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Research Article

The Main Patterns of Individual Adaptation

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Abstract:

With all the variety of individual phenotypic adaptation, its development in higher animals is characterized by certain common features.

There are two stages in the development of most adaptive reactions, namely: the initial stage of urgent but imperfect adaptation and the subsequent stage of perfect, long-term adaptation.

The urgent stage of the adaptive reaction occurs immediately after the onset of the stimulus and, therefore, can be realized only on the basis of ready-made, previously formed physiological mechanisms. The obvious manifestations of urgent adaptation are the animal's flight in response to pain, an increase in heat production in response to cold, an increase in heat transfer in response to heat, an increase in pulmonary ventilation and minute volume of blood circulation in response to lack of oxygen. The most important feature of this stage of adaptation is that the body's activity proceeds at the limit of its physiological capabilities — with almost complete mobilization of the functional reserve — and does not fully provide the necessary adaptive effect. Thus, running of an unadapted animal or human occurs at near-maximum values of the minute volume of the heart and pulmonary ventilation, with maximum mobilization of the glycogen reserve in the liver; due to insufficiently rapid oxidation of pyruvate in muscle mitochondria, the level of lactate in the blood increases. This lacedemia limits the intensity of the load — the motor reaction can be neither fast enough nor long enough.

Keywords: individual adaptation; interrelation; function; systemic structural trace

Introduction

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The urgent stage of the adaptive reaction occurs immediately after the onset of the stimulus and, therefore, can be realized only on the basis of ready-made, previously formed physiological mechanisms. The obvious manifestations of urgent adaptation are the animal's flight in response to pain, an increase in heat production in response to cold, an increase in heat transfer in response to heat, an increase in pulmonary ventilation and minute volume of blood circulation in response to lack of oxygen. The most important feature of this stage of adaptation is that the body's activity proceeds at the limit of its physiological capabilities — with almost complete mobilization of the functional reserve — and does not fully provide the necessary adaptive effect. Thus, running of an unadapted animal or human occurs at near-maximum values of the minute volume of the heart and pulmonary ventilation, with maximum mobilization of the glycogen reserve in the liver; due to insufficiently rapid oxidation of pyruvate in muscle mitochondria, the level of lactate in the blood increases. This lacedemia limits the intensity of the load — the motor reaction can be neither fast enough nor long enough.

Thus, adaptation is implemented "from the spot", but it turns out to be imperfect.

Quite similarly, when adapting to new complex environmental situations implemented at the brain level, the stage of urgent adaptation is carried out due to ready-made pre-existing mechanisms and is manifested by a well-known period of "generalized motor reactions" or "period of emotional behavior" in the physiology of higher nervous activity. At the same time, the necessary adaptive effect, dictated by the body's needs for food or self-preservation, can to remain unfulfilled or to be provided by an accidental successful movement, i.e. it is unstable.

The long-term stage of adaptation occurs gradually, as a result of prolonged or repeated exposure to environmental factors. In essence, it develops on the basis of repeated implementation of urgent adaptation and is characterized by the fact that as a result of the gradual quantitative

accumulation of some changes, the body acquires a new quality - it turns from an unadapted into an adapted one. This is an adaptation that ensures that the body performs physical work that was previously unattainable in its intensity, the development of the body's resistance to significant altitude hypoxia, which was previously incompatible with life, the development of resistance to cold, heat, and large doses of poisons, the introduction of which was previously incompatible with life. This is also a qualitatively more complex adaptation to the surrounding reality, which develops in the process of learning based on brain memory and manifests itself in the emergence of new stable temporary connections and their implementation in the form of appropriate behavioral reactions.

Comparing the urgent and long-term stages of adaptation, it is not difficult to conclude that the transition from an urgent, largely imperfect stage to a long-term one marks a pivotal moment in the adaptation process, since it is this transition that makes it possible for an organism to live in new conditions, expands its habitat and freedom of behavior in a changing environment.

