
REVIEWS AND THEORETICAL ARTICLES

*On the 80th anniversary of publication of M.E. Lobashev's article
“Physiological (Paranecrotic) Hypothesis of the Mutation Process” in 1947.*

Stress, Signaling Heredity, and the Physiological Hypothesis of the Mutation Process

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Abstract—The concepts of the “physiological” nature of the mutation process began to emerge in Russian genetics in the 1940s. By 1947, Russian geneticist M.E. Lobashev had formulated a physiological hypothesis for the process of mutation. Its confirmation initiated the development of quite a number of research areas, which progressed to varying levels of success. For example, the role of the animal nervous system in regulating genome function was only studied very narrowly, if at all. Even now, research is limited primarily to the induced epigenetic modifications and their role in gene activity regulation. The role of the central nervous system in maintaining genome integrity, preserving its structure and the nucleotide sequence of body cells is still understudied. Meanwhile, as early as the early 1970s, information appeared about the influence of the nervous system of laboratory animals and its higher regions on the frequency of induced chromosomal aberrations in somatic cells. Later, the nervous system was shown to influence the frequency of crossing over, as well as the frequency of univalent formation in meiosis. It means that the nervous system of animals can restructure and disintegrate the genome to varying degrees. Stress, an integral component of which in mammals is a specific state of the nervous system, particularly the hypothalamic–pituitary–adrenal axis, also influences genetic processes. Given the ever-increasing pressure of stressors on the genome regulation and integrity, the latter deserves special attention. It is the structural integrity of genomic DNA that ensures the safekeeping of genetic information. Depending on stress, it can be easily disrupted, especially in excitable individuals reacting to stressors (including artificially created, often false ones), causing various genetically determined consequences. The high saturation of the environment with “conditioned reflex” stressors of anthropogenic origin (both true and perceived) induces the development of stress and various severe post-traumatic disorders in human populations. Furthermore, the relationship between the central nervous system and genetic consequences is underestimated and understudied. A significant portion of these consequences could easily be avoided by preventing the creation of perceived stressors.

Keywords: mutation process, animals, central nervous system, stressor, reflex, post-stress disorders

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INTRODUCTION

Rapid progress in the 21st century causes various stressors that put pressure on the human psyche. Stress can be caused by: job, family, economic and socio-cultural pressure, health problems. Any of these and many other causes can lead to “physiological” changes in the work of different systems of the human body. The effects of such stressors are manifested both at physiological and at the mental, behavioral and social levels [1–3]. Throughout life, any person can experience adverse and even traumatic events. According to

the review of the World Mental Health Consortium [4], 70% of 68 thousand respondents from 24 countries informed about the occurrence of traumatic events (stressors) in their lives, four or more events for more than 30% of them. Of the 29 types of traumatic events identified, five most frequent events (life-threatening diseases or injuries, sudden death of a beloved person, abuse, and robbery) were experienced by almost 52% subjects.

Such events are prerequisites for the formation of stress and post-traumatic stress disorders (PTSD),

which is associated with mental and physical health disorders. However, still hardly anyone mentions stress-induced genetic disorders in human and animal organisms. There are a lot of studies concerning epigenetic changes, which regulate the work of stress-sensitive genes, and very few works on the mutagenic effect of stress. In the meantime, the evidence of the effect of the nervous system on genetic processes, including mutagenesis, was obtained in the early 70s of the past century [5]. Moreover, the evidence of the involvement of higher-level parts of the brain in regulation of not only the mitotic index but also the mutation process was obtained in laboratory mice [5, 6]. Since stress in mammals is a special state of the nervous system, the present work will compare the data on stress and the role of the nervous system in the mutation process.

DEVELOPMENT OF CONCEPTS OF THE EMERGENCE OF MUTATIONS AS A *PHYSIOLOGICAL* PROCESS

Back in 1927, at the 5th International Genetic Congress in Berlin, Herman Meller in his report "Problem of Gene Variability" hypothesized that "... the agents that affect chemical reactions (*within the body—author's note*) can cause changes in genes per se" [7]. This hypothesis was based on the study of temperature effects on *Drosophila* individuals (*Dr. melanogaster*).

