, 98}. These systems, in turn, can be fine-tuned and regulated, resulting in regulated changes in the level of mutational variation^{31, 94}. The activity of horizontal transfer in prokaryotes is associated with the systems of DNA reparation. Activation of stress-induced mutagenesis leads to mobilization of conjugative elements, which are used as vectors of genes responsible for antibiotic resistance^{7, 43}.

Much of hereditary variation is associated with

the transfer of mobile genetic elements in the genome of different species^{3, 28, 32, 54, 56, 73}. Their transfer is often directional, sometimes involves large numbers of elements and may result in quite definite hereditary changes¹⁰³.

An impressive array of epigenetic mechanisms involved into the formation of variation provides an insight into the molecular basis of hereditary changes. At the same time, the work of epigenetic systems is essentially non-stochastic. It is a reflection of a system of negative feedbacks in the genome in the narrow sense of the word (that is, a set of species-specific sequences of DNA bases) and other molecular components of the cell associated with the regulation of the protein-synthesis apparatus, the dynamic chromatin structure, "protein" heredity and "small RNA" systems (see a series of reviews in Epigenetics²⁷).

Ideas about the systemic organization of the genetic-epigenetic mechanisms are reflected in the conclusion that the reconstructions of the hereditary apparatus comply to the external influences. It is not so much the matter of correlations between the impact of the environment and the frequency of hereditary changes (see above, regulation of reparation systems) but rather the compliance of molecular reconstructions of the genetic-epigenetic system to this impact. Examples of this compliance are a higher level of mutations in those bacterial sites whose products are involved in the potential compensation of the external influence^{41, 42}; amplification of functional areas of genome associated with the resistance to chemicals in protists¹⁵; specific inserts into genome CRISPR cassettes in bacteria ensuring resistance to bacteriophages^{55, 56, 91}; similar mechanisms in eukaryotes associated with the activity of piRNA and siRNA^{3, 52}; paramutations, whose manifestation also seems to be associated with the mechanisms of RNA interference⁴⁷. All this prompts one to revisit the idea of the direct inheritance of adaptive molecular changes at the epigenetic and even the genome level in the light of molecular mechanisms^{34, 55, 56, 65}.

Nature of Variation: Summing Up

It has been shown that at least in some cases variation is directional and its directionality is sometimes induced (by environmental impacts). Recalling the two

major requirements to hereditary variation, we can make the following conclusion: observations on intra- and interspecies variation point to a considerable "non-homogeneity" of the variation implemented in the character space. The fact that this non-homogeneity, rather than being a *post hoc* result of differential mortality, is associated with the character of variation is proved by: a) the presence of general trends, variation series; b) direct assessments of variation at early stages of ontogenesis; c) the analysis of molecular mechanisms underlying hereditary variation; d) the possibility of direct observation (in case of laboratory models for the study of variation). Moreover, it can be considered as a fact that the frequency of hereditary reconstructions increases under the influence of external factors. There are also indications of correlations between the character of the impacting factor and the type of hereditary reconstructions. The structure of variation is "non-homogenous" in its manifestations at the level of organisms and considerably "non-homogenous" in manifestations of hereditary changes at the level of genome and the associated epigenetic processes.

To conclude, at the imaginary axis of the "homogeneity" of hereditary variation (from entirely random to strictly canalized) its modal values are evidently confined to the area of consistent expression of characters. This entails a conclusion that orthogenetic mechanisms of the formation of variation predominate while natural selection plays a secondary role. Its importance diminishes even further in the context of data indicating that hereditary variation may conform to certain environmental impacts.

Further analysis would focus on the possibility of natural selection "using" hereditary variation. Let us imagine a change in the genotype of an individual as a signal which should correlate with the ability of this individual to make a greater or a smaller contribution into the gene pool of the next generation. The character of variation is no longer of interest to us; the emphasis will be on the consequences of its indisputable presence.

System Filters and the Effectiveness of "Hereditary Signal" for Natural Selection.

Variation determines the range of the morpho-functional variants of individuals of a given population or species. We will consider each change in

the genotype structure as a "signal", which is to be reflected in the characteristics of the individual (regardless of the nature of the inherited reconstructions, be they gene mutations, chromosomal reconstructions, changes associated with ploidy, reconstructions in the course of crossing-over or recombination variation of combinations during sexual process). In the long run, these characteristics should be reflected in the differential contribution of individuals into the gene pool of the next generation. From now on, we will ignore the changes that occur in the genotype but are not manifested, by definition, in the morphological and functional characters of an individual (for instance, synonymous base substitutions).