It is advisable to consider the mechanism of this transition on the basis of the idea accepted in physiology that the body's reactions to environmental factors are provided not by individual organs, but by systems organized and subordinated to each other in a certain way. This idea allows us to state that the reaction to any new and sufficiently strong environmental impact — to any violation of homeostasis — is provided, firstly, by a system specifically responding to this stimulus, and, secondly, by stress-realizing adrenergic and pituitary-adrenal systems that respond non-specifically to a variety of changes in the habitat.

Using the concept of "system" in the study of phenotypic adaptation, it is advisable to emphasize that in the past, the closest thing to revealing the essence of such systems that provide a solution to the main task of an organism at a certain stage of its individual life was the creator of the doctrine of the dominant, one of the greatest physiologists of our century, Ukhtomsky. He studied in detail the role of internal, hormonal needs of the body, the role of entero- and extroceptive afferent signaling in the formation of dominants, and at the same time considered The dominant as a system is a constellation of nerve centers that subordinate the executive organs and determine the direction of the body's behavioral reactions — its vector.

Ukhtomsky wrote: "The external expression of a dominant is a certain work or working position of the body, supported at the moment by various stimuli and excluding other work and postures for the moment. For such work, or sometimes it is necessary to assume that not a single local focus is aroused, but a whole group of centers, perhaps widely scattered in the nervous system. The sexual dominant hides the excitation of the centers in the cortex, and in the subcortical apparatuses of vision, hearing, smell, touch, in the medulla oblongata, and in the lumbar parts of the spinal cord, and in the secretory, and in the vascular system. Therefore, it must be assumed that behind each natural dominant lies the excitation of a whole constellation (constellation) of centers. And the holistic dominant needs to distinguish primarily between cortical and somatic components" [1].

Developing the idea that the dominant unites nerve centers and executive organs located at various levels, Ukhtomsky sought to emphasize the unity of this newly emerged system and often called the dominant an "organ of behavior."

"Every time," he noted, "there is a dominant symptom complex, there is also a certain vector of behavior. And it is natural to call it an "organ of behavior," although it is mobile, like Descartes' vortex motion. Defining the term "organ" as, I would say, a dynamic, mobile actor, or a working combination of forces: I think it is extremely valuable for a physiologist" [1]. Later, Ukhtomsky took the next step, designating the dominant as a system, he wrote: "From this point of view, the principle of dominance can naturally be stated as an application to an organism of the beginning of possible movements, OR as a general and at the same time very specific expression of those conditions that transform a group of more or less disparate bodies into a fully connected system acting as a mechanism with an unambiguous action" [1].

Considering the transition from urgent adaptation to long-term adaptation in terms of the concept of a functional system, it is not difficult to notice an important, but not always properly taken into account, circumstance, which is that the presence of a ready-made functional system or its formation in itself does not mean sustainable, effective adaptation.

Indeed, the initial effect of any unconditional stimulus that causes a significant and prolonged motor reaction is to excite the corresponding afferent and motor centers, mobilize skeletal muscles, as well as blood circulation and respiration, which together form a single functional system specifically responsible for the implementation of this motor reaction. However, the effectiveness of this system is low (running can be neither long nor intensive- it becomes so only after repeated repetitions of a situation that mobilizes the functional system, i.e. E. after training, which leads to the development of long-term adaptation).

Under the influence of lack of oxygen, the effect of hypoxemia on chemoreceptors, directly on nerve centers and executive organs entails a reaction in which the role of a functional system specifically responsible for eliminating lack of oxygen in the body is played by the circulatory and respiratory organs that are regulatively connected and perform an increased function. The initial result of the mobilization of this functional system after the rise of an unadapted person to an altitude of 5,000 m is that cardiac hyperfunction and pulmonary hyperventilation are very pronounced, but nevertheless they are insufficient to eliminate hypoxemia and are combined with more or less pronounced adynamia, apathy or euphoria, and eventually with a decrease in physical and intellectual performance.. In order for this urgent but imperfect adaptation to be replaced by a perfect, long-term one, it is necessary to stay at altitude for a long time or repeatedly, i.e. prolonged or repeated mobilization of the functional system responsible for adaptation.