In 1940, Yu. Ya. Kerkis wrote in his article about the effect of physiological condition of the body on the emergence of mutations, based on the previous data obtained by Russian and foreign scientists [8]. In spite of the fact that the molecular structure of genes was not determined at that time, the authors understood even then that: "... the question about the nature of mutations and the causes of their emergence is, given the current state of science, the problem of intracellular physiology." He wrote: "... the cause is the entire diversity of forms and ways of interaction between gene substance and its environment, understood in the broadest sense of the word, i.e., beginning from the medium directly contacting the gene and ending with external environment for the entire organism as a whole. The change in the intracellular biochemical balance as a result of impaired metabolism (or any other element of the physiological state of a cell) induced by any factor is probably the major and most common cause of alterations in the hereditary substance" [8].

In 1946, Russian geneticist M.E. Lobashev defended the dissertation for the Doctor of Science Degree, presenting a physiological hypothesis of the mutation process. The author generalized many-year studies of the mutation process by Russian and foreign researchers. He considered mutations as a result of damaging effects of external conditions, which depended on sensitivity of the body and its individual cells to the latter [9].

The damage (depending on physiological condition of the body) could be realized as a mutation or could be restored to the initial state, i.e., repaired. Thus, establishment of a mutation was a complex physiological process closely related to the metabolic state of cells and interacting organismic systems [9]. Some pathways of mutagenesis, among the variety of possible ones mediated by various systems of the body, are still understudied.

The physiological theory of M.E. Lobashev resulted in more intensive domestic studies of mutagenesis in the field referred to as "physiological genetics" (see, e.g., compilations "Physiological Genetics" [10] and "Physiological Genetics and Behavioral Genetics" [11]). Further extension and generalization of provisions of the theory at different times was highlighted in the reviews by N.N. Khromov-Borisov [12], S.G. Inge-Vechtomov [13, 14], and A.S. Zhuk et al. [15].

ROLE OF THE NERVOUS SYSTEM IN THE REGULATION OF GENETIC PROCESSES

The development of provisions of the physiological (paranecrotic) hypothesis of the mutation process [9] proceeded in different directions. One of the them in my view, underestimated by researchers, concerned the effect of the central nervous systems of animals (in particular, its higher-level parts) on genetic processes. I.P. Pavlov's works on the development of conditioned reflexes in animals can be considered the prerequisites of such trend. The research data, both his own and obtained by his numerous followers, convincingly show that all processes in the body (including the development of germ cells), metabolism and even "intracellular changes are directly dependent on the tone of the cerebral cortex" "via cortical regulation" [16].

Touching upon the problem of intracellular mechanisms of unconditioned or conditioned reflex salivation in dogs (despite the difference in the highways of the brain: via the medulla oblongata or cerebral hemispheres, respectively) [17], today it is impossible to imagine that this occurs without *regulatory* changes in the work of the genome, at least in salivary gland cells. Adaptive functional changes in the work of different organs and tissues within the body directly depend on the respective changes in the work of cells and their genomes [18]. The modern reviews of epigenetic data confirm the concepts of transcription regulation by environmental impacts and even of transgenerational epigenetic effects [19–22]. It is clear that any environmental impact cause both specific and nonspecific changes in the cells of the animal nervous system. Such changes (in particular, modulation of the activity of DNA methyltransferases, histone deacetylases (HDACs), TET dioxygenases) mediate the emerging epigenetic (regulatory) modifications of cellular genomes, which are represented at different levels, right up to the behavior of a recipient stressed organ-

ism [23]. All these data confirm the provision stated as early as in 1973 “that one of the significant functions of the nervous system is regulation of the state of the genetic apparatus by the principle of feedback according to the current needs of the body, environment and individual experience” [5].