The "hereditary signal" becomes involved in interactions representing various hierarchical levels of the systemic organization of living nature. Starting at the suborganismic level, the interactions ascend to the biocenotic one. Each level is characterized by specific mechanisms of maintaining systemic integrity; the systemic integrity of the next level comprises all subordinate mechanisms but cannot be reduced to their sum. These holistic notions are reflected in the concept of emergence and can be aphoristically expressed as "the whole is bigger than the sum of its parts" (⁶⁸; see ²² for historical context and development of the notions of emergence into the Synergism Hypothesis).

Hereafter the term "system filter" will be used to denote the system of feedbacks and compensatory regulations at each of the hierarchical systemic levels and the passage of the hereditary "signal" through a series of "system filters" will be considered. Special attention will be given to the stability of the "signal" since only *stable* passage through the filters may result in the implementation of the natural selection mechanism.

A. Filter no. 1. Epigenetic Regulation.

The change in the genotype structure (the "hereditary signal") should be considered in the context of a system of dynamic molecular interactions including the regulation of genes' activity by transcription factors, RNA interference, alternative splicing, DNA methylation and histone acetylation, mobility control of mobile genetic elements. In general, all these mechanisms belong to the system of epigenetic

regulation of the genetic networks ^{27, 126}. The consequences of structural changes in the genome would thus be reflected in multiple compensatory reactions of the epigenetic system of the cell. The systemic character of the complex of these reactions is expressed in the maintenance of a stable whole (the entire molecular-genetic system of the cell) on the basis of the compensatory reactions of its elements (changes of the state of functional blocks of this system).

B. Filter no. 2. Ontogenetic regulation (integrity of the morphoprocess).

Transformed by the epigenetic system of the cell, the "hereditary signal" is involved into a system of dynamic self-organization of the organism. In essence, the functioning of all the mechanisms mentioned above (epigenetic regulation) should be considered in dynamics, that is, in the context of the entire morphoprocess ^{11, 37, 76}. However, there is more to the regulative connections of the morphoprocess than epigenetic regulation. Cell communications and positional information are crucial for the morphoprocess of a multicellular organism, and so are systems determining the cell cycle length, the choice of cell fate, the character of growth and the ontogenetic features of the life cycle stages. On the whole, the implementation of a particular morphoprocess ³⁷ is characterized by a system of correlations, whose interactions can be considered as a dynamic self-organization with a high degree of equifinality.

The changes in the genome structure that fail to ensure the implementation of the ontogenesis would invariably result in the arrest of the morphoprocess and the death of the organism. Other changes involved in the dynamic self-organization of the morphoprocess are part and parcel of the stable functional system.

C. Filter no. 3. Physiological Regulation.

Physiological regulation is a system of mutual influences, including feedbacks, of the organism's parts ensuring its functional integrity. An important feature of this level is the need to ensure effective performance under changing conditions of the environment. This involves: maintenance of the optimal level of metabolism depending on nutrition, breathing, osmoregulation and excretion; adequate intensity of locomotion; effective

functioning of the integrating systems (circulatory system, neuroendocrine system, nervous system). The existence of an organism in a changing environment critically depends on dynamic correlations between these functions, that is, the system of compensations and feedbacks underlying the functional stability of the entire organism¹⁰⁹.

It is physiological regulation that, in combination with the dynamic impacts of the environment, brings out the implemented range of hereditary variation, referred to as phenotypic plasticity. From this viewpoint, the potential effectiveness of the physiological system filter is determined by the range of physiological tolerance and physiological resistance of an individual¹³.

It is evident that the system of physiological regulation would compensate hereditary changes that may negatively affect some physiological parameters. At the same time, a considerable change in the morpho-functional characteristics of an individual would reflect the fact that the "hereditary signal" had passed through the first two system filters and failed to be compensated by the third. Then we may expect an individual to approach the limits of the resistance range. Its viability would decrease considerably and the probability of its death would increase dramatically. However, this would be the case only if the system filters of the higher levels fail, too (see below).

D. Filter no. 4. Behavioural Regulation.

The passage of the "hereditary signal" through epigenetic, ontogenetic and physiological "system filters" would herald the emergence of an individual whose functionality sets it aside from other individuals of the population. Then the filter of the next level comes into play.

