Humoral Link of Control in the Adaptation Process

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Abstract: Upon receiving a signal about physical load, simultaneously with the "activation" of the motor response and the mobilization of the functional system responsible for adaptation to this load, there occurs a neurogenic activation of the hypothalamic-pituitary-adrenal and adrenergic systems, which constitute the hormonal-humoral link of control in the adaptation process. The functional significance of this link, as noted above, is determined by the fact that it, through its effect on metabolism and the function of organs at the cellular and molecular levels, ensures a more complete mobilization of the functional system responsible for adaptation and its ability to maintain work at an elevated level for a prolonged period. The degree of activation of this link and its role in the mechanism of adaptation to load in an unadapted organism, i.e., at the stage of "urgent" adaptation, and in an organism trained for load, i.e., with established adaptation, are not the same.

Key words: humoral link; adaptation process

Introduction

Upon receiving a signal about physical load, simultaneously with the "activation" of the motor response and the mobilization of the functional system responsible for adaptation to this load, there occurs a neurogenic activation of the hypothalamic-pituitary-adrenal and adrenergic systems, which constitute the hormonal-humoral link of control in the adaptation process. The functional significance of this link, as noted above, is determined by the fact that it, through its effect on metabolism and the function of organs at the cellular and molecular levels, ensures a more complete mobilization of the functional system responsible for adaptation and its ability to maintain work at an elevated level for a prolonged period. The degree of activation of this link and its role in the mechanism of adaptation to load in an unadapted organism, i.e., at the stage of "urgent" adaptation, and in an organism trained for load, i.e., with established adaptation, are not the same.[2]

Urgent Adaptation to Physical Load. The Role of the Stress Response

Urgent adaptation is characterized by significant, often excessive activation of the hypothalamic-pituitary-adrenal and adrenergic systems. This is accompanied by intense release of corresponding hormones and mediators, including corticoliberin, somatoliberin, ACTH, somatotropin, other pituitary hormones, catecholamines, glucocorticoids, and mineralocorticoids. The release of triple hormones and catecholamines leads to the release or suppression of the secretion of hormones of the next line of regulation—insulin, glycogen, thyroid hormones, calcitonin, etc. This manifestation of the universal stress response of the organism, which arises in response to environmental factors, was first described as a nonspecific component of the organism's holistic adaptation to changed environmental conditions. The main results of this reaction are:

- Mobilization of the organism's energy resources and their redistribution with selective direction to the organs and tissues of the functional adaptation system;
- Potentiation of the work of this system itself;
- Formation of the structural basis for long-term adaptation;
- In the case of excessive stress response, disturbances in cellular membranes occur, leading to enzymeemia and damage.[9]

Sympathoadrenal (Adrenergic) System To date, a large experimental material has been accumulated, unequivocally indicating that under the influence of physical loads, activation of the sympathoadrenal system occurs in humans and animals. This is manifested by increased release of catecholamines from the adrenal glands and norepinephrine from the endings of sympathetic fibers in the tissues, and accordingly, a multiple increase in the level of catecholamines and their metabolites in blood and urine.[1]

Increased release of norepinephrine from sympathetic terminals and secretion of adrenaline and norepinephrine from the adrenal glands during physical exertion may either not lead to a change in the level of catecholamines in the adrenal glands, heart, and

other tissues, or may cause a decrease and even depletion of these compounds. The level of catecholamines in tissues, under otherwise equal conditions, is determined by the ratio of two main processes: the release of catecholamines from tissues and the restoration of their reserves; the latter is realized through synthesis and reuptake. Normally, these processes are coordinated based on a feedback mechanism, whereby increased release of catecholamines leads to the activation of their synthesis. If the load is small or short-lived, then the activated synthesis can quickly restore the level of catecholamines in tissues, and their level does not change significantly.[13]