Quite similarly, when a poison, such as nembutal, is introduced into the body, the role of a factor specifically responsible for its destruction is played by the mobilization of the microsomal oxidation system localized in liver cells. Activation of the microsomal oxidation system undoubtedly limits the damaging effect of the poison, but does not eliminate it completely. As a result, the intoxication pattern is quite pronounced and, accordingly, the adaptation is imperfect. Subsequently, after repeated administration of nembutal, the initial dose ceases to cause intoxication. Thus, the availability of a ready-made functional system responsible for adaptation to this factor, and instant activation this system by itself does not mean instant adaptation.

When more complex environmental situations affect the body (for example, previously unknown stimuli — danger signals — or situations that arise in the process of learning new skills), there are no ready-made functional systems in the body that can provide a response that meets the requirements of the environment.

The body's response is provided by the already mentioned generalized orientation response against a background of sufficiently severe stress. In such a situation, some of the numerous motor reactions of the body turn out to be adequate and receive reinforcement. This becomes the beginning of the formation of a new functional system in the brain, namely, a system of temporary connections, which becomes the basis for new skills and behavioral reactions. However, immediately after its occurrence, this system is usually fragile, it can be erased by inhibition caused by the emergence of other behavioral dominants that are periodically realized in the body's activity, or extinguished by repeated non-reinforcement, etc.

In order to develop a stable, guaranteed adaptation in the future, it takes time and a certain number of repetitions, i.e., the consolidation of a new stereotype. In general, the meaning of the above is that the presence of a ready-made functional system with relatively simple adaptive reactions and the emergence of such a system with more complex reactions implemented at the level of the cerebral cortex do not in themselves lead to the instant emergence of stable adaptation, but are the basis for the initial, so-called urgent, imperfect stage of adaptation. For the transition of urgent adaptation to a guaranteed, long-term one, some important process must be implemented within the emerging functional system, ensuring that the existing adaptation systems are fixed and their capacity is increased to a level dictated by the environment. [2]

In recent decades, it has been found that an increase in the function of organs and systems naturally entails activation of the synthesis of nucleic acids and proteins in the cells that form these organs and systems. Because in response to the demands of the environment, the function of the systems responsible for adaptation increases, that is where the activation of the synthesis of nucleic acids and proteins develops first of all.

Activation leads to the formation of structural changes that fundamentally increase the capacity of the systems responsible for adaptation. This is the basis for the transition from urgent adaptation to long—term adaptation - a crucial factor in the formation of the structural basis for long-term adaptation.

The sequence of phenomena during the formation of long-term adaptation is that an increase in the physiological function of cells in the systems responsible for adaptation causes, as the first shift, an increase in the rate of RNA transcription on the structural DNA genes in the nuclei of these cells.

An increase in the amount of messenger RNA leads to an increase in the number of ribosomes and polysomes programmed with this RNA, in which the process of cellular protein synthesis proceeds intensively. As a result, the mass of structures increases and the cell's functional capabilities increase, a shift that forms the basis for long—term adaptation.

It is essential that the activating effect of increased function, mediated through the mechanism of intracellular regulation, is attributed specifically to the genetic apparatus of the cell. The introduction of actinomycin to animals, an antibiotic that attaches to guanylic DNA nucleotides and makes transcription impossible, deprives the genetic apparatus of cells of the ability to respond to an increase in function. As a result, the transition from urgent adaptation to long-term adaptation becomes impracticable: adaptation to physical stress, hypoxia, the formation of new temporary bonds and other adaptive reactions turn out to be impossible under the action of non-toxic doses of actinomycin, which do not disrupt the implementation of ready-made, previously established adaptive reactions. [3]

Based on these and other facts, the mechanism by which the function regulates the quantitative parameter of the activity of the genetic apparatus, the rate of transcription, was designated by us as "the relationship between the function and the genetic apparatus of the cell" [3]. This relationship is two-way. The direct connection is that the genetic apparatus — the genes located in the chromosomes of the cell nucleus, indirectly through the RNA system, provide protein synthesis — "make structures", and structures "make" a function. The feedback is that the "intensity of the functioning of structures" — the amount of function per unit mass of an organ - somehow controls the activity of the genetic apparatus.