Organisms interact with the environment through a system of their behavioural reactions. This system, formed in ontogenesis, is a powerful system filter on the way of the hereditary character to its reflection in the survival/fertility of the progeny of the organism. More or less considerable changes in the functional characteristics of an individual determined by its hereditary features would elicit corresponding behavioural modifications. From the point of view of an overall fitness of an individual, such modifications — behavioural programmes — would act as

compensations improving survival and reproductive success. The resulting behavioural plasticity underlies the effectiveness of a vast range of morpho-functional variants present in the population of the species. Behavioural adaptations may prompt the choice of specific microhabitats and diet preferences, determine activity periods and the character of interactions with conspecific and heterospecifics. Formally, this system filter may be described as ensuring the existence of an organism and the implementation of its major functions in a heterogeneous environment.

The plasticity of behavioural mechanisms underlies the existence of a vast number of functionally different individuals in the population. Because of this plasticity, the diversity of morpho-functional variants in the population cannot be classified into *a priori* more or less successful (or unsuccessful), with the obvious exception of non-viable individuals. Indeed, the realization of these variants in a heterogeneous environment is mediated by behaviour. And, what is more, it is the diversity within the population that assures its stable existence as a system.

All the above "filters" represented systems of the self-organization of an organism (functioning, of course, in the context of environmental conditions). Yet though the systemic characteristics of an organism in the hierarchy of living systems (level of organization^{10, 76, 88}) are expressed most vividly, organisms themselves form part of more complex systems (complexity of organization¹⁰). These supraorganismic systems — populations and communities — are distributive¹¹¹: the degree of their organization is lower than that of their constituting elements. However, they also have some features of systemic organization and so a certain degree of systemic integrity ensured by regulatory mechanisms. Earlier we have highlighted various aspects of systemic organization of an organism. Now it is time to look for systemic properties of more complex systems — populations and communities.

E. Filter no. 5. Populational Compensations

Should the "hereditary signal" pass through all the above compensatory filters, it would not necessarily mean that natural selection would immediately set to work. In this case, too, the "hereditary signal" cannot be directly translated into stable reproductive success

(stable in the series of generations) or failure of the signal's carriers. The potential relative importance of the "hereditary signal" for the reproduction of the population can only be determined in the context of the system of populational regulation. This system can be considered as the "filter" of the fifth level.

At least two groups of system mechanisms ensuring the integrity of the population as a system and its successful reproduction should be taken into account here. 1. A group of mechanisms of "populational mutual assistance" (see, e.g., ⁶¹). 2. A group of mechanisms ensuring populational compensations of abundance (birth and mortality rates, emigration and immigration). Moreover, it should be emphasized that populational compensations (feedbacks) may act during the lifetime of several generations. The impact of frequency-dependent differential mortality is one of such mechanisms ^{5, 110}.

So, the reproductive value of an individual — the carrier of the hereditary signal — may be identified only in the context of the surrounding conspecifics. Its hereditary morpho-functional characteristics (the signal that has passed through filters of levels 1–4) may, of course, correlate with its reproductive success. However, the strength of this correlation will be considerably modified by the system of populational interactions of the individual in question. The signal itself becomes distinctly conditional ¹¹. That is, it can be assessed only in the context of interaction.

F. Filter no. 6. Biocenotic (ecosystem) Compensations.

So far, discussing populational compensations, we have considered only the organism's interactions with conspecifics. This was justified as long as we had to identify the system of dynamic feedbacks at the population level. However, each individual is also involved in biocenotic interactions, and these should be taken into account as well. The passage of the "hereditary signal" through filters 1-4 would not only reserve its carrier a special place in the system of populational regulation (filter of the fifth level) but also, and simultaneously, alter the interactions of this individual with heterospecifics in the community. The change would manifest itself most conspicuously in the interactions of the individual with organisms whose populations form stable biocenotic links in the community (parasitic and predator-prey systems,

mutualistic complexes of species etc.; ⁹). These diverse interactions are characterized by stable feedback-based regulations. A change in the properties of an element (individual with modified characters) would be necessarily expressed in compensatory changes of the interaction with the heterospecifics. From the point of view of the "hereditary signal" under discussion, this biocenotic complex of feedbacks is the filter of the sixth level. Within this filter, the correlation of the hereditary signal (which has passed through filters 1-4) with the reproductive success of an individual may level out. Or else the sign of the correlation may change from plus to minus or vice versa. Importantly, in this case as well as in the case of populational system filter the character is necessarily conditional. Its value may be assessed only in the context of the structure of a concrete biocenotic connexion ⁹, moreover, in the context of a concrete phase of its dynamic state. A change in the properties of an individual becomes an indispensable structural-functional element in the system of regulatory links within the community. In a way, the "hereditary signal" becomes involved in the structure of the supraorganismic system. It is only from this viewpoint that its significance can be assessed.