ROLE OF THE CENTRAL NERVOUS SYSTEM IN *STRUCTURAL* REORGANIZATION OF THE GENOME IN ANIMALS

However, the role of the nervous system and its central parts in the induction of *structural* changes in the genome was studied much less frequently. Induction of the latter changes with the involvement of the nervous system was demonstrated in the works of G.G. Polyanskaya and R.I. Tsapygina (M.E. Lobashev's coworkers at the Department of Genetics and Selection of the Leningrad State University) in the studies (respectively) on the denervation of organs and the development of conditioned reflexes [5]. For example, preganglionic sympathectomy performed on one eye of a laboratory mouse not only changed the dynamics of the mitotic index in the cells of this eye, but also influenced realization of chromosomal aberrations in its tissues.

Researchers have also developed a conditioned reflex to an indifferent signal, blue-green light, in combination with an unconditioned stimulus: electric current. The latter per se caused a decrease in the mitotic index and in the frequency of X-ray-induced chromosome aberrations. A similar effect was observed when only the conditioned (the former indifferent) signal was switched on. Differentiating signal (green light), the response to which was quenched, did not cause any cytogenetic effects. The study clearly showed both the role of the sympathetic nervous systems and individual experience (the higher parts of CNS) in the regulation of genetic and cytogenetic processes [5].

In 1983, the effects of natural olfactory stressors on disintegration of the genome of male germ cells assessed by the level of chromosome aberrations in meiosis was shown in laboratory mice [24, 25]. The effect of emotional stress on crossover frequency in mice was also shown [26]. This fact correlates with the data on the increased frequency of chromosome aberrations and sister chromatid exchanges in rats exposed to stress [27, 28]. Later, similar effects (the increase in the frequency of chromosome aberrations) were revealed in the dividing cells of bone marrow in males. Some sex-specific “pheromonal” signals demonstrated an “anti-stress” (antimutagenic) effect in individuals of the opposite gender. The absence of direct contact (except for olfactory) with pheromones and the low concentration of stressors used, close to the natural one, made the assumption of direct genotoxic effect unlikely. Disappearance of cytogenetic effects of the used olfactory impacts in the cells of peripheral

organs during temporal inactivation of the olfactory epithelium of the nasal cavity of animals, as well as functional changes in olfactory bulbs [29], confirm the involvement of the central nervous system in the regulation of genome integrity of animal cells.

The nerve cells of the forming brain of rat embryos exhibited a relationship between genetic processes such as the formation of chromosome aberrations and proliferative activity with chromatin structure, depending on the excitability threshold of rats of the two contrasting lineages [30]. The induction of reparative synthesis, the formation of chromosome aberrations and DNA fragmentation in human cells demonstrate that the emotional stress caused by social factors significantly modulates these processes [31, 32]. It should be noted that cytogenetic evaluation of the frequency of chromosome aberrations demonstrates only part of the realized mutagenic activity of the nervous system and its central parts, which can be relatively easily observed visually.

There are also interesting data obtained by other researchers [33], who exposed male Sprague Dawley rats (recipients) at the age of six weeks to a complex of stressor signals (visual, acoustic, and chemosignals) from “donor rats” that had experienced a 5- or 10-h exposure to electric current (foot shock, 2 mA, 10- or 110-s intervals between the pulses, respectively) for 4 days. It was shown that the stressors (the design of experimental facility excluded direct contact with the “donor” animals) increased the level of 8-hydroxy-2'-deoxyguanosine in the DNA of hepatocyte nuclei 1.5–2 times. This is the evidence of oxidative damage to the nuclear DNA in rat liver cells under the exposure of the CNS of recipient animals not to an unconditioned stressor (electric current) but to conditioned signals already mediated by a stressed organism. Such increase is comparable with the effect of intraperitoneal injection of the known hepatocarcinogen, 2-nitropropane (100 mg/kg). At the same time, a rapid drop in the level to the control parameters suggests active repair in rat liver cells after the psychogenic impact ended. The authors assume that the repeating and/or long-term stress exposure can lead to the emergence of point mutations inducing cancer diseases, especially in case of disorders of repair mechanisms [33]. Stress-induced mutagenesis in mice may be associated with the altered activity of cytochrome P450 [34]. Detection of the enhanced comet frequency in the cells of different organs in mice and rats after stress exposure [35, 36] suggests structural reorganization of the cellular genome (fragmentation, appearance of alkali-labile sites) and its enhanced exposure to mutagenic factors.