It turned out that an important feature of the process of hyperfunction — hypertrophy of the heart with narrowing of the aorta, a single kidney or lung after removal of a paired organ, a lobe of the liver after removal of its other lobe - is that activation of the synthesis of nucleic acids and protein, which occurs in the coming hours and days after the onset of hyperfunction gradually stops after the development of hypertrophy and an increase in the mass of the organ.

This dynamic is determined by the fact that at the beginning of the process, hyperfunction is carried out by a non-hypertrophied organ and an increase in the amount of function per unit mass of cellular structures causes activation of the genetic apparatus of differentiated cells. After the complete development of organ hypertrophy, its function is distributed in an increased mass of cellular structures, and as a result, the amount of function performed by a unit of mass of structures returns or approaches the normal level. After that, the activation of the genetic apparatus stops, and the synthesis of nucleic acids and proteins also returns to normal levels [4].

If you eliminate the hyperfunction of an organ that has already undergone hypertrophy, the amount of function performed by 1 g of tissue will become abnormally low. As a result, protein synthesis in differentiated cells will decrease and the mass of the organ will begin to decrease. Due to the decrease in the organ, the amount of function per unit mass gradually increases, and after it becomes normal, the inhibition of protein synthesis in the cells of the organ stops — its mass no longer decreases.

These data provided the basis for the idea that in differentiated cells and mammalian organs formed by them, the amount of function performed by a unit of organ mass (intensity of functioning of structures - IFS) plays an important role in regulating the activity of the cell's genetic apparatus. An increase in the IFS corresponds to a situation where "functions are closely structured." This causes activation of protein synthesis and an increase in the mass of cellular structures. A decrease in this parameter corresponds to a situation where "the functions are too spacious in the structure," resulting in a decrease in synthesis intensity followed by the elimination of excess structure. In both cases, the intensity of the functioning of the structures returns to a certain optimal value, characteristic of a healthy organism.

Thus, the intracellular mechanism, which implements a two-way relationship between the physiological function and the genetic apparatus of a differentiated cell, ensures that IFS is both a determinant of the activity of the genetic apparatus and a physiological constant, maintained at a constant level due to timely changes in the activity of this apparatus [4].

It is clear that with the same duration of the average daily activity, i.e., with the same time during which the organ operates, the average daily IFS will be higher for an organ that functions at a higher level.

Thus, in a healthy body, the stress developed by the myocardium of the right ventricle is somewhat less than the stress developed by the myocardium of the left ventricle, and the duration of ventricular function during the day is equal; accordingly, the content of nucleic acids and the intensity of protein synthesis in the myocardium of the right ventricle is also less than in the myocardium of the left [5]. There is also evidence that the different intensity of functioning of structures that develop in different tissues during ontogenesis affects not only the intensity of RNA synthesis in DNA structural genes and, through RNA, the intensity of protein synthesis. It turned out that IFS acts more deeply, namely, it determines the number of DNA matrices per unit of tissue mass, i.e. the total the power of the genetic apparatus of the cells forming the tissue, or the number of genes per unit mass of tissue. This effect was manifested in the fact that the DNA concentration for the left ventricular muscle is 0.99 mg/g, for the right ventricular muscle — 0.93, for the diaphragm — 0.75, for skeletal muscle — 0.42 mg/g, i.e. the number of genes per unit mass varies in different types of muscle tissue in proportion to IFS. The number of genes is one of the factors determining the intensity of RNA synthesis. Accordingly, in further experiments, the researchers found that the intensity of RNA synthesis, determined by the inclusion of labeled carbon glucose 14C, is 3.175 beats/min for the left ventricle, 3.087 for the right ventricle, 2.287 for the diaphragm, and 1.154 beats/min for the skeletal muscle of the limb per amount of RNA contained in 1 g of muscle fabrics[5].