Conclusion

Two major components should be considered when discussing whether the mechanism of natural selection does act in reality: the structure of variation and the complex of hierarchical "system filters".

As concerns the structure of variation: we can conclude that new hereditary characteristics of individuals fall onto a narrow "strip of land" in the sea of potential possibilities (see Fig. 1). Hereditary variation is non-homogenous, and very much so. This means that it is the characteristics of variation that determine the direction of potential evolutionary changes. It is utopian to think that natural selection has a virtually unlimited range of hereditary variations at its disposal. It does not, and this is due, first of all, to the character in which variation is manifested, its non-homogeneity. We have to admit that in the continuum of possible evolutionary mechanisms ranging from orthogenetic (a narrow range of the potentially possible variation is implemented) to selectogenetic (much of the potential variation is implemented) orthogenesis rules the day (see Fig. 1).

Just how effective can selection be within the range of

implemented variation, narrow as it is? Does dispersion of genotypes within this range correlate directly with differential mortality/contribution into the gene pool of the next generation?

Evidently, no. The consequences of changes in the genotype of an organism are involved into a system of hierarchical multiple compensation, from the molecular to the biocenotic level. For clarity, in this work we have represented this process as passage through a series of "system filters" (Fig. 2). Each filter is represented by multiple feedbacks, compensatory reactions, which maintain the integrity of systems at the epigenetic, ontogenetic, physiological, behavioural, populational and biocenotic level. The first system filters (1-3) are responsible for the integrity of the organism as such. Filter 4 minimizes the costs of interaction of the organism with the environment in the broad sense of the word. Filters 5 and 6 characterize the integrity of supraorganismic systems. The importance of a given hereditary character for the reproduction success of its carrier cannot be assessed because the systems of self-organization at each level alter the value of the "hereditary signal". Strictly speaking, hereditary change is *a priori* neutral, that is, neither positive nor negative. Characters of an organism are involved in the structure and functions of systems of a higher rank. The actual, "current" value of a given character makes sense only in dynamical interaction with the elements of these systems. Taken together, this means: any non-lethal character is conditional. The significance of a character for reproduction is dynamical and impossible to formalize.

I would not want to convey a false impression that high mortality does not occur in natural populations and that all variation is involved in the self-organization of living systems. Clearly, this is not so. Numerous observations indicate that mortality may be high and variable. There are cases, especially among r-strategists, when much less than 1% of the progeny reaches the reproductive stage. The idea that I did want to convey was that mortality is not directly correlated with the character of variation. The "system filters" change any potential correlation beyond recognition, so that a direct correlation of the type "genotype — phenotype" becomes nonsensical. It does make sense, however, for one particular group of phenomena, which we will now

discuss.

Of course, there are characters which, under given external conditions, exceed the limits of stability of the hierarchical systemic organization. These anomalous characters reflect hereditary changes whose consequences cannot be, as a rule, involved into systemic self-organization at any level. They lurk at the periphery of the implemented range of hereditary variants. Individuals with these characters die more often or fail to reproduce properly. The correlation between a given hereditary change and reproductive success (or lack thereof) is evident here. Natural selection may come into play. However, this exception highlights the general rule: the mechanism of natural selection has a small sphere of influence. It amounts to eliminating variants that cannot be involved into correlative systems of any level.

A dramatic change in environmental conditions may open a possibility for the anomalous characters to be involved in the general system of regulation. Viable individuals strikingly different from conspecific ("hopeful monsters") may arise. The plausibility of such "systemic mutations"³³ and their potential evolutionary role are broadly discussed^{39, 57, 116, 124}.

Interestingly, such an approach also works a radical change on our understanding of the conditions most favourable for increasing the rate of evolution. An increased rate may be observed during periods when mortality is low, environmental conditions are favourable and natural selection is down to a minimum. On the contrary, a high mortality and the narrowing of the possible range of forms (that is, a high rate of natural selection) should slow down evolution dramatically. Hereditary changes entailing serious consequences, which cannot be involved into compensatory interactions, result in the death of individuals.