The works of M.E. Lobashev and his colleagues [16, 37, 38] in conditioned reflex biology and genetics made it possible to develop and effectively use the conditioned-reflex technique for demonstration of the involvement of higher parts of animal CNS in the reg-

ulation of mutation process. However, the genetic significance of the mechanism for creating temporary connections (i.e., conditioned reflex) has not been understood completely until now [39].

Conditioned reflex (CR) can be defined as an organismal response (analogous to the unconditioned one) to the previously indifferent stimulus after the repeated combined exposure to indifferent and unconditioned stimuli. At the same time, especially during the development of CR under laboratory conditions, the unconditioned stimulus (electric current, forced swimming, immobilization, etc.) is often a rather strong stress factor. At the genetic level, yet it has been poorly studied what changes are caused by the stress induced by a conditioned stimulus only (in other words, a “conditioned stressor”). It can only be assumed that the effect will depend on the unconditioned stimulus used, the efficiency of learning, and the time after its completion. If the unconditioned stimulus is a real stressor, then the conditioned (indifferent) one will transform into the same stressor (with all the ensuing consequences, both unspecific and specific).

SIGNIFICANCE OF STRESS IN ANIMAL AND HUMAN LIFE

Although the concept of stress in unicellular organisms is different from the classical one (in mammals), in essence, it is an *overstrained* state of cells (their inability to adapt to environmental changes), which in multicellular organisms underlies the formation of an organismal stress. First, relatively simple model objects (various types of both pro- and eukaryotes) were used to demonstrate the role of stressors (unconditioned damaging environmental factors) in regulating the rate of mutation process and adaptivity. Stable terms such as “stress-induced mutagenesis” (SIM) and “stress-induced evolution” (SIE) have emerged [40, 41].

In mammals, stress implies the emergence of a special state of both central and peripheral nervous systems, in particular, activation of the hypothalamic–pituitary–adrenal axis. Therefore, it would be logical to assume that it exerts a significant effect on genetic processes in the bodies of stressed animals and humans. However, stressors in highly organized animals may be both unconditioned (a real damaging effect on the body) and conditioned (a “conditioned-reflex” activation of real stress via temporary connections) [37]. In the latter case, activation begins from the integrative nerve center in the brain and spreads further along the efferent branch of the reflex arc.

The psychosomatic effects of stress include not only hormonal shifts, but also the disorders in: the memory, the perception of the environment, the work of the immune, gastrointestinal and cardiovascular systems [42]. Many of them are underlain by epigene-

tic regulatory changes [43]. In the review by Gidron et al. [44], the authors analyzed 21 works on animals and humans: a relationship between the effects of psychosocial factors and DNA damages was shown. The role of the latter in the induction of malignant transformation of cells as a consequence of psychological stress is discussed [45]. Increased instability in the regions of long trinucleotide repeats, which are the cause of various diseases, has been shown for human cells under stress [46].

Recently, more and more works have been devoted to the role of stress in evolutionary changes, e.g. [47, 48]. The authors discuss the relationship between cellular stress and phenotypic variability, as well as organismal stress-induced evolution [49]. The genetic effects of stress include such as: identification of hidden genetic variation, activation of mobile elements in the genome, enhanced recombination and higher mutation rate. The relationship between transcriptional activity of the genes and their mutation frequency [50], as well as the activation of stress-induced genes, suggests that these very genes under stress situations will be preferential targets for mutations.

There is a point of view that rapid adaptation to stress “laboratory” conditions in animals at the organismal level is achieved not through formation of new stress-specific pathways but through reorganization of the set of existing pathways and functions in the body [51]. Apparently, “...by the method of conditioned reflexes, by regulating excitation and inhibition processes, concentrating them at a certain level, it is possible to reproduce the historically established complex chains of innate reflexes. Such events obtained at a time in dogs allowed I.P. Pavlov to hypothesize that innate reflexes (*or unconditioned, UCR—author’s note*) during the evolution were formed by the principle of conditioned reflex” [16].