With more intense and prolonged exertion, the rate of catecholamine synthesis may lag behind their release and expenditure, and then the level of these compounds in tissues decreases, although the level in the blood remains elevated. This lag may be associated with both insufficient power of the enzymatic apparatus of synthesis and insufficient supply of precursors. During exhausting loads, a situation arises where the synthesis of catecholamines not only lags behind their increased release but decreases below normal, i.e., the synthesis apparatus is depleted. This leads not only to a sharp drop in the level of catecholamines in tissues but also to a decrease in their release from the adrenal glands and tissues into the blood.[5]

It is significant that as a result of training, as will be shown later, not only does the response of the adrenergic system to exertion decrease, but the power of the catecholamine synthesis apparatus increases, which prevents the depletion of their content during intense work and the disruption of the function of the adrenergic system. With the activation of the sympathoadrenal system and the increase in the level of catecholamines in the blood during physical exertion, there is a correlation with changes in the secretion of pancreatic hormones—glucagon and insulin. It has been shown that during muscular exertion, proportionally to its magnitude and duration, there is an increase in glucagon secretion and its concentration in the blood; this increase during loads close to maximum can be 2-3 times.[8]

At the same time, the rise in glucagon concentration in the blood has a certain latent period, which is shorter the greater the intensity of the load; therefore, with small and short-term loads, no changes in glucagon levels are observed. The main factor causing an increase in glucagon secretion during exertion is considered to be the effect of catecholamines on the beta-adrenergic receptors of the alpha-cells of the pancreas. This action is realized as a result of the activation of the adrenergic system and the increased influx of norepinephrine from sympathetic terminals in the gland, as well as adrenaline and norepinephrine from the blood the maintenance of high levels of glucagon in the blood. [3]Against the background of an already decreasing level of catecholamines, the maintenance of a high level of glucagon in the blood, according to some authors, indicates the presence of some additional stimulators of hormone secretion during physical exertion.

It is possible that this factor is glucose, as it has been shown that a decrease in blood glucose levels increases glucagon secretion. It is assumed that the basis of this action lies in the direct influence of hypoglycemia on the alpha cells of the pancreas. Hyperglycemia, on the contrary, leads to a decrease in the level of glucagon in the blood.

Simultaneously with the increase in glucagon secretion during physical exertion, a decrease in insulin concentration in the blood is observed. However, unlike the dynamics of glucagon, the level of which increases proportionally to the duration and intensity of the load, the decrease in insulin concentration occurs during moderate and intense loads, but is not observed during heavy, near-maximum loads.[14]

On the contrary, during short-term intense exercises, an increase in insulin concentration in the blood is noted; this may be related to the simultaneous hyperglycemia observed. Indeed, it has been shown that the introduction of glucose and the rise in its level in the blood prevents a decrease in insulin concentration during work; in this situation, an increase in hormone concentration is observed.

It is assumed that the main factor determining the decrease in insulin concentration in the blood during muscle work is the effect of norepinephrine and, to a lesser extent, epinephrine on adrenergic receptors. Thus, the above shows that the activation of the adrenergic system during physical exertion and the accompanying increased release of norepinephrine from sympathetic terminals in the pancreas has an opposite effect on the secretion of glucagon and insulin.

The effect of the mediator on the alpha cells of the gland stimulates glucagon secretion, while the effect on the beta cells suppresses insulin secretion. During physical exertion, there is a regular increase in the release of somatotropin (growth hormone) from the adenohypophysis into the blood, which is due to the increasing secretion of the corresponding releasing factor, somatoliberin, in the hypothalamus under these conditions.

The concentration of somatotropin in the blood gradually increases, proportionally to the intensity and duration of work, and can exceed the initial level by several times; at the same time, a certain latent period is observed, which is shorter the greater the power of the work. The elevated level of somatotropin in the blood is maintained throughout the work and, after its completion, is replaced by a slow, gradual decrease in hormone concentration to the initial level; however, during prolonged exercises, the hormone content may decrease even during work, which, apparently, is determined by the fact that in an untrained body, the secretion of the hormone cannot sufficiently long cover the uptake of the hormone by the tissues.