Thus, IFS, which develops during ontogenesis in young animals whose cells have retained the ability to synthesize DNA and divide, can determine the number of genes per unit mass of tissue and indirectly the intensity of RNA and protein synthesis, i.e., the perfection of structural support for cell function.

The above clearly indicates that the relationship between the function and the genetic apparatus of the cell, which we will refer to as the relationship G ⇄ F, is a permanent mechanism of intracellular regulation that is implemented in cells of various organs.

At the stage of urgent adaptation, in case of hyperfunction of the system specifically responsible for adaptation, the implementation of G ⇄ F lawfully ensures activation of the synthesis of nucleic acids and proteins in all cells and organs of this functional system. As a result, certain structures accumulate there — a systemic structural trace is realized [5].

Thus, when adapting to physical exertion, pronounced activation of nucleic acid and protein synthesis naturally occurs in neurons of motor centers, adrenal glands, skeletal muscle cells, and heart cells, and pronounced structural changes develop [6]. The essence of these changes is that they provide a selective increase in the mass and power of structures responsible for management, ion transport and energy supply.

It has been established that moderate cardiac hypertrophy is combined with increased activity of the adenyl cyclase system during adaptation to physical exertion [5] and an increase in the number of adrenergic fibers per unit mass of the myocardium [6]. As a result, the adrenoreactivity of the heart and the possibility of its urgent mobilization increase. At the same time, an increase in the number of H-chains, which are carriers of ATPase activity, is observed in the myosin heads. ATPase activity increases, resulting in an increase in the rate and amplitude of contraction of the heart muscle [6]. Further, the power increases the calcium pump of the sarcoplasmic reticulum and, as a result, the rate and depth of diastolic relaxation of the heart [5]. In parallel with these shifts in the myocardium, there is an increase in the number of coronary capillaries [7], an increase in the concentration of myoglobin and the activity of enzymes responsible for the transport of substrates to the mitochondria [7], and the mass of the mitochondria themselves increases. This increase in the power of the energy supply system naturally leads to an increase in the resistance of the heart to fatigue and hypoxemia [6].

Such a selective increase in the power of structures responsible for control, ion transport and energy supply is not an original property of the heart, it is naturally realized in all organs responsible for adaptation. In the process of adaptive response, these organs form a single functional system, and the structural changes developing in them represent a systemic structural trace that forms the basis of adaptation.

In relation to the process of adaptation to physical exertion, this systemic structural trace at the level of nervous regulation is manifested in hypertrophy of neurons of motor centers, an increase in the activity of respiratory enzymes in them [7]; endocrine regulation — in hypertrophy of the adrenal cortex and medulla [8]; executive organs — in hypertrophy of skeletal muscles and an increase in the number of mitochondria in them 1.5—2 times [6]. The latter shift is of exceptional importance, since in combination with an increase in the power of the circulatory and respiratory systems, it provides an increase in the aerobic capacity of the body (an increase in its ability to utilize oxygen and carry out aerobic ATP resynthesis), necessary for the intensive functioning of the movement apparatus. As a result of an increase in the number of mitochondria, an increase in the aerobic capacity of the body is combined with an increase in the ability of muscles to utilize pyruvate, which is formed in increased quantities during exercise due to the activation of glycolysis. This prevents an increase in lactate concentration in the blood of adapted people [8]. An increase in lactate concentration is known to be a factor limiting physical activity, however, lactate is a lipase inhibitor, and, accordingly, lactemia inhibits the use of fats. With advanced adaptation, an increase in the use of pyruvate in the mitochondria prevents an increase in the concentration of lactate in the blood, ensures the mobilization and use of fatty acids in the mitochondria, and ultimately increases the maximum intensity and duration of work.

Consequently, the branched structural trace expands the link that limits the body's performance, and this is exactly how in this way, we form the basis for the transition of an urgent but unreliable adaptation into a long-term one.