The ability of the central nervous system of animals to rapidly form temporary interneuronal contacts, which determines the development of conditioned reflexes, was the property that provided a rapid feedback between the body and the environment, i.e., the possibility of active adaptation to the changing environment [52]. This fact is confirmed by the modern data on synaptic plasticity and rearrangement of neuronal networks depending on individual experience [53]. In particular, it can be exemplified by the specific dendrodendritic ensemble of mitral, granule and tufted cells of olfactory bulbs, which is formed depending on their activity [54].

The question about the mechanisms of transformation of temporary conditioned reflexes into unconditioned ones is still up for debate. This “genetically” significant problem was discussed by M.E. Lobashev [52]. He differentiated between the “physiological” and “genetic” points of view and tried, even then, to combine them. Assuming that a certain CR is a phenotypic adaptation developed within a series of gener-

ations, Lobashev stated that stabilizing selection “... under the cover of adaptive phenotype could, by way of selecting modifiers, fix the given reflex hereditarily, genotypically. In this case, within a series of generations, a conditioned reflex can become inherited through selection and transform into an unconditioned reflex” [52]. Since stress is a nonspecific response of the body (general adaptation syndrome), it can be supposed that at least some part of stress-induced genes will be in common for both somatic and generative cells. Artificial selection (or selective breeding) carried out by man in the desired direction is a strong stressor for animals, which activates certain gene networks. Consequently, the desired combinations of alleles (genotypes) can be selected, which determine, e.g., specific behavioral features.

The complex of innate herding behavior in dogs mentioned by L.V. Krushinsky can be considered as an example [55].

The authors of this article also personally observed the herding behavior in his German shepherd (1.5 years old) that lived in a city and had never met sheep in its life. At the same time, it: (1) met sheep *for the first time* in its life; (2) felt their presence, apparently by smell or sound, at a distance of 200–300 m (*without seeing* animals in high grass); and (3) gathered them into a group and drove them to author’s feet. The dog demonstrated quite a number of behavioral traits untypical of it: extraordinary excitement; running, circling, chasing sheep; attack simulation, specific jumping (with all fours) and barking (yapping), and other changes typical of sheep dogs.

It should be noted that, according to the pedigree data, at least seven generations of the dog’s ancestors were *solely* urban dogs and were not specially trained for shepherd service. The dog was taken away from its mother as a 1.5-month-old puppy; the development of a complex of conditioned reflexes (“shepherd behavior”) is low probable under such circumstances. The question how such behavioral complex could be maintained in a dog, if (according to the pedigree) the selection among its ancestors was carried out only with respect to constitution and general trainability, requires separate discussion.

Probably, the transformations of conditioned reflexes (“complex fixed actions”) into unconditioned ones can really be implemented only in case of: (i) long-term (hundreds, thousands of years, and more) constancy of environmental combinations of a specific unconditioned stimulus and an “indifferent” signal; (ii) combined activation of the respective neural networks (“reflex arcs”); (iii) the presence of dendrodendritic or other interactions between different interneuronal networks, one being activated by the unconditioned signal and the other being activated by the conditioned signal; (iv) the presence of allelic variants in any gene(s) contributing to the maintenance of particular dendrodendritic interactions; and (v) in

case of strict selection of the most adapted individuals, which must imply the selection of genotypes with adaptive allele combinations. In fact, such a chain of reasoning answers the question about possible mechanisms of inheritance of acquired characters.

Transformations (of CRs into unconditioned reflexes), like other evolutionary changes, can be accelerated in a stressed organism [56] through the activation of previous and the emergence of new “hot spots” in the genome of its cells. Intensification of the mutation process will be accompanied by the emergence of new allelic variants of the genes and strict “directional” selection in favor of the genotypes expressed as phenotypes most adapted to the environment.