The role of physical activity in somatotropin secretion a significant role in increasing somatotropin secretion during physical exertion is played by the decrease in blood glucose levels, which serves as a stimulus for the release of somatoliberin from the hypothalamus. Furthermore, there is evidence indicating a direct potentiating effect on the synthesis of somatotropin in the pituitary cells by glucocorticoids and thyroid hormones, the levels of which also rise in the blood during physical activity. However, the primary stimulus for the release of growth hormone is apparently catecholamines, which act on the adrenergic receptors of the hypothalamus and trigger the release of somatoliberin.[18]

Physical activities systematically cause a rapid activation of adrenal cortex function and the release of corticosteroids into the blood. This activation results from neurogenic stimulation of the hypothalamus, leading to the secretion of the corticoliberin releasing factor, which enters the pituitary and activates the production and secretion of corticotropin (ACTH) — a stimulator of the cells in the zona fasciculata of the adrenal cortex that secretes glucocorticoids. This process leads to a rapid increase in the level of glucocorticoids in the blood, which occurs within the first minutes of muscle work. Moreover, the higher the intensity of the work, the steeper the rise in hormone concentration in the blood.

During prolonged work, the response of the pituitary-adrenal system is characterized by phases; at the beginning of the work, there is an increase in the level of these corticosteroids in the blood, followed by a decrease in hormone concentration, and then an increase again, and so on. After the work is completed, there is a quite prolonged period of "aftereffects," also characterized by phases. It is significant in our exposition that in an untrained organism, the response of the pituitary-adrenal system is generalized, excessive, and can quickly become depleted under sufficiently prolonged and intense loads.

Data on changes in thyroid gland function during physical activity are quite contradictory. As early as 1963, Skebelskaya suggested that any influences leading to an increase in ACTH and corticosteroid levels in the blood cause suppression of thyroid hormone synthesis, which is then followed by an increase in the secretion of these hormones. Subsequent studies found that during exhausting loads in untrained animals, the concentration of protein-bound iodine in the blood either does not change or decreases. At the same time, studies on humans have shown that work on a cycle ergometer leads to an increase in the concentration of free and total thyroxine in the blood.

It was established that the level of thyroid hormones in the blood depends on the intensity of the load: intense exercises on the cycle ergometer caused an increase in the concentration of thyroxine and triiodothyronine in the blood, while during moderate work, the level of thyroxine did not change, and triiodothyronine decreased. A similar dependence was observed for the concentration of thyrotropin in the blood. Despite such contradictory data, there have recently been opinions that physical activity leads to the gradual activation of the hypothalamic-pituitary-thyroid system and, accordingly, the function of the thyroid gland. In favor of this judgment, apparently, data may testify that under conditions of physical load, the introduction of triiodothyronine (the main physiologically active product of the thyroid gland) leads to an increase in performance, while suppression of gland function results in a decrease in the body's performance. In response to physical load, the secretion increases and the concentration in the blood of hormones regulating water-electrolyte balance—aldosterone, renin, vasopressin—rises. It is significant that in most cases, the enhancement of the secretion of these hormones occurs when the work is accompanied by water loss due to increased sweating and disturbances in water-salt balance. Therefore, in such sports as swimming, where heat dissipation is high and there is no need for sweating, a significant increase in the secretion of aldosterone and vasopressin is not observed. The secretion of aldosterone, a hormone of the glomerular zone of the adrenal cortex, increases gradually with loads, proportionally to the power of work, and the increase in the concentration of the hormone in the blood can be multiple. Its secretion is regulated by changes in the ratio of sodium and potassium concentrations in the blood and adrenal glands, the activity of vascular baro- and volume receptors, corticotropin, and others.[15] The most significant role in this belongs to renin—the hormone of the juxtaglomerular cells of the kidneys. An increase in the content of this hormone in the blood leads to the activation of angiotensin I and its transition to the active form—angiotensin II, which stimulates the secretion of aldosterone. The increase in renin levels in response to load can also be multiple, and the main stimulator of this process is the decrease in plasma volume and central venous pressure that occurs during muscle work, which reflexively causes the activation of the juxtaglomerular apparatus with the participation of vascular receptors. Significant influence is also exerted by changes in the ionic composition of the blood: the intake of table salt prevents, while a salt-free diet enhances the secretion of renin during loads. The increase in vasopressin secretion by the hypothalamus during muscle work is also associated with the loss of water by the body caused by sweating and shifts in the balance of electrolytes in the blood, but this increase is significantly less than the rise in the concentration of aldosterone and renin. During physical loads, a significant but slow (over 3-4 hours) increase in the level of calcitonin in the blood—a hormone regulating Ca^{2+} metabolism—is observed in humans and animals. The increase in the secretion of this hormone by the thyroid gland during physical load is associated with the activation of parafollicular cells of the gland, caused by increased production of corticosteroids and their catabolic effect on bone tissue. The increased secretion of calcitonin and the simultaneous decrease in the level of parathyroid hormone (the hormone of the parathyroid gland) during load, apparently, contributes to the reduction of the increase in Ca^{2+} concentration in the blood, caused by. Intensive load, as well as the increase in the influx of Ca 2+ to working muscles and the limitation of this influx in the case of exhausting loads. We do not stop here on the changes in sex hormones during physical exertion, since they are not directly related to muscle work, however, we note that they play a certain role in the processes of proliferation and protein synthesis, and therefore, obviously, their secretion level should be taken into account when analyzing the mechanisms of long-term adaptation.[7]