Quite similarly, the formation of a systemic structural trace and the transition of urgent adaptation to long-term adaptation occur with prolonged exposure to high-altitude hypoxia compatible with life. Adaptation to this factor is characterized by the fact that the initial hyperfunction and subsequent activation of the synthesis of nucleic acids and proteins simultaneously cover many body systems and, accordingly, the resulting systemic structural trace turns out to be more extensive than during adaptation to other factors. Indeed, hyperventilation is followed by activation of the synthesis of nucleic acids and proteins and subsequent hypertrophy of the neurons of the respiratory center, respiratory muscles and the lungs themselves, in which the number of alveoli increases. As a result, the power of the external respiration apparatus increases, the respiratory surface of the lungs and the oxygen utilization coefficient increase, and the efficiency of the respiratory function increases. In the

hematopoiesis system, activation of the synthesis of nucleic acids and proteins in the bone marrow causes increased formation of red blood cells and polycythemia, which increases the oxygen capacity of the blood. Finally, activation of the synthesis of nucleic acids and proteins in the right and, to a lesser extent, the left parts of the heart ensures the development of a complex of changes, in many ways similar to those that occur during adaptation to physical exertion. As a result, the functional capabilities of the heart, and especially its resistance to hypoxemia, increase.

Synthesis is also activated in systems whose function is not enhanced, but, on the contrary, impaired by oxygen deficiency, primarily in the cortex and the underlying parts of the brain. This activation, as well as the activation due to increased function, is apparently caused by an ATP deficiency, since the G ⇄ F relationship is realized. It should be noted here that the activation of nucleic acid and protein synthesis under consideration, which develops under the influence of hypoxia in the brain, becomes the basis for vascular growth, a steady increase in glycolysis activity, and thus contributes to the formation of a systemic structural trace that forms the basis for adaptation to hypoxia.

The result of the formation of this systemic structural trace and adaptation to hypoxia is that adapted people acquire the opportunity to carry out such physical and intellectual activity in conditions of oxygen deficiency, which is excluded for the unadapted[9].

When adapting to certain factors, the systemic structural footprint turns out to be spatially very limited — it is localized in certain organs. Thus, when adapting to increasing doses of poisons, activation of the synthesis of nucleic acids and proteins in the liver naturally develops. The result of this activation is an increase in the power of the microsomal oxidation system, in which cytochrome 450P plays a major role. Externally, this systemic structural trace can be manifested by an increase in liver mass, it forms the basis of adaptation, which is expressed in the fact that the body's resistance to poisons such as barbiturates, morphine, alcohol, nicotine increases significantly [10].

The effect of the power of the microsomal oxidation system on the body's resistance to chemical factors is apparently very high. Thus, it has been shown that after smoking one standard cigarette, the concentration of nicotine in the blood of non—smokers is 10-12 times higher than that of smokers, whose microsomal oxidation system capacity is increased and on this basis, adaptation to nicotine has formed.

With the help of chemical factors that inhibit the microsomal oxidation system, it is possible to reduce the body's resistance to any chemical substances, in particular to drugs, and with the help of factors that induce an increase in the power of microsomal oxidation, it is possible, on the contrary, to increase the body's resistance to a variety of chemicals.

Limited localization often has a structural trace when the body adapts to damage, namely, when compensating for the removal or disease of one of the paired organs: kidneys, lungs, adrenal glands, etc. In such situations, the hyperfunction of the only remaining organ through the GLF mechanism leads, as indicated, to the activation of the synthesis of nucleic acids and proteins in its cells. Further, as a result of hypertrophy and hyperplasia of these cells, pronounced hypertrophy of the organ develops, which, due to an increase in its mass, acquires the ability to realize the same load that two organs previously realized [11].

Conclusion

Consequently, the systemic structural trace forms the general basis of various long-term reactions of the body, but at the same time, adaptation to various environmental factors is based on systemic structural traces of various localization and architecture.

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