SIGNALING HEREDITY: GENETIC CONSEQUENCES OF “INFORMATION” STRESS

“Signaling heredity,” i.e., the ability of an animal to form temporary connections (CRs), dramatically increases individual adaptive capacities of an organism. Being the consequence of directional selection, CR in a changing environment is not only the mechanism of active adaptation to this environment, but also an evolutionary tool. Through various signals, the accumulated individual experience (the system of interrelated CRs) is passed down through generations and, hence, we can speak about one more mechanism of inheritance of traits. This mechanism becomes particularly important in humans [52]. It should be noted that learning in this case can be presented as continuous development of CRs, which, in contrast to animals, occurs rather rapidly (sometimes at a single presentation). It can also be stated that individual’s memory is nothing else but the stored networks of “temporary connections” between neurons, the activation of which causes an appropriate response. This is in agreement with the views of Eric Kandel, the Nobel Prizewinner (2000), who writes that memory is specific changes in the work of nerve cells. Learning and memory are associated with the pattern of signal transmission between neurons and with the possibility of strengthening or weakening these connections [57]. Similar ideas were proposed forty years earlier by M.E. Lobashev, who wrote: “... the architectonics of the nervous system that provides temporary connection closure, i.e., unconditioned reflexes, which are the basis of conditioned-reflex activity, ...” develops into a behavioral stereotype in animal’s ontogeny [52].

Speech as a mechanism of signal transmission, which is especially developed in humans, was a fundamentally new and efficient way to increase the role of “signaling or, as it can also be called, conditioned, temporary heredity, which provides the continuity of generational experience” based on the formation of temporary connections, i.e., conditioned reflexes [52]. As it seems nowadays, its emergence is geneti-