The physiological significance of the hormonal shifts listed above is determined, as already noted earlier, primarily by their participation in the energy supply of muscle work, in the mobilization of the body's energy resources. The main role in the mobilization of carbohydrate and fat depots during load is played by catecholamines. It is known that by acting on beta-adrenergic receptors, i.e., through the activation of the adenylate cyclase system and cAMP-dependent protein kinases, as well as with the participation of alpha-adrenergic receptors, catecholamines activate key enzymes of glycogenolysis and glycolysis, and as a consequence, these processes in skeletal muscles, heart, liver increase the release of glucose into the blood from the liver and its transport to the myocardium and skeletal muscles, potentiate gluconeogenesis in the liver from amino acids and lactate.

In addition, catecholamines through the indicated mechanisms activate lipoprotein lipases and lipases in adipose tissue, lipoprotein lipases in skeletal muscles, heart and contribute during muscle work to the reduction of triglyceride content in these tissues and the formation and release of free fatty acids. A significant role in the mobilization of carbohydrate depots, especially in the liver, during load is played by the increase in glucagon secretion, which is essentially a synergist of catecholamines in the process of activating glycogenolysis. This hormone also plays an important role in gluconeogenesis, which is stimulated in the liver during load, since the glucagon/insulin ratio regulates this process.

Catecholamines, stimulating the secretion of glucagon in the pancreas and suppressing the secretion of insulin, increase this ratio and thereby contribute to the formation of glucose in the liver. A significant influence on this process is also exerted by the change in the ratio of glucocorticoids/insulin, as the increase in glucocorticoid concentration activates gluconeogenesis enzymes, for example, glucose-6-phosphatase, while insulin, on the contrary, suppresses this enzyme. [19] In addition, glucocorticoids contribute to the increase in glucose production in the liver due to the suppression of protein synthesis in tissues and thereby increase the pool of free amino acids (catabolic effect) and influx into the liver, as well as the activation of transamination and deamination enzymes of amino acids and involvement in gluconeogenesis.

In particular, the enzyme alanine aminotransferase is activated, and the glucose-alanine cycle is realized, which is of great importance for the growth of energy supply during urgent adaptation to load and consists in the fact that part of the mobilized amino acids can be oxidized in skeletal muscles. nsulin, as is known, increases the transport of glucose and its utilization in skeletal muscles. Therefore, a decrease in insulin concentration in the blood during physical exertion should, to some extent, reduce the influx of glucose from the blood into the muscles and its utilization. However, during exertion, blood supply to the working muscles increases, and therefore their supply of insulin can likely be maintained at a level close to necessary even under conditions of reduced hormone concentration.[20]

It should also be noted that some limitation of glucose consumption from the blood by the muscles may play a role in the energy supply of the brain, as it contributes to reserving glucose for nerve cells, which can absorb and utilize glucose without the involvement of insulin and, unlike muscles, cannot utilize free fatty acids. In the mobilization of resources during physical exertion, glucagon participates alongside catecholamines, exerting a similar activating influence on hormone-dependent lipoprotein lipases in the heart and skeletal muscles, which are involved in the mobilization of free fatty acids from adipose tissue and their release from triglycerides in the plasma and intracellular triglycerides, as well as somatotropin, corticotropin, and thyroxine.