Taken together, the "system filters" described in this work form a unified and dynamic system of hierarchical multiple compensation. This system would "check" all hereditary changes of organisms (to note again, within the limited range of hereditary variation, see — variation). In other words, the consequences of hereditary changes are involved in a hierarchical system of multiple compensations. One may well say that this system and its functioning are self-organizing. Therefore, the adaptive nature of an individual is subject

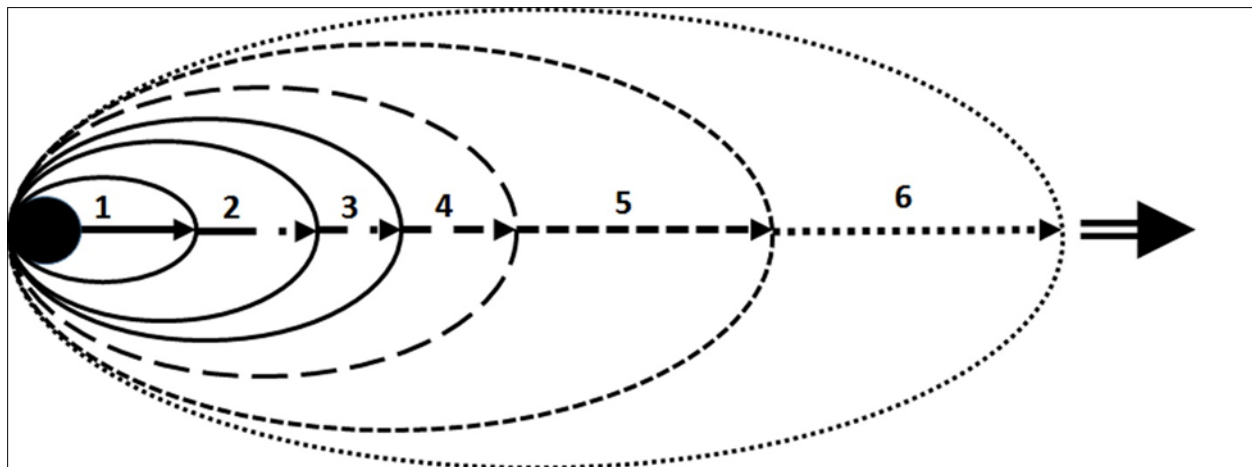


Figure 2. Schematic representation of "system filters" at work. Successively increasing ellipses denote "system filters": 1 – epigenetic; 2 – ontogenetic; 3 – physiological; 4 – behavioural; 5 – populational; 6 – biocenotic. The black circle denotes a conditional change of the hereditary material of the organism. Arrows indicate successive passage of the signal through the system filters. As the signal passes through the filters, the adaptive significance of this hereditary change for the organisms in the structure of its population and community is formed.

to the Law of Multilevel Self-Organization. It is here, in the crucible of "system filters", that the adaptive nature of characters is formed. The variation of hereditary material is mere clay from which it is modelled.

A clear formulation of this law might make the case for the orthogenetic interpretation of evolution. The weakness of the orthogenetic approach is its failure to explain clearly the formation of adaptive characters underlying the fitness of an organism, in other words, purposefulness^{77,78}, see Lyubishchev⁷⁰ for the general discussion of the problem of purpose in evolution). At the same time, an increasingly critical attitude to selectogenetic explanations of the formation of purposefulness begs the question of the relative role of natural selection and self-organization in evolution^{22,35,46,66,86,87,89}. The mechanism suggested in this work implies that the hereditary signal itself (inherited change in the genotype) is not adaptive at all. Adaptive characters are formed in a hierarchical system of filters, from epigenetic to biocenotic.

In the light of the above, what can be said about the potential "creative" role of natural selection in the formation of adaptations and the change of the modal characters of species? We have to admit that the directionality of the evolutionary process is based on the limited range of hereditary variations. At the same time, the distribution of frequencies of different implemented mutations within this range is essentially

non-homogenous. This consideration alone indicates that natural selection cannot determine the direction of the evolutionary process. During the development of an organism with certain hereditary changes the consequences of the "genetic signal" are involved in a multilevel system of correlations, into block reconstructions. If that happens, the correlation between the type of hereditary changes and the certainty of its contribution to the progeny becomes dynamic and conditional. It is so strongly disfigured that natural selection, stripped of its creative role, becomes entirely helpless.

Does natural selection still play a role in the functioning of living systems? Undoubtedly, it does. This is the role of a conservative mechanism eliminating the variants that, under given environmental conditions, cannot be integrated into the hierarchical system of filters. This mechanism prevents the implementation of the entire potential diversity of these systems. However, the significance of this phenomenon lies in a different plane. Therefore, it would be better, for the sake of clarity, to call it "differential mortality" rather than continue using the term "natural selection", strongly linked with the idea of "creative evolutionary potential".

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