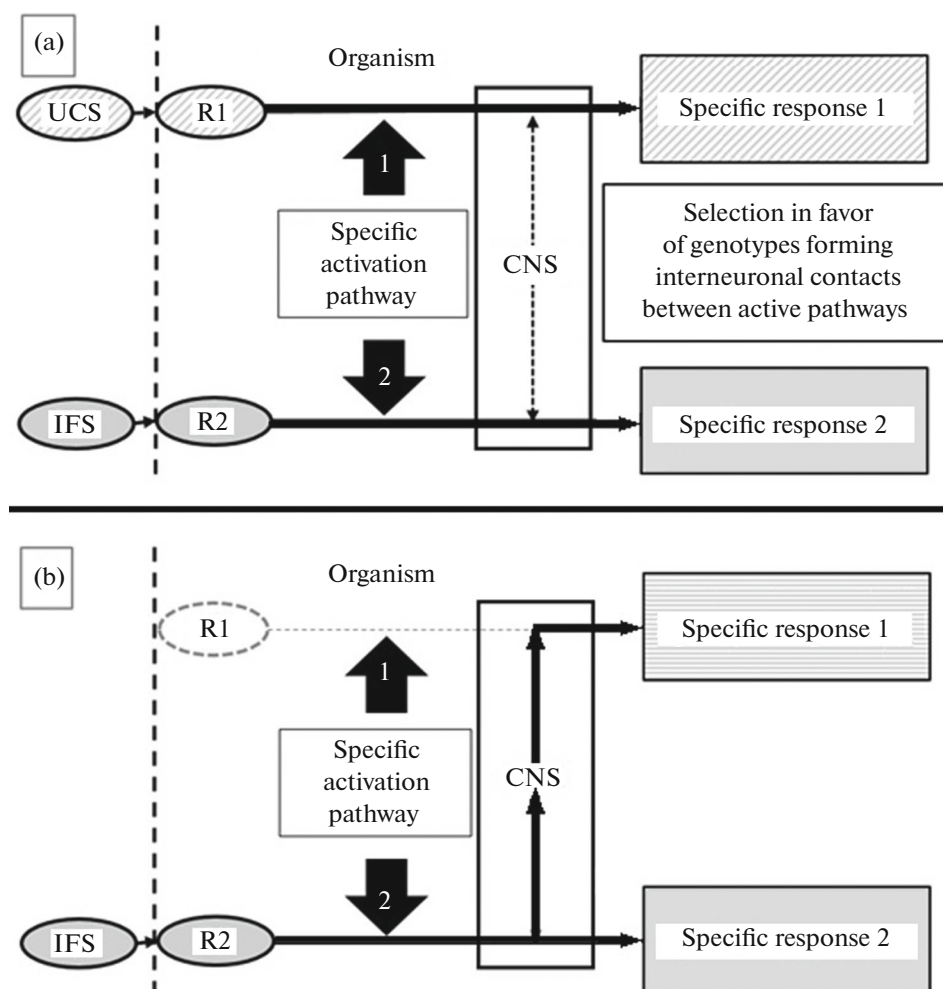


Fig. 1. Simplified diagram of the concept of the transition of conditioned reflexes into unconditioned ones. (a) Pathways of response formation to unconditioned and indifferent stimuli (UCS and IFS), (respectively); R1 and R2 are the corresponding activated receptors; CNS, the central nervous system; Specific responses (1 and 2) represent historically developed “unconditioned reflexes:” the first one can form a state of stress under certain conditions; the second one cannot form it. The dashed double-headed arrow indicates the formation of bidirectional excitation transmission channels (of the “dendrite–dendrite” type or other) during multiple combined repetitions (UCS + IFS). (b) After natural selection, even in the absence of UCS, activation of the indifferent pathway and the descending part of the unconditioned reflex pathway occurs in organisms with genotypes that provide dendritic–dendritic or other signal contacts between the activated pathways. The possibility of triggering an unconditioned reflex response by a conditioned signal appears.

cally determined by a combination of allelic variants of several hundreds of structural and regulatory genes and provided due to the coordinated regulation by FOXP2-type transcription factors.

One of the peculiar features of the mechanism of “signaling heredity” at the current stage is its continuous overstrain caused by excessive and often insignificant information. It is memorized and stored sometimes throughout the entire life of an individual. In the modern world, we create still new stressors and thereby induce stresses. This is referred to as a “stress of civilization”, “undesired tempo of life” for many people, which leads not only to functional impairment of the nervous system, but also to cardiac and kidney

diseases, increased mortality, and other consequences [58]. False stressors, which we take for true ones, can form a real stress in some people. This is facilitated by information overload, “due to which it is extremely difficult, if not impossible for us, to distinguish important things from all this white noise”. Rapid development of information technologies, together with positive moments, has led to phenomena such as “pitching” (super-brevity in presentation of ideas) and “clickbaiting” (the strive to catchy headlines). Hence, a significant proportion of people become addicted to “doomscrolling”. A “Dead Internet” theory has emerged and is gradually being supported by facts. As a result of all the above, scientific facts are often dis-