A significant potentiating influence on the function of catecholamines during exertion, including their effect on energy supply processes, is exerted by glucocorticoids, which stimulate the synthesis of adrenaline and suppress the activity of cAMP phosphodiesterase, i.e., contribute to an increase in the content of catecholamines and enhance their effectiveness on adenylate cyclase and the growth of cAMP levels in cells. A certain role in this process is played by the decrease in insulin concentration, which is the antagonist of glucocorticoids concerning protein synthesis in the liver and muscle tissue, and therefore the decrease in the concentration of this hormone during physical exertion potentiates the catabolic effect of glucocorticoids, i.e., the "outflow" of amino acids from tissues and their entry into the liver, as well as contributes to the stimulation of gluconeogenesis by glucocorticoids in this organ.

Thus, the hormonal shifts discussed play an important role in the mobilization and redistribution of the body's energy resources, aimed at enhancing the energy supply of working muscles, the heart, the brain, and other organs of the functional adaptive system. At the same time, these hormonal changes ensure the mobilization of the function of this system during exertion. For instance, catecholamines, through the adenylate cyclase system and cAMP-dependent protein kinases, exert a positive inotropic effect on the heart, increasing the amplitude and speed of contraction and the efficiency of relaxation of the cardiac muscle. Activation of sympathetic regulation of skeletal muscles and, accordingly, the increased release of norepinephrine in them facilitates neuromuscular transmission during exertion, increases the strength of muscle contraction, thereby contributing to the realization of the motor response. [10]

The lipotropic effect of catecholamines, associated with their action on lipases, lipoprotein lipases, phospholipases, and the activation of lipid peroxidation, causes a change in the lipid microenvironment of enzymes and receptors in cell membranes, which, at a non-excessive intensity of this process, activates these components in the cells of the heart, skeletal muscles, brain, and other organs of the functional system, playing an important role in mobilizing this system at the urgent stage of adaptation.

A significant role in the mobilization and maintenance of heart and muscle function during physical exertion is played by increased secretion of glucocorticoids. Acting through a specific system of cellular receptor proteins, these hormones ensure timely resynthesis and preservation of the population of Na^+K^+-ATPase, and possibly other transport ATPases, which is necessary to maintain optimal water and sodium content in cells, as well as to carry out the process of excitation.

Thus, in studies by Kyrge, a direct relationship was found between the activation of Na^+-K^+-ATPase in the myocardium during physical exertion and the increase in blood concentration of corticosterone. It was established that the increase in enzyme activity at the beginning of exertion was preceded by an increase in hormone levels, while the decrease in enzyme activity during exhausting loads was associated with a decrease in glucocorticoid activity in the blood. It was also shown that a drop in glucocorticoid levels contributes to the development of myocardial edema and impairment of its contractile function.[17]

An important role in the mobilization and maintenance of the function of receptor structures and transport ATPases at the urgent stage of adaptation to loads, as well as especially during the establishment of long-term adaptation, apparently belongs to thyroid hormones. Evidence supporting this assumption comes from data obtained on cardiac muscle. For example, in one study, it was shown that in rats, the reactivity of the adenylate cyclase system of cardiomyocytes in relation to catecholamines and the inotropic effect of catecholamines on the heart as a whole are reduced in hypothyroidism and increased approximately 10 times in hyperthyroidism compared to euthyroid control.

Studies have established that the activity of Ca^{2+}-stimulated ATPase of the SR and its ability to absorb Ca^{2+} in the myocardium of hypothyroid animals are reduced, while in hyperthyroid animals, they are increased. [2]Noting the important positive role of hormonal shifts and responses in the implementation of the urgent stage of adaptation to physical exertion, it should be emphasized that during prolonged and intense loads acting on an untrained organism, excessive activation of the hormonal regulatory department, primarily the adrenergic system, often leads to the manifestation of a negative, damaging effect of the stress response.

This effect is due to the influence of an excess of catecholamines on organs and tissues, where the aforementioned lipotropic influence of these compounds becomes excessive and, through a chain of processes, leads to the activation of lipid peroxidation in cells. In combination with other reasons for the activation of the process of peroxidation under intense loads, namely hypoxemia, tissue hypoxia, and acidosis, this action of catecholamines leads to damage to cell membranes, disruption of their functions, and the functions of cells and organs as a whole. As a result, the positive effects of catecholamines, expressed in the mobilization of energy supply and the performance of the system responsible for adaptation, turn into damaging, negative effects. Indeed, it has been shown that under maximum loads in an untrained organism, a decrease in performance is accompanied by the activation of peroxidation and enzymeemia. Preliminary administration of antioxidants that inhibit the activation of peroxidation prevents these phenomena and leads to an increase in endurance.

Returning to the positive role of the stress response in the implementation of the urgent stage of adaptation, it should be emphasized that a number of hormones have a direct inductive effect on the synthesis of nucleic acids and proteins, thereby ensuring the realization of the stage of generalized activation of nucleic acid and protein synthesis following the catabolic phase of the stress response, i.e., the anabolic stage of the reaction. At this point, in the functional system responsible for adaptation, this activation is summed with the increase in synthesis caused by the hyperfunction of the organs and tissues of this system, thus laying the foundation for the formation of a structural basis for long-term adaptation to physical load.[6]

As already mentioned, an important role in the mobilization of protein metabolism, the creation of a pool of free amino acids for transamination in the necessary directions, and the induction of enzyme synthesis belongs to glucocorticoids. Catecholamines have the ability to induce the synthesis of nucleic acids and proteins. In particular, it has been proven that the administration of a relatively small dose of isoproterenol, an analogue of norepinephrine, causes a sharp increase in the synthesis of ribosomal and transport RNA in cardiac muscle. Accordingly, the influx of a significant amount of norepinephrine into the myocardium may be one of the factors causing the activation of protein synthesis and the development of hypertrophy of the cardiac muscle during adaptation to physical loads.

An important role in inducing protein synthesis during adaptation to physical loads belongs to thyroid hormones. It has been shown that the suppression of thyroid function by the administration of thiouracil prevents, while the administration of small doses of triiodothyronine enhances the development of endurance during training. It has been shown that the administration of thiouracil reduced, while the administration of thyroid hormone increased the activity of mitochondrial enzymes in skeletal muscles. [21] This indicated that thyroid hormones participate in the formation of adaptation to physical loads at the level of mitochondria in skeletal muscles. The nature of these facts becomes clear in light of previous data that thyroid hormones in doses close to physiological levels lead to an increase in.

RNA concentration in tissues increases the inclusion of labeled amino acids into proteins, and these effects are prevented by RNA synthesis inhibitors—actinomycin D—and protein synthesis inhibitors—puromycin.

Moreover, studies using electron microscopy have shown that when small doses of thyroid hormones are administered to normal and thyroidectomized rats, the increase in mitochondrial enzyme activity in skeletal muscles is accompanied by a rise in the number

of mitochondria per unit body mass. Thus, the presented evidence indicates that during adaptation to physical loads, thyroid hormones participate in enhancing the power of the mitochondrial system in skeletal muscles and other tissues through direct induction of mitochondrial protein synthesis.

Thyroid hormones play a significant role in adaptation to physical loads and in the activation of the synthesis of various structures of the heart muscle and the development of its hypertrophy. This is supported by data showing that changes in the contractile function of the myocardium during heart hypertrophy induced by the administration of thyroxine are similar to those observed in hypertrophy caused by adaptation to physical loads.