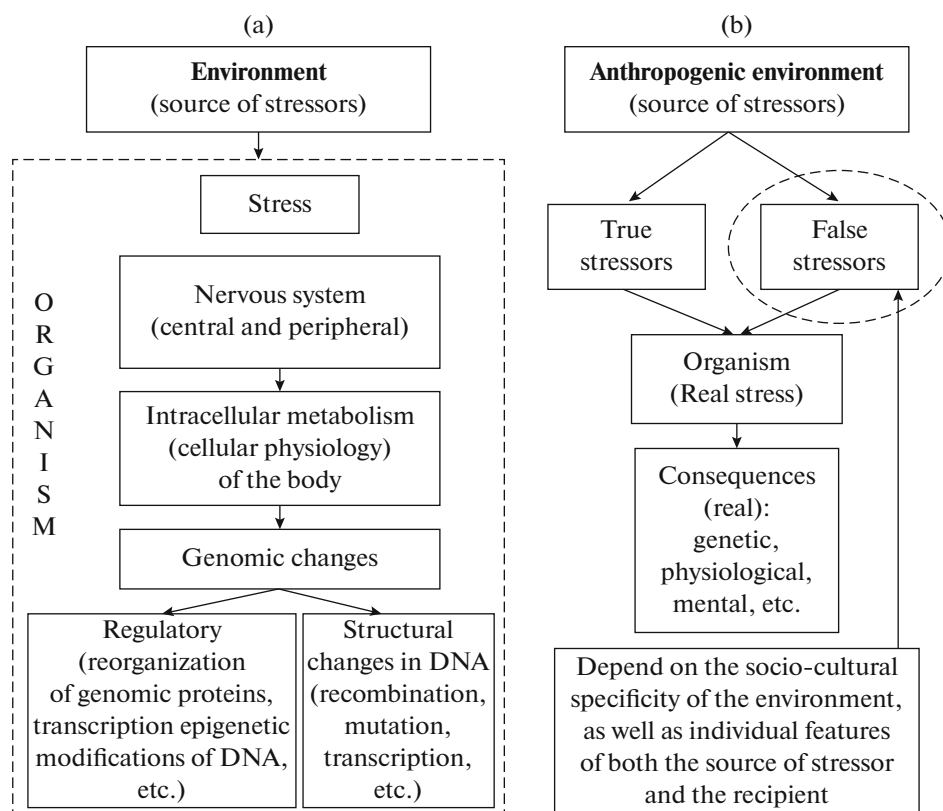


Fig. 2. The effects of natural and anthropogenic stressors (a and b, respectively) in animals and humans. The dashed line indicates human-created “imaginary” (false) stressors whose effects could have been avoided.

torted and pseudo scientific “fake news” are being spread. In view of saturation with “negative” news, the level of stress in people increases. It should be specially emphasized that the creators of the corresponding software are quite subjective, which is reflected in the principles for the algorithms they create. In short, it is precisely the pace and demands of our hectic modern life that “drive us crazy” (cause stress) [59]. A person determines stressors subjectively, in accordance with his/her “attitude of mind” and surrounding social and cultural environment [60].

“We return to the list of ... the factors that we consider stressors: traffic jams, financial difficulties, overtime work, tense relationships. Few of them are “real” in the sense that would be clear for this zebra or that lion. In our privileged life, we are uniquely clever to invent these stressors ...” [61].

When discussing the problems of oversaturation of the modern environment with real and imaginary stressors, we research little into the mechanisms of adaptation to these stressors. When speaking about signaling heredity, M.E. Lobashev and his colleagues considered “unconditioned-reflex” and “conditioned-reflex” adaptation to real stressors. The authors believed that the mechanism of unconditioned-reflex adaptation is the decrease in the strength of impulses “sent from peripheral receptors,” followed

by the “decrease in excitability in the unconditioned center” with multiple applications of an unconditioned stimulus [38]. However, reflex “fading” can be considered as a consequence of depletion of the resources of receptor cells. In this case, the latter: (i) will either stop to respond to the impact or (ii) respond more slowly (weakly, irregularly) as the resources are replenished; or (iii) cell destruction will begin, if the acting factor is damaging. Even if this is not too critical for the organism, it hardly can be referred to as a full-fledged adaptation.

False (imaginary) stressors will deplete the body’s reserves at the stages of reception, even in case of multiple repetitions of these pseudo stressor signals. However, later, when interacting with the neural networks forming the general adaptation syndrome, there may be undesired consequences. Directed “false” conditioned reflex effects in some strata of the community, groups of people, or separate individuals can be perceived as true ones (due to individuality of signaling inheritance of person’s own experience); they may acquire the strength of real stressors and cause numerous negative post-stressor effects (including those at the genetic level in somatic and generative cells).

Unfortunately, scientists, paying attention to important professions in the field of information technologies (e.g., mass media), insufficiently educate

their workers with respect to the danger of increasing the level of stress among the masses of people. In some cases, we obviously underestimate the rate of formation of temporary connections, especially the role of signaling heredity, in distribution of both adaptive and desadaptive individual experience. In the latter case, this is fraught with the induction of dangerous stress-related diseases, post-stressor disorders and, which is especially dangerous, genetic consequences (up to destabilization and even disintegration of the genome of cells of the brain, the cardiovascular, hematopoietic, immune and reproductive systems, other organs and tissues). Many negative consequences could be avoided by non-creating false stressors and by limitation of their spread.

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This work does not contain any studies involving human and animal subjects.

CONFLICT OF INTEREST

The author of this work declares that he has no conflicts of interest.

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