Inducers of protein synthesis during adaptation to physical loads also include somatotropin, insulin, and androgens. It is significant that these hormones exert a mutual additive effect on protein synthesis. For example, as noted above, thyroid hormones activate the secretion of growth hormone and, accordingly, its effect on the process of its synthesis. Insulin also has a strong potentiating effect on growth hormone through the hypoglycemia it induces, etc.

It is important that the secretion of anabolic hormones occurs gradually, with a latent period, when the realization of their action is already "prepared" by the catabolic effect of glucocorticoids, the secretion of which increases very rapidly and provides a pool of free amino acids for the activation of protein synthesis primarily in the organs of the functional system.[16]

Thus, the activation of the hormonal-humoral regulatory link at the stage of acute adaptation to physical loads plays an important role not only in the realization of this stage but also in the formation of the subsequent stage of adaptation—long-term adaptation or training.

Long-Term Adaptation to Physical Load

In the process of forming a stable long-term adaptation to physical load, a restructuring of the hormonal-humoral regulatory apparatus of the functional system responsible for adaptation occurs. The results of this restructuring are characterized by two main features.

The first feature is that in the adapted organism, the hormonal regulatory link functions more economically. In response to the same load, such an organism experiences significantly less activation of the hormonal system and, accordingly, a smaller increase in concentration.

Hormones in the Blood than in the untrained. This is accompanied by a limitation of the stress response and the absence of a negative, damaged component of this response, associated with excessive "release" of hormones. The second feature of adaptive restructuring is that in the adapted organism, the power of the hormonal-humoral regulation apparatus is increased. [11] This, firstly, prevents the exhaustion of the apparatus during prolonged loads and thereby creates the possibility of adequate hormonal support for prolonged muscle work, and secondly, increases the maximum response of the apparatus under extreme loads, which ensures maximum mobilization of the functional system.

Let us consider some examples illustrating these positions. In the process of forming adaptation to physical loads, the response of the adrenergic system to non-extreme, standard loads decreases. This is expressed, firstly, in that trained people and animals experience significantly less release of catecholamines and an increase in their concentration in the blood and urine in response to such loads than untrained individuals. Secondly, it has been established that in trained animals, intense load either does not cause a decrease in norepinephrine in the myocardium, brain, and other tissues at all, or this decrease is significantly less than in untrained individuals.

It should be noted that at rest, in most cases, there are no significant differences in the concentration of catecholamines in the tissues and blood of adapted and non-adapted people and animals. The economization of the response of the adrenergic system to load during training is formed gradually. [12]. Thus, in individuals trained for endurance, the increase in catecholamine concentration in the blood in response to intense load gradually decreased over 3 weeks of training; after 3 weeks, it was significantly less than in untrained individuals, and after 8 weeks of training, it was not observed at all.

The mechanism of economization of the functioning of the adrenergic system with training is still not sufficiently clear; however, it is undoubtedly of a central nature and is associated with the more important functioning of the central neurogenic link in the management of adaptation in the trained organism. At least two circumstances may underlie this mechanism. Firstly, the economy of the response of the adrenergic system to load may be due to the phenomenon of increased adrenergic reactivity of tissues observed with training. Many researchers have shown that in trained greyhounds, standard stimulation of the stellate ganglion caused a stronger hemodynamic response than in untrained mixed-breed dogs. The authors consider the increased adrenergic reactivity of the circulatory system as a manifestation of training for prolonged muscle work.

Research shows that in cats adapted to swimming for several weeks, the magnitude of the lyotropic response of the heart and the increase in the concentration of cAMP in response to the administration of isopropyl norepinephrine is significantly higher than in

non-adapted individuals. The conclusion that adaptation to Physical exercise includes the economy of mediators of the adrenergic system, based on the sensitivity of tissues to them.

However, it should be noted that not all data in the literature is unambiguous in this regard. There is information indicating that adrenergic reactivity of tissues in detrained animals and metabolic reactions to the action of catecholamines in trained individuals may be less than in untrained ones, or may not differ from them at all